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THE

SCIENCE AND PRACTICE OF MEDICINE.

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THE
SCIENCE AND PRACTICE
OF
MEDICINE.

BY

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PROFESSOR OF PATHOLOGY IN THE ARMY MEDICAL SCHOOL.

SIXTH EDITION.

GREATLY ENLARGED, RE-MODELLED, CAREFULLY REVISED, AND MANY PORTIONS RE-WRITTEN,

(ADOPTING THE NEW NOMENCLATURE, AND FOLLOWING THE ORDER OF CLASSIFICATION OF DISEASES PUBLISHED BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON, 1869.)

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THE

SCIENCE AND PRACTICE OF MEDICINE.

PART III.—CONTINUED.

GENERAL DISEASES.—*Continued.*

CHAPTER XI.—CONTINUED.

DETAILED DESCRIPTION OF THE GENERAL DISEASES COMPREHENDED IN SECTION B.

SCROFULA.

LATIN EQ., *Struma;* FRENCH EQ., *Scrofule;* GERMAN EQ., *Scrophulose;* ITALIAN EQ., *Scrofola.*

Definition.—*A constitutional disease, resulting either in the deposit of tubercle or in specific forms of inflammation or ulceration.*

Pathology.—The diseases with which the name of scrofula is associated are manifested by a remarkable tendency to certain forms of nutritive disorder, and hence they are strikingly wasting in their effects upon the body. They comprehend the following specific forms, namely:—(*a.*) *Scrofula,* with *tubercle;* (*b.*) *Scrofula,* without *tubercle,* and certain local scrofulous affections, such as *tubercular meningitis, hydrocephalus, scrofulous ophthalmia, tubercular pericarditis, scrofulous diseases of glands, phthisis pulmonalis* or *pulmonary consumption, acute miliary tuberculosis,* or *tabes mesenterica, tubercular peritonitis*—each of which will be considered in its proper place, under the local diseases of the organs affected.

Many of these are characterized by the growth of a peculiar substance in the tissue of some organs rather than in others, to which the name of *tubercle* has been given. These growths occur in the tissue of the alimentary canal; in the peritoneum, arachnoid, or pleura; in the lungs, liver, spleen, or kidney; in the tissue of the lymphatic glands, especially the cervical, inguinal, and mesenteric glands; and sometimes in the pancreas and the tonsils.

These lesions owe their existence to constitutional conditions. There can be no doubt that, in the condition of *scrofula with tubercle*—the *tubercular* or *wasting* diseases—as well as in those in which *scrofula* is expressed *without tubercle,* there is a *latent* condition existing before the tubercles are apparent in the one case, and a scrofulous diathesis in the other.

The relation of the nutritive and other morphological changes between the solids and the fluids of the body has everything to do with the development of the scrofulous state. In cases where the tendency to the growth of *tubercles* is hereditary, the operation of agents from without act as stimuli or excitants to the growth of them.

Previous to 1829, when the late Sir James Clark published his classic work *On Climate*, the tendency of pathological researches into the nature of the diseases just mentioned was to keep up the idea that these diseases, and especially *pulmonary consumption*, were merely local, and referable to a local cause.

The terms *phthisis, consumption*, or *wasting*, were originally used vaguely to designate a variety of chronic diseases, described entirely by local symptoms and physical signs, and therefore having few characters in common, except the marked emaciation which attends them. When researches in morbid anatomy became more frequent and efficient, the local lesion most commonly connected with the wasting of the body was discovered to be a peculiar morbid condition of the lungs, and to this the name of "*consumption*" was applied, and afterwards more definitely, "*pulmonary consumption*." From the frequency of this local lesion the pulmonary state of necessity attracted far greater attention than any other form of *scrofula* in which *wasting* was a marked feature; and when lesions in other organs were found associated with "*pulmonary consumption*," they were regarded as complications rather than as local manifestations of one and the same general constitutional state, and were looked upon as the primary cause of the emaciation. In 1819 Laennec first showed, by his accurate *post-mortem* observations on the state of the lungs, that growths to which the name of "tubercles" were applied formed almost the sole cause of *consumption*, and consequently he restricted the term *phthisis* to "*the disease produced by tubercles* in the lungs. Louis and Andral confirmed the observations of Laennec; and thus the view of this morbid state, based as it was on morbid anatomy *alone*, led to the enunciation of limited and erroneous doctrines regarding the real nature of the disease which the existence of *tubercles* indicates, either in the lungs or in any other part of the body.

The relations subsisting between *pulmonary phthisis, consumption*, or *wasting*, and the occurrence of "tubercle," must now be even still more modified under the combined researches of morbid anatomy and progressive knowledge in pathology. *Consumption*, or wasting, in the common acceptation of the term, is not now found due always to tubercle (ADDISON, GAIRDNER, REINHARDT, VIRCHOW, JENNER, and MACLACHLAN), so that the terms *phthisis* and *tubercle* cannot now be considered always as synonymous. Ulceration of the lungs and partial destruction of pulmonary tissue are found to arise from other causes than the existence of *tubercles*, which become equally efficient causes of phthisical mischief (BAYLE, ARMSTRONG, GRAVES, STOKES, and MACLACHLAN). Nay, we know also that many states of the body to which the name *scrofula* or *struma* is applied are not necessarily attended with "tubercles" at all; yet, when the local lesions of a tubercular nature are observed to be connected with marked constitutional states under various circumstances, the connection between *scrofula with and without tubercle* is seen to be of the closest description, and their pathological history is now generally believed to be identical.

The extensive observations of morbid anatomists since the time of

Laennec, elucidated by the most learned pathologists of the age, now tend to establish on a very broad foundation the doctrine first so strenuously and ably advocated by Sir James Clark, that the morbid conditions now mentioned are due to what he termed a "tuberculous cachexia;" and accordingly it is proposed to consider the *constitutional* disease, of which the occurrence and growth of "tubercles" is but a local expression, under the general title of SCROFULA, of which the varieties are (a.) *Scrofula with tubercle;* and (b.) *Scrofula without tubercle;* while the local scrofulous affections are those named at the commencement of this topic.

Clinical and pathological research tend more and more to confirm the belief in the constitutional origin of scrofula. There is undoubtedly a predisposition to scrofulous growths, either hereditary or acquired; and that there is a bad habit of body—a cachexia—which precedes the development of tubercle, is now almost a general belief.

The development of tubercles, wherever found, is undoubtedly a local lesion which indicates a *constitutional disease.* It has been hitherto, however, the custom to name the local lesions as the *disease,* rather than to describe the constitutional state. Thus, when the lesions have been most marked in the bones and glands, the name of *scrofula* has been given to the condition; when the lungs are the site of the deposits, *phthisis* or *consumption* is the name by which the condition is familiar; and when in the glands of the mesentery, it has been called "*tabes mesenterica;*" and in the meninges of the brain it has been named "*hydrocephalus,*" or "water in the head." The constitutional disease under which all these affections are now comprehended is named "scrofula," and is shortly defined as "*a constitutional disease resulting either in the deposit of tubercle or in specific forms of inflammation and ulceration.*"

The changes in the general system by which the scrofulous cachexia is brought about are apparent in the physical condition of the patient, and in the exercise of some of the vital functions especially connected with nutrition. To the late Dr. Tweedy Todd, Sir James Clark, and Professor Bennett, the profession is principally indebted for the clear and earnest elucidation of *scrofula* or *tuberculosis* considered as a constitutional affection; and on our knowledge of it as such rests our only hope of success in the prevention and treatment of this most formidable scourge of civilized society.

Many observations have been made and statements recorded with the view to connect scrofula with morbid states of the blood, but hitherto no constant morbid condition of the circulating fluid can be said to be peculiar to the disease. As Dr. Bennett observes, we must look to something beyond—we must look to the pabulum which ministers to the nutrition of the body itself through the blood; for with an impoverished state of that fluid there is doubtless an impoverished state of the tissues. But there are some curious and detached observations which, when connected together in certain pathological relations, appear to throw more light on the nature of the constitutional state which leads to the development of scrofula than any single observation of individual authors. These I would thus shortly enumerate:—

1. There is to be noticed the albuminous character of the fluid

material which infiltrates the tissues of an organ previous to the process of *tuberculization*, by which this infiltration is in part changed by coagulation into *grey tubercles*, each of which may block up from three to twenty air-vesicles in the lung.

2. There is in some cases, as shown by Drs. Alison, Williams, Bennett, and Rokitansky, an obvious affinity between the lymph of the blood and tubercle.

3. There is an albuminosity or venous state of the blood which is considered by some as peculiar to the scrofulous state of the constitution.

4. There is the peculiar state of the blood, amounting to an appearance of leuchæmia, which immediately succeeds digestion in healthy persons, as observed and described by Dr. Andrew Buchanan, of Glasgow, in *The Transactions of the Philosophical Society* (vol. ii.) of that city, resembling the molecular and corpuscular elements of chyle or lymph, and consisting of fat emulsionized with albumen. To this substance he gave the name of *pabulin*, and which is still further elaborated in the blood, in the glands, and in the lungs, before it takes part in the general morphological changes connected with nutrition.

5. The observations of Dr. Acherson, of Berlin, and of Dr. Bennett, relative to how nutrition may be impeded by diminishing the *molecular* state of the nutritive elements, and improved by increasing them.

6. The observations of Panum and Parkes relative to the precipitation of albumen by acids and neutral salts, in which Dr. Parkes especially shows that the albumen as it exists in the serum of the blood is usually in that condition in which it is most easily precipitated by acids and chloride of sodium (*Med. Times and Gazette*, July, 1850 and 1852).

7. The excess of intestinal acidity in the alimentary canal of phthisical patients, as shown by Dr. Bennett, by which, under some conditions, the albuminous constituents of the food are rendered easily soluble, whilst the alkaline secretions of the saliva and the pancreatic juice are more than neutralized, and so rendered incapable of transforming the carbonaceous constituents of food into oil, or of so preparing fatty matters introduced into the system as will render them easily assimilable. Hence an increased amount of albumen enters the blood compared with fatty elements.

According to the observations of Mr. Jonathan Hutchinson, acid eructations were present in 62 *per cent.* of the cases of dyspepsia which preceded the deposition of tubercle, and were a prominent symptom in 46 *per cent.* of cases reported on by him in an admirable paper on the forms of dyspepsia preceding and attending phthisis (*Med. Times and Gazette*, vol. x., 1855).

Any one of these statements considered by itself does not seem of much importance; but considered as a whole, in their relation to nutrition and their influence upon morphological changes between the solids and the fluids of the body, they leave very little room for doubt that the bad habit of body in scrofulous affections associated with the growth of *tubercle-matter* must be established in the first instance through the digestive processes, as first described by the late Dr. Tweedy Todd under the name of *strumous dyspepsia*, and which has been since so fully described by the late Sir James Clark, Bennett, Hutchinson, and others.

The more closely these links of circumstantial evidence can be bound together, the more intimate a pathological relationship will be found to exist between the albuminous constituents of the food, the blood, and tubercle, and the saline constituents of the circulating fluid, the mal-assimilation of food, and the waste of the tissues, as connected with the development of the bad habit of body associated with scrofula.

Although it must be confessed that we do not fully understand the living processes by which all the molecular changes take place to which I have just adverted, yet there can be no doubt that in the seven statements enumerated we obtain some glimpses of a rational pathology which may yet tend to explain the very complex *constitutional morbid state* which precedes and is associated with the growth of tubercle in scrofulous affections, or with the specific inflammations and ulcerations of scrofula.

The *diathesis* which expresses the latent existence of the scrofula has been variously described by the terms *tuberculous, scrofulous,* or *strumous diathesis.* There is perhaps no subject in the whole range of medical science which the student ought to study more carefully than the cachexia, or special form of ill health, associated with the occurrence of tubercle and *scrofulous affections.* As a practitioner he will find that he becomes often painfully concerned in the deepest interests of families and society, through the threatened or actual ravages of scrofulous diseases. The extensive prevalence of the scrofulous cachexia—the great and almost inevitable mortality of the scrofulous diseases themselves, when completely developed, stamp the morbid state associated with them as a topic which at the outset of the student's career ought to engage a large share of study. Most assuredly the physician will have to turn his knowledge of the pathology of scrofula to account in every phase of his professional life; nay, further, when he knows, what experience has now adequately demonstrated, that the scrofulous cachexia springs from causes over which the public, rather than the medical profession, have control, he must be at once impressed with the belief, and encouraged with the hope, that when he acquires the confidence of the public in the practice of his profession, he may exercise a powerful influence for good in teaching how much the public may control the ravages of *consumption* by prudent marriages, sanitary attention to offspring, and the necessity of free ventilation and of fresh air in places inhabited by man.

There are several circumstances which show the great influence of public sanitary measures in controlling the development of scrofula, when these measures are scientifically directed to the preservation of the general health, and especially when men are associated together in great communities—an influence much greater than the best directed efforts of the medical profession can establish through their *materia medica.* It is by the mode of life as citizens of the world, in the social relations of husbands and wives, parents and children, and in the public relation of masters and workmen, that the extent and ravages of consumption and scrofula are to be controlled. It is by a strict attention to the rearing of offspring, and in the subsequent regulation of food, clothing, cleanliness, occupation, the choice of a profession, and by many other circumstances which have an obvious influence (perhaps at first sight inappreciable) on the maintenance of the general health, that our hopes of success as practitioners of medicine must rest in the prevention of that bad habit of body which develops and propagates the scrofulous diseases in civilized society.

When the late Sir James Clark, in 1835, published his treatise on pulmonary consumption, he expressed some doubts as to tuberculous diseases being comparatively more prevalent at the time he wrote than they were some fifty or a hundred years before. He was also of opinion that while many circumstances favoured the probability of a diminution of tuberculous diseases, there were circumstances which might materially counteract such an influence; and while he was convinced that tuberculous diseases had increased, in the middle and upper ranks of life at least, he believed that, as a rule, the constitutions of the three past generations had deteriorated progressively from father to son.

The annual returns of our Registrar-General up to 1846 show a progressive increase in the mortality from consumption. In 1854, however, we find it recorded "that phthisis is twice as fatal as any other disease in England, but that within the last eight years it appears to have declined to some extent." The inquiries of Dr. H. Greenhow show that this diminution is in a great degree due to those hygienic measures which have contributed to diminish the causes of *miasmatic* diseases in general; and which have especially lessened the prevalence of those *febrile exanthematous diseases* which, by weakening the constitution, tend to bring about those conditions under which that bad habit of body is established which leads to the growth of tubercles and the development of scrofulous affections. To no kind of sanitary measure are we more indebted for this result than to the influence of vaccination in diminishing small-pox—a disease which, of all others, seems to have tended to the development of the scrofulous cachexia as a sequel to its existence. Accordingly we find it recorded by Dr. Greenhow that "during the middle of last century, before vaccination was known, the scrofulous death-rate was more than five times as great as our present one; and the pulmonary death-rate of the present time is 7 *per cent.* lower than the pulmonary death-rate of 1746-55."

While, therefore, such statements and careful observations, extended through long periods of time, show how much may be done by general sanitary measures in preventing the extension of scrofulous diseases, there is still great necessity for a careful study of the nature of these diseases; for we find them in reality *decimating* the civilized part of the world, cutting off in some instances as many as 35 *per cent.* of our metropolitan populations, and a much larger *per centage* of the army.

Morbid Anatomy of Tubercle—The peculiar growth (erroneously called a deposit) which sometimes attends the diseases now under consideration is named *tubercle* from its external form, occurring as it does in small nodules, isolated or grouped together, or as large irregular masses, dispersed through the textures of an organ. One essential character of *tubercle* is its incapacity to development beyond the state in which it first becomes visible, and in which state it may remain latent. It generally, however, exhibits a tendency to degenerate in various ways, involving in such degeneration the destruction of the tissue with which it is surrounded. It contains no trace of fibrous development (ROKITANSKY).

The term *tubercle* is always understood to refer to adventitious masses of this nature, the type of which is found in the lungs as the essential anatomical constituent of pulmonary consumption. But the same material which composes *tubercles* in the lungs is also found in many different forms in other organs; and wherever it occurs it is described as *tubercle* or *tuberculous matter;* and *tuberculous disease,* or *tuberculosis,* is the usual designation of the specific malady of which the essential feature is the production of this peculiar matter (PAGET). A review of the opinions of recent writers, by Dr. Jenner, as to the nature of *tubercle,* in *The British and Foreign Medico-Chirurgical Review* for January, 1853, shows that the most eminent pathologists of the day are not at one as to the nature of this morbid product. "The opinions entertained regarding the nature of *tubercle,*" writes Sir William Jenner, "may be divided broadly into two classes." One class of pathologists holds that tubercle is an exudation essentially morbid in character (ROKITANSKY, BENNETT, ANCELL, LEBERT). Another class holds that tubercle is merely a retrograde metamorphosis of pre-existing structures, tissue-elements, or morbid products (WILLIAMS, REINHARDT, HENLE, GULLIVER, ADDISON).

Virchow may be said to hold a doctrine combining both views. For, while he holds that tubercles are essentially composed of dead tissue-elements, whether these are physiological or morbid products, he also holds that a local process in all cases leads to an exudation of a material which is poured out during what he terms "a tuberculous inflammation," and which becomes organized to a certain extent, and then dies, breaks up, shrivels, and so leaves a knot to which the name of tubercle is given. This process Virchow calls *tuberculosis;* and *scrofulosis* is the general constitutional state in which this *tuberculizing* process occurs, and which commonly leads to *tuberculosis;* or, in the words of Paget, "the relation between the two (terms) is, that the *scrofulous* constitution implies a peculiar liability to the *tuberculous* diseases." According to Virchow, *tuberculosis* is the local process in scrofulous affections in which there occurs an exudation of a material, nutritive or pathological, which develops into cells, and that these cells *tuberculize,* or undergo the tuberculous metamorphosis. *Tuberculization* is therefore the local process by which the metamorphosis of the elements of a part into tubercle is effected by endogenous development, atrophy, shrivelling, and dessication of its textural element.

A form of *tubercle* thus results out of the detritus of the metamorphosed and atrophied cells, with the remains of the vessels and other structures of the part in which they were seated (JENNER).

Whatever may be the view entertained regarding the exact nature of *tubercle,* this morbid product appears to us under two conditions, in forms more or less spherical, the contour of the masses being influenced —(1.) By the nature and movements of the surrounding tissue; and (2.) By the form of the part in which it first accumulates. The more recent and accurate microscopic observations which have been made into the nature and seat of the tuberculous deposit serve but to establish and confirm the more crude but scientific generalization made by the late Sir Robert Carswell, when he wrote that "the *free surfaces* of mucous membranes form the chief seat of tuberculous deposit." It is necessary, however, to extend the significance of the term *free surface*, and make it now apply to the ultimate and microscopic *cul de sac* terminations of mucous tubes.

Grey and *yellow* tubercle-masses are the names by which such deposits are described, and they are first visible in the form of roundish granulations about the size of millet-seeds, and isolated or in groups of nodular masses of more or less irregular form.

The *grey tubercle* is tough, soft, and compressible, of a pearly-grey colour, and semi-transparent. Microscopically, it is seen to be composed of irregular-shaped bodies, approaching a round, oval, or triangular form, and varying in size from $\frac{1}{4000}$th to $\frac{1}{2000}$th of an inch. These sometimes appear to be imbedded in a hyaline adhesive basis-substance, infiltrated with granules and molecules varying from a point scarcely measureable in size to the $\frac{1}{6000}$th part of an inch in diameter. The most characteristic semi-transparent grey granulations appear to contain more of the hyaline basis or connecting substance than of formed elements, the whole field of view being more transparent, and the elements less well expressed or defined. Acetic acid (weak, one part to four or six of water) dissolves many of the granules, and renders all the corpuscles more transparent, while a similarly weak solution of potash completely dissolves them.

Various opinions are entertained as to whether or not *tubercle-masses* ever consist of *tubercle-cells* with *nuclei.* Rokitansky holds that there are cells present containing one or more nuclei, and which indicate an endogenous development and growth of the elements just noticed. Gulliver and Vogel believe in the existence of such nucleated cells; Bennett, on the other hand, has never been able to discover nuclei in the corpuscles of tubercle. By many these cell-like elements are described and believed to be nuclei (Bennett, Schroeder Van der Kolk, Rokitansky, Paget, and Sieveking); and the view now related as entertained by Virchow, how existing tissue-elements may tuberculize, renders it highly probable that they may be the nuclei of epithelial cells, as suggested by Van der Kolk, or of other cell-elements, normal or pathological, as believed by Virchow; or they may be cell-particles, like nuclei, of slow formation and without any tendency to reproduction, but tending to disintegrate or break down into molecules, as described by Bennett. Paget enumerates the elements of tubercle as follows:—

> "1. Molecules, granules, and oil-particles, usually of small size and extremely predominant in *yellow tubercles.* 2. Nuclei of cytoblasts, of various shapes and structure, but all degenerate or defective; some glittering, hard-edged, wrinkled, and withered; others granular; and few or none with distinct nucleoli. 3. Nucleated cells, similarly misshapen, withered, or granular. 4. Certain compound cells, as described by Van der Kolk, and consisting of epithelium charged with the nuclei which become the common tubercle-corpuscles."

When the masses of grey tubercle exist in the substance of the lungs, their resemblance to millet-seeds has sometimes procured for the deposit the name of miliary tubercles; and when the lung is cut through, the elastic nature of its texture causes it to contract upon itself, so that the parenchyma recedes from the tubercle-deposits visible on the surface of the section, and the deposits appear slightly raised from the cut surface, and the finger may feel them as little resisting bodies set in the lung (PAGET). When examined with a moderately magnifying hand lens, the borders of these masses are seen to be irregular, with short projecting processes. The contraction of the lung and the consequent squeezing of the *tubercle*-mass, probably render these appearances mere secondary forms assumed by the grey granulations.

Yellow tubercle is of various shades of colour, and occurs in masses of variable size, generally larger than the *grey* deposits. These masses are opaque, friable, and of a cheesy lardaceous consistence. The masses of yellow tubercle are more commonly grouped so close together that the movements of the lungs cause them to become fused in uniform tubercle masses half an inch or more in diameter.

Microscopically, *yellow tubercle* contains a much greater abundance of fine molecules than the grey tubercle, and there are also present in it elements similar to those in grey tubercles, which are shrivelled, indented, and wrinkled. There are thus no positive or characteristic morphological elements in tubercle, and the pathologist is therefore obliged to make the microscopic diagnosis of tubercle by a process of exclusion or elimination. For example, if he finds that the substance he is examining consists of the elements just enumerated, and that there are *no blood-vessels nor blood-spaces* in its interior, and that there is nowhere in it any fibrous matrix enclosing cells, he is surely dealing with a tubercular formation. But, on the other hand, if he finds, besides these elements, a fine vascular provision for its nutrition and growth, with cells of a fully developed kind, and others degenerating, with blood-spaces, and having an areolar matrix-tissue, then he is most probably dealing with a malignant infiltrating growth, or a pneumonia in the lung, between the gradual invasion of which and tubercle there are points of analogy as well as of difference. The walls of the pulmonary artery are thickened in both; also the lining membrane of the bronchial tubes. In pneumonia, also, as well as where tubercle is being developed, the air-cells are increased in capillary action, followed by obstruction and breaking up of tissue.

The developmental origin of tubercle is to be sought for, in the first instance, as a growth from the elements of the surrounding tissue, nourished by the adjoining capillaries; and if an exudation of a fluid kind is present, its infiltration assumes the granular form of albuminoid elements, and is incapable of further growth, but capable of receiving a deposit of earthy salts in its substance, or of otherwise degenerating.

The fatty degeneration of tubercle seems to be a subsequent process, associated with a softer state of the mass in which the organic cell-elements still remain. To this oily or fatty degeneration is due the *yellow colour* of tubercle; and hence, also, this yellow form is regarded as a secondary form to the grey granulation. It is in reality a degeneration or retrograde change. And this oily degeneration is not so favourable a local change as that by which the tubercle already formed wastes and *dries up*, while its further development ceases, and the mass assumes a cartilaginous-like consistence. At the same time calcareous salts are deposited in the mass, which then hardens or cretifies.

In the *grey tubercle* this salutary change is uniform throughout the mass; in the yellow tubercle the calcareous salts are usually found in larger amount round the peripheric portion of the tubercle, closing in a pultaceous friable substance, composed of free fat-granules, aggregated globules of fat, brown or black pigment-masses, and plates of cholesterine.

Vogel, Wedl, Virchow, and several other first-rate observers, are agreed that "an organized new formation constitutes the basis of the pathological structure known as tubercle."

To verify this, the study of tubercle as a morbid growth ought to be commenced with the development and growth of the new structure in the serous membranes, as in the arachnoid or pleura. In these textures we are not so apt to regard half-destroyed tissues as new products—a risk we always run in the examination of pulmonary tubercle.

When thus examined in its early state, tubercle takes the form of a granule or a knot, as Virchow has demonstrated (fig. 1.) from examination of tubercle, commencing in serous membranes like the pleura and arachnoid. The knot constitutes a new formation of cellular development, having its first beginnings in the connective tissue, and appearing, *ab initio*, in the shape of a granule. The special anatomical characteristic of the new growth is its extreme richness in nuclei. By isolating the constituents of the young tubercle-knot, either very small cells with one nucleus are obtained—so small that the membrane closely invests the nucleus—or larger cells with multiple division of the nuclei, so that 12, 24, or 30 are contained in one cell. The nuclei are always small, and have a homogeneous glistening appearance. The elements of scrofulous tubercle are relatively the smallest of any histological formation. It is only at its commencement it is pervaded by blood-vessels. When it enlarges, the little cells are so closely pressed together, that the blood-vessels are

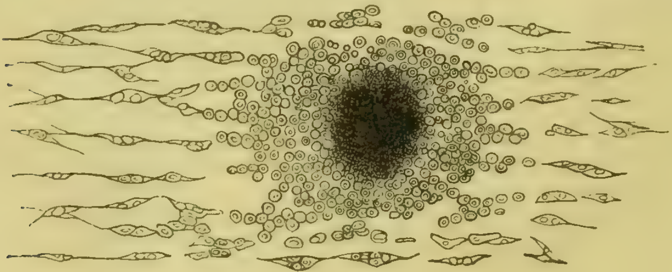

Fig. 1.*

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* Development of tubercle from connective tissue in the pleura. The whole succession of transitions is seen from the simple connective-tissue corpuscles, the division of the nuclei and cells, up to the production of the tubercle-granule, the cells of which in the middle are disintegrating into fatty granular debris. 300 diameters. (After VIRCHOW).

gradually obliterated, and only such large ones remain as traverse the tubercle altogether. Fatty degeneration very soon sets in at the centre of the growth, where the oldest cells exist; and when all trace of fluid disappears, the cells of the new growth begin to shrivel, the centre of the tubercle becomes yellow and opaque, and a yellow spot is seen in the midst of the grey translucent granule, knob, or tubercle. This is the commencement of the so-called *cheesy metamorphosis.* This change advances from without, outwardly progressing from cell to cell, till the whole tubercle-granule is gradually involved in the change. The elements of tubercle at last lie so closely together that the growth destroys itself; inducing, by pressure, the disappearance of the blood-vessels which supply its nutrition. The tubercle then begins to die away, and nothing remains but debris, in the form of the shrunken cells, and cheesy matter,—a condition common alike to *pus, cancer,* and *sarcoma.* The nature of tubercle cannot be learned after it has reached this stage. It is only at the early period, when engaged in proliferation, that its anatomical nature and structure can be determined. The histological elements of tubercle resemble those of the lymphatic glands more closely than they do any other normal element of the body, (*Cellular Pathology,* Lecture xx.)

Few objects of morbid anatomy have been submitted to more minute research than "tubercle;" and there is scarcely any organ where the growth may not occur. Considerable differences of opinion have been entertained respecting the relation of the two varieties of tubercle which have been described. Laennec taught, and it has been the general belief since his time, that the *grey tubercle* is the earliest stage of the deposit which subsequently becomes converted into *yellow.* Dr. Walshe has also investigated this point, and has taught a similar doctrine. Rokitansky, in the first edition of his great work on morbid anatomy, regarded the *yellow* and the *grey* as essentially distinct forms, and considered it an error to believe that the one is converted into the other. He now affirms that *grey tubercle* is sooner or later converted into *yellow tubercle,* as Laennec first taught, and which is now the common belief. When the grey tubercle does not pass into the yellow, it withers away. It loses its lustre, and becomes dry, dense, and hard, and shrivels into an indistinct shapeless fibrous-like mass. Sometimes such a change is associated with a calcareous degeneration, the surrounding tissue in which it is imbedded becoming dark with the deposit of pigment.

It is now believed and taught by Rokitansky, Virchow, and others, that certain abnormal products, not apparently at first tubercular, afterwards *tuberculize,* and assume the appearance of yellow tubercles. To this change Virchow proposes to restrict the term "*cheesy metamorphoses.*" The metamorphosis of *grey* to *yellow* tubercle generally commences in the centre of the growth, and subsequently the yellow tubercle undergoes most important secondary changes in a sanative point of view. These changes consist—(1.) in softening; (2.) in cretification or calcification; but it is not to be understood that the first of these two changes always passes into the second.

The descriptions already given, and the metamorphosis about to be noticed, illustrate the distinctive and peculiar characters of tuberculous growth—namely, its early degeneration and abortiveness. It seems to be a substance which acquires a certain stage of organic development, when it is arrested in its course, recedes, and degenerates. Such characters may be read from the shrivelled or granular state of the so-called free nuclei and the barren cells. The changes about to be noticed show a still more retrograde decay (PAGET).

When tubercle softens, its substance breaks down into a tolerably uniform creamy pus-like fluid, in which are to be seen an immense number of fine granular points or molecules. The basis-substance appears to soften first; but in some kinds of softening much of the corpuscular forms are retained in a thin, whey-like, flocculent fluid, approaching in character to what is known as scrofulous pus, both microscopically and to the naked eye. The softening appears, on the whole, due to the breaking up of the corpuscular elements, as well as of the basis-substance. When softening begins in the centre of the yellow crude masses, it leads one to believe that fluid may transude from the surrounding effusion or hyperæmic state, and penetrate to the centre of the whole mass, where it may accumulate and increase the softening, without becoming developed into the visible cell-elements of tubercle. As each tubercle or group of tubercles undergoes this softening process, the softened mass occupies a cavity, which thus becomes an abscess, though not a purulent one, and the tissue involved by the growth of the tubercle is destroyed. It dies with the increased growth and softening of the mass. During this process of softening, tuberculous matter continues to grow in other portions of the organ, and when it occurs in the lung, generally from above downwards, so that excavations or cavities are found at the apex of the lung, while lower down yellow tubercle-masses, beginning to soften, exist; and still lower down the *yellow tubercle* is found in a crude state, as if newly changed, or in the process of being changed, from the grey granulations which are disseminated through the base of the lung.

Pathologists are now agreed that the production of tubercle is quite independent of inflammation, in the ordinary acceptation of that term, implying thereby the presence of pain, heat, swelling, redness, lymph, or pus; but that in the great majority of cases inflammation is set up round the tubercle-masses, and plays an important part in promoting further growth of tubercle, which proceeds in a rapid course to softening and destruction of tissue. The phenomena of the growth of tubercle are also associated with the existence and supply of material which forms the fibrous thickenings and capsules around tubercular cavities or ulcerations. These phenomena also bring about the separation of the tubercle-mass from the surrounding parts, and if it is eliminated in any way, a cavity or an ulceration remains. When thus enclosed by fibrous thickenings or capsules, the softened tubercle may undergo the process of *cretification* or *calcification;* in other words, it becomes converted into a greasy, fatty, chalky mass, which gradually becomes hardened into a brittle substance. There appears to be either a deposit or a liberation of calcareous particles in the mass, for the elements become mixed up with gritty particles of earthy salts; and at the same time there is absorption of the animal part, so that the original size of the tubercle-growth is diminished. When tubercle is thus completely calcified, a thin section presents a granular appearance, but with no definite forms, combined with more or less pigment.

Tubercle has been analyzed by many chemists. The analysis by the late Dr. R. M. Glover, of Newcastle, appears to be the most accurate and extensive.* His conclusions are as follow:—

> "1. That tubercle consists of an animal matter mixed with certain earthy salts.
>
> "2. That the relative proportion of these varies in different specimens of tubercle. That animal matter is most abundant in recent, and earthy salts in chronic tubercle.
>
> "3. That the animal matter certainly contains a large amount of albumen, while fibrin and fat exist in small but variable proportions.
>
> "4. The earthy salts are principally composed of the insoluble phosphate and carbonate of lime, with a small proportion of the soluble salts of soda.
>
> "5. That very little difference in ultimate composition has yet been detected between recent tubercle and the other so called compounds of protein."

Thus much is known regarding the histology of tuberculous growths, and is here stated minutely, because we have the testimony of most experienced morbid anatomists, such as Bennett, Paget, and many others, that the microscope alone can decide as to the nature of those growths which closely resemble tubercle when examined by the naked eye. Indeed, such are the vague definitions given of *tubercle*, that, in the words of Bennett, "every morbid anatomist must frequently have experienced much difficulty in endeavouring to determine by the naked sight whether a certain morbid product be or be not tubercle."

The term "tubercular deposit" can scarcely be regarded as correct, although in common use. The expression ought rather to be "*scrofulous* or *tuberculous inflammation;*" just as we have been accustomed to use "*tubercular meningitis*" as synonymous with "*scrofulous or tuberculous inflammation of the meninges.*"

In the *peritoneum* the seat of scrofulous inflammation is immediately beneath the serous coat, where sanguineous congestions and minute extravasations first take place during the rapid formation of the tubercular granulations.

In the *mucous membrane* of the alimentary canal the seat of scrofulous inflammation is principally in the lower part of the small intestines. The tubercle grows originally in the submucous tissue, and elevates the mucous membrane stretched over the granulations. They project in the form of nodules assembled together in several groups. New granulations spring up between the older ones; and as these growths degenerate and soften rapidly in the intestines, a fusion of the whole very soon takes place. The circulation in this part of the intestine is impeded, and its vitality is gradually destroyed; its mucous membrane is lost, and ulcerations form, whose shapes are decided by the form of the original growth. Thus:—A single tubercle-nodule gives a crater-like ulcer. A number produces extensive ulceration, with irregularly excavated borders, the growth of the tubercle-masses being continued at the margins of the ulcers; and hence the thick border, with particles of yellow crude tubercle imbedded in it, so characteristic of these ulcers. Similar material may also be seen on the floor of the ulcers, the softening of which leads to perforation of the peritoneum.

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* This analysis, to be satisfactory, should be made on tubercles at different periods of life. The tubercle of the *young* strumous subject is believed to differ greatly from the tubercle-growth found in *advanced life*, especially in a gouty habit.

A considerable portion of the scrofulous exudation may transude in a fluid state, in the first instance, through the capillaries, and collect in those places outside the vessels that offer least resistance (BENNETT). While, therefore, *infiltration* more or less extensive, as described by Baillie, is the first condition in which the exudation can be observed to exist, *tubercle* may be observed to accumulate on the free surfaces of mucous and serous membranes, or on the outer surface of minute blood-vessels. While, therefore, scrofulous material does not differ in its seat from the simple or cancerous exudations, when these form minute growths in the lungs or other parts, yet there is an important difference in regard to their relation with the blood-vessels which requires to be specially noticed. It was first pointed out by Dr. William Stark that when attempts are made to inject a tuberculated lung, the finest injection will not reach, far less penetrate, the *tubercle-masses.* Blood-vessels which are of a considerable size at a little distance from tubercle-masses speedily become contracted, so that a large vessel, which at its origin measured nearly half an inch in circumference, could not be cut open further than one inch; and when cut open, such vessels presented a very small canal, filled by a coagulated substance. Schroeder Van der Kolk made similar experiments. He sometimes found that large vessels remained pervious which crossed a tuberculous cavity in a lung, but that all the small or capillary branches which adhered and were given off from the larger trunks were obstructed and impervious. These observations lead to the conclusion that obliteration commences in the smaller vessels and proceeds to the larger trunks, and are of some importance with reference to hæmoptysis and to the process of softening, and other changes which are observed to take place in this *non-vascular* growth. The process of *softening* is not so uniform in its progress as the notice already given might lead one to suppose.

Cancerous growths, on the other hand, are highly organized, containing large, voluminous, and generally multiform elements.

It appears difficult in every instance to ascertain at what part softening commences, so that it is incorrect to give a general description of this change as always commencing in one place. In one case it may begin in the centre and proceed to the circumference of the growth; in another it may begin at the circumference and go round the whole mass, detaching it from the surrounding textures; in a third case it may begin at once in the centre and at the margins; and in other instances it has been observed to commence at the same time in several parts of the substance of the tubercular mass; and this is especially the case where the masses are large (CRAIGIE, BENNETT). The portion or spot about to soften loses its firmness and becomes friable, the cells swell and break up, adding thus by their solution to the diffluency of the mass. Inspected with a common hand lens, the mass seems as if perforated here and there with holes, and this softening extends by degrees throughout the whole growth. The air-vesicles necessarily are destroyed by this process, and the terminal extremities of the bronchi are amongst the first structures to suffer. The mucous membrane which covers them is red and villous in the vicinity of the softened part, and as tuberculosis proceeds, the whole of the pulmonary mucous membrane presents the anatomical characters of chronic bronchitis. The softening of the growth and the inflammation of the textures seem to act and re-act upon each other; liquefaction seems to extend more rapidly when local inflammation is set up, and the softenings, on the other hand, appear to augment the local re-action.

Healing of Local Lesions in Scrofula.—It is of the greatest importance to study how some of the most unmanageable and hopeless diseases will sometimes spontaneously become cured, and how the local lesions will heal. That scrofula sometimes tends to these results we have abundant testimony daily afforded in almost every *post-mortem* examination of those who die of *phthisis pulmonalis.* Laennec, Carswell, Clark, and Bennett have recorded their testimony on this interesting point; and the practical result of their observations shows that if the further growth of tubercle can be arrested, the masses already existing may silently retrograde, become absorbed, or diminish in size, and the part cicatrize, or it will remain latent as a cretaceous mass in the lung.

Symptoms of Scrofula, or of the Cachexia which Precedes and Accompanies the Growth of Tubercle.—Ever since scrofula has become more studied as a constitutional affection, it has been rendered more apparent that impairment of the digestive organs is the primary disorder of function which ushers in the cachexia, and that a certain form of dyspepsia is not only present in the hereditary strumous constitution, "but is capable of generating the bad hăbit of body, and of leading ultimately to the deposition of tubercle."

It was first observed by Dr. Wilson Philip "that there were some forms of indigestion which ended in phthisis;" and it was subsequently distinctly stated by Lepelletier that the causes of scrofulous disease were referable—(1.) To those agents which impair the assimilative action; (2.) To the elements of nutrition being insufficient, by the influence of bad diet, impure air, deficient exercise of the general functions, and obstruction of the functions of excretion.

It was reserved, however, for the late Dr. Tweedy Todd, as already noticed, to show that in cases in which the strumous cachexia was present a particular form of dyspepsia prevailed, to which he gave the name of "strumous dyspepsia." Subsequently the views of Dr. T. Todd were more or less fully accepted and developed by Sir James Clark, Bennett, Ancell, and others in this country; but the most precise and most recent exponent of them has been Mr. Jonathan Hutchinson, in two valuable papers in *The Medical Times and Gazette*, vol. x., 1855. Mr. Hutchinson, from statistical evidence, shows that the tuberculous cachexia is preceded by a *peculiar form of dyspepsia;* in other words, that there is a peculiar form of dyspepsia which has a natural tendency to end in phthisis; and that it is a form of dyspepsia not common to other conditions of the system—different, for instance, from that which precedes and attends gout, chlorosis, or cancer, although accurate observation is wanting on the exact symptoms of dyspepsia which are associated with these diseases.

The conclusions which have been arrived at by Mr. Hutchinson are as follow:—

1. In a very large majority of cases of established phthisis a condition of well-marked dyspepsia is present as a complication. Out of fifty-six cases it was absent in four, present mildly in twenty-one, moderately in twenty-two, and severely in nine.

2. Of the form of dyspepsia most common in established phthisis, the prominent symptoms relate to difficulty in the assimilation of fatty matters. The patient acquires a remarkable distaste for all fats, which occasionally extends itself to sugar and even to alcohol; he suffers much from "biliousness," heartburn, flatulence, and, above all, from acid eructations after taking food; everything he takes "rises acid," to use a common expression of such patients, but more particularly everything containing fat, oil, or sugar (*butyric* fermentation).

The *dislike for fat* was present in 71 *per cent.* of the cases of confirmed phthisis, and had existed through life in 48 *per cent.* It invariably produced what is commonly called "biliousness," and "rose acid" from the stomach. The fat of fresh meat was generally the first to disagree, then salted meats, such as bacon, and lastly butter; but many could enjoy butter who could not touch any other kind of animal fat. Such an intense dislike to an important element of a mixed diet indicates a deficiency on the part of a patient to digest it. Coincident with the development of such symptoms *emaciation* usually becomes apparent, the adipose tissue of the body already existing being re-absorbed by the blood to supply the respiratory element deficient in that fluid. The fat disappears from the subcutaneous tissue, hollows of the cheeks, orbits, and mammary glands, and other parts where it generally abounds in quantity, just as it disappears from the body of an animal during hybernation. The patient now gradually loses weight and becomes thinner. Other tissues of the body, such as the muscles, skin, and areolar tissue, subsequently begin to be used up for a similar purpose, till the patient is wasted to a living skeleton.

The *acid eructations* were present in 62 *per cent.* of the cases, and were a prominent symptom in 46 *per cent.*, small quantities of an extremely acid fluid being repeatedly brought up into the pharynx at various periods after taking food, and in bad cases these eructations were persistent throughout most of the day, and almost always attended by heartburn. *Sick headaches* and *biliousness* were very commonly present.

Dr. Pollock's experience shows that *acidity* of digestion has only a constant relation to the acute form of the disease. Of that form it is a distinctive character. Laennec, Louis, and Pollock all record diseased states of the stomach in *post-mortem* examination of phthisical patients.

3. The majority of cases of *phthisis pulmonalis,* whether hereditary or otherwise, are *preceded* by a well-marked stage of dyspeptic symptoms. By this it is meant that symptoms referable to the digestive organs have

preceded those connected with the lungs. Out of the fifty-two cases in which, as has been shown, dyspepsia was present, it had followed pectoral symptoms in nine, was developed nearly coincidently with them in ten, and preceded them in thirty-three.

4. The symptoms of the dyspepsia premonitory of phthisis are the same in character with those which complicate it when developed. The very earliest are alterations in the tastes, and the most constant of all is a disrelish for fat.

5. The subjects of phthisis have in a large number of cases had peculiarities of likes and dislikes for different articles of food even from very early life, and whilst seemingly in perfect health. Amongst those peculiarities the dislike of fat, often amounting to extreme aversion, ranks first. Thus it might be predicated of a family in which one child distinguishes itself from its brothers and sisters by constant refusal to eat fat, that such a child will, *cæteris paribus*, be more prone to become the subject of tuberculous disease in after life than any of the others.

Besides the indications afforded by the functions of digestion, there is a peculiar modification of the whole organization, as regards structure, form, and the exercise of functions generally, which impresses distinctive characters on the scrofulous cachexia when it is of hereditary origin. Miller gives the following concise description of this organization:—

"The complexion is fair, and frequently beautiful, as well as the features. The form, though delicate, is often graceful. The skin is thin, of fine texture; and subcutaneous blue veins are numerous, shining very distinctly through the otherwise pearly-white integument. The pupils are usually spacious; and the eyeballs are not only large but prominent, the sclerotic showing a lustrous whiteness. The eyelashes are long and graceful—unless *ophthalmia tarsi* exist, as not unfrequently is the case; then the eyelashes are wanting, and their place is occupied by the swollen, red, unseemly margin of the lid.

"In the phlegmatic form the complexion is dark, the features disagreeable, the countenance and aspect altogether forbidding, the joints large, the general frame stinted in growth, or otherwise deformed from its fair proportions. The skin is thick and sallow; the eyes are dull, though usually both large and prominent; the general expression is heavy and listless; yet not unfrequently the intellectual powers are remarkably acute, as well as capable of much and sustained exertion. The upper lip is usually tumid, so are the *columna* and *alæ* of the nose, and the general character of the face is flabby; the belly inclines to protuberance; and the extremities of the fingers are flatly clubbed, instead of presenting the ordinary tapering form" (*Principles of Surgery*, p. 55).

Canstatt has given the following description:—"An unusually large head, coarse features, a thick chin, swollen abdomen, enlarged cervical glands, and flabby, spongy flesh. The skin is of remarkable whiteness, with a tendency to redden easily, and through which the rose-pink or blueish subcutaneous veins are visible, a deep redness of the cheeks and lips, blueness of the thin and transparent sclerotic, which imparts a swimming and languishing look to the eyes. The muscles of such persons are thin and soft, and their weight is light in proportion to their stature, indicating a slightness of their bones. The teeth are handsome and of a blueish lustre, though long and narrow; and the hair is soft."

The growth of the body is generally unsteady in its progress; very often it is slowly and imperfectly developed; in other cases it is unusually rapid, particularly towards puberty. The physical powers are generally feebly developed, and incapable of sustained exercise. The muscles of the limbs, though full, are soft, flabby, and weak, and have neither the form nor the firmness of health. The general circulation is feeble, the weak pulse and cold extremities indicating the debility. Digestion is feebly and imperfectly performed, the bowels being irregular, and more frequently slow in their action than the reverse.

The mucous membranes generally are very susceptible to disordered action. Discharges from the nose, ears, or eyes are not uncommon; the tonsils enlarge, and the air-passages inflame from the slightest causes. The insensible perspiration is defective, and is said to be unduly acid, and loaded with sebaceous matter; while, on the other hand, copious partial perspirations are common, particularly on the feet, where the odour is often fetid.

In children, the impaired functions of the digestive organs are indicated by increased redness of the tongue, especially toward the extremity and along the margins. The anterior part is thickly spotted with small red points of a still brighter colour, the central portion being more or less furred, of a white or of a brown colour, in the morning. Thirst prevails; the appetite is variable, more frequently craving than deficient, seldom natural; and the breath is fetid. The bowels are occasionally loose, and the evacuations are always unnatural, generally of a pale greyish colour, of the consistence and appearance of moist clay; and they are often mixed with mucus and partially digested food. While the urine is often turbid, and sometimes high coloured, it is also often abundant and pale. The skin is generally harsh and dry, or subject to cold perspirations, particularly the hands and feet, which are habitually cold; and copious partial night sweats are common. The sleep is seldom sound; the child is restless, talks in his sleep, or grinds his teeth (T. TODD, CLARK).

Subsequently, when the disordered state has continued for some time, the internal fauces become full and red, and inflammatory sore throats are common, the tonsils often becoming permanently enlarged. The nostrils are generally dry, or thick mucus may be discharged in large quantity. Epistaxis occasionally occurs.

The nervous sensibility is sometimes greatly increased, and the intellectual functions are often performed with a preternatural degree of activity. So frequently is this observed, that it has become a popular belief and saying regarding such a child, "that it may be too wise to live long."

Inflammation in any tissue in a scrofulous subject generally assumes a slow chronic type, accompanied with little pain or heat; and suppurating parts heal very slowly.

When the scrofulous cachexia becomes fully developed into *phthisis pulmonalis* the duration of the disease ranges from about nine months to two years; but in what are called "acute" cases of consumption, it may terminate fatally in three or even in two months, and occasionally in as short a period as three weeks, or even less.

The most reliable results of the duration of phthisis are those given by Dr. Pollock. His experience shows that it lasts much longer than has been generally believed. Early invasion, subsequent subsidence of symptoms, and long tolerance of the deposit obtain in many cases, and thus show an undoubted tendency to extreme chronicity. Since greater accuracy in diagnosis has prevailed, consumption has been known to last much longer,—the average duration, out of 3,566 cases observed by Dr. Pollock, being *two years, six months,* and *three-fifths nearly,* including cases of the most acute form.

Causes.—In describing the pathology and the symptoms of the scrofulous cachexia, the cause of the constitutional disorder has already been in some measure indicated.

The tendency to the formation of tubercle in scrofula is not equally great at all periods of life, nor in all parts of the body. Tubercle in the bronchial glands, the lungs, the cervical glands, the mesenteric glands, the spleen, the pleura, the liver, the small intestines, and the brain, is most common in infancy, childhood, and early adolescence. But the occurrence of tubercle, which forms so large a portion of the lesion in all scrofulous affections, although it has been found in the fœtus, and at every period of life up to eighty, yet it will be observed that tubercle, after the age of fifteen, is more frequently met with in the intestines, the mesenteric, cervical, and lumbar glands. Scrofulous disease is frequently developed during infancy; and "I have met with several cases of infants," writes Sir James Clark, "dying of consumption within the first year of life, in whom the lungs were not only extensively tuberculous, but contained large caverns, with all the characters of those found in the lungs of adults." After the second year of life, pathologists agree as to the universal prevalence of scrofulous disease. Age exercises a paramount influence on the generation of scrofula. It is peculiarly a disorder of childhood and youth (GUERSENT, LOMBARD, PAPAVOINE, ALISON, CLARK, BENNETT). Dr. Alison demonstrated that the mortality from scrofulous diseases in the children of the lower orders in Edinburgh and other large towns was so great that they died in the enormous proportion of *forty-five* or *fifty* to *five* and even *three*, as compared with the agricultural and upper classes. Tubercles prevail most through the third, fourth, fifth, and sixth years, when the annual growth does not exceed one-tenth of the child's weight, and the mortality declines to nearly one in a hundred. More than a fourth of those who die in the interval from birth to puberty are affected with scrofulous disease; yet the disease itself causes death in about one-sixth only of the cases (CLARK). The greatest number of deaths occur between the ages of twenty and thirty; the next in proportion between thirty and forty; the next between forty and fifty. The mortality is at its maximum about thirty, and from that age declines.

The broadest fact established regarding the exciting cause of scrofula is, that the domesticated animal is more liable to scrofulous disease than the same animal in a wild state. The stabled cow, the penned sheep, the tame rabbit, the monkey, the caged lion, tiger, or elephant, are almost invariably cut off by scrofulous affections—no doubt due to deficient ventilation and the abeyance of normal exercise of the pulmonary function.

On this point Dr. Clymer has drawn up the following appropriate results:—

> "There appears to be little doubt that persons who breathe continuously air vitiated by respiration furnish a large percentage of phthisical cases. This cause is potently aided by the coincident conditions of deficient exercise, poor feeding, and, often, excessive work. Baudelocque, years ago, asserted that impure air was the chief cause of phthisis. Carmichael, in his work on *Scrofula* (1810), gives a number of examples of the influence of foul air and want of exercise in the production of the disorder. Lepelletier, Neill Arnott, Toynbee, Guy, Greenhow Baly, and others, have collected a good deal of evidence in support of this cause of tuberculosis. Of 104 compositors who worked in rooms of less than 500 cubic feet for each person, 12·50 per cent. had had hæmoptysis; of 115 in rooms having from 500 to 600 cubic feet, 4·35 per cent. had had hæmoptysis; and in 101, in rooms of more than 600 cubit feet, 1·98 had had hæmoptysis (GUY). In the prison of Leopoldstadt, at Vienna, very badly ventilated, in the years 1834-1847, 51·4 per 1,000 died from phthisis, there being 42 cases of acute tuberculosis; while in the well-ventilated House of Correction, in the same city, the deaths from phthisis were only 7·9 per 1,000. The great prevalence of phthisis in the Indian jails is ascribed to bad air and bad food, (Parkes, *loc. cit.*, p. 96, 2d ed.) Dr. Henry MacCormac is, perhaps, the most enthusiastic advocate of the doctrine of the effects of re-breathed air in the etiology of tuberculosis; so far does he carry his doctrine that he makes it almost an exclusive cause of phthisis. 'Wherever the air, habitually respired, has been respired in whole or in part before, there tubercular deposits are found.'* The greater relative frequency of phthisis in females Dr. Lawson attributes to their indoor occupations and sedentary habits.† The chief cause of the extraordinary frequency of phthisis in the European armies, to be presently spoken of, Dr. Parkes, says 'can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault. This is the conclusion which the Sanitary Commissioners for the British Army came to in their Report, after assigning all probable influence to exposure on duty, intemperance, syphilis, and faulty diet. The disease is found to prevail in the most varied stations of the army, and in the best climates, but where there was always the one common condition—the impure air of the barrack system. And, as if to clench the argument, there has been of late years a most decided decline in phthisical cases in these stations (Gibraltar, Malta, Ionia, Jamaica, &c.); while the only circumstance which has notably changed in the time has been the condition of the air. So also the extraordinary amount of consumption which prevails among the men of the Royal and Merchant Navies, and which, in some men-of-war, has amounted to a

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* *Consumption, as Engendered by Re-breathed Air*, &c., 2d ed. London, 1865.

† *A Practical Treatise on Phthisis Pulmonalis.* Cincinnati, 1861.

veritable epidemic, is in all probability attributable to the faulty ventilation' (PARKES). The statistics of the Hospital for Consumption and Diseases of the Chest, London, show that among 3,214 men, who composed all the cases of decided phthisis which were registered as in-patients in ten years, more than one-half—1,812—had followed indoor occupations; and of the 2,413 females nearly all had.

TABLE SHOWING THE OCCUPATIONS OF 5,627 PERSONS OF BOTH SEXES AFFECTED BY PHTHISIS.*

MALES.		MALES.	
Bakers, ...	64	Various, ...	49
Bookbinders, . . .	17	Mechanics, . . .	176
Bricklayers, . . .	109	Painters, ...	105
Butchers, ...	3	Printers, ...	103
Carpenters, . . .	295	Publicans, ...	46
Clerks and Shopmen, .	394	Railway, ...	38
Coachmen, . . .	211	Sailors and Watermen, .	74
Gardeners, . . .	82	Servants, ...	285
Labourers, . . .	539	Shoemakers, . . .	171
Smiths, ...	89	Soldiers and Police, . .	103
Teachers, ...	42	Other Trades, . . .	150
Tailors, ...	145	At Home, ...	278
Weavers, ...	11		
At Home, ...	63	Total, ...	3,642
FEMALES.		FEMALES.	
Servants, ...	984	Governesses, &c., . .	80
Domestic, ...	447		
Milliners, ...	397		
Laundresses, . . .	77	Total, ...	1,985

"Dr. Pollock, one of the physicians, states that the occupation of out-patients would give the same result. It must then be admitted, from overwhelming testimony, 'that deficient ventilation and crowded apartments are eminently productive of tubercular disease.' Those trades which require a stooping posture—as tailors, boot-makers, weavers—which by mechanical hindrance to the free entry of air into the lungs, restrict the expansion of the chest-walls, and result in imperfect performance of the respiratory function, and consequent interference with the proper nutrition of the pulmonary tissues, and general lessened vitality of the system, furnish, no doubt, a large proportion o cases of tuberculosis. The influence of lessened breath-motion as an exciting cause of phthisis has been particularly insisted on by Dr. Edward Smith, in his excellent work on *Consumption, its Early and Remediable Stages.*"

Hirsh also has collected a large number of facts, proving that in foundling hospitals, orphan asylums, boarding schools, factories, and similar institutions, the continual abode in a badly ventilated atmosphere, saturated with steam, and poisoned by animal effluvia, and the products of putrefaction, is in the highest degree favourable to the development of scrofula; and that, according to the experience of these institutions, where there is no lack of cleanliness, good food, and warm clothing, the above pernicious agents alone suffice to induce the disease (quoted by Niemeyer).

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* Second Medical Report of the Hospital for Consumption and Diseases of the Chest, 1863.

Bowditch, in America, ascribes the most efficient development of scrofula to moisture and damp.

The disease springs most frequently, however, from the combined effects of all these different anti-hygienic influences, the chief of which is bad nourishment and want of fresh air.

Parents whose digestive organs are habitually disordered, who suffer from gout, the injurious influence of metallic and other poisons, such as *mercury*, *malaria*, *syphilis*, *chlorosis*, or *paludal anæmia*, *the debility of advanced age*, so that a cachectic state is induced, entail upon offspring begotten during such morbid states of the parent, to an extent still undetermined, but obvious and appreciable, a tendency to the scrofulous constitution, not necessarily to be expressed by the growth of tubercle in the lungs, but certainly and surely by various forms of dyspepsia and a precarious state of health.

There are many circumstances in the state of the parents' health, presumed, with justice, to influence the health of the children born under them. Among these may be mentioned a disordered state of the health of the mother, interfering with the state of the fœtus *in utero*, depressing passions, and generally an unhealthy mode of life. "In the present state of our knowledge," writes Sir James Clark, "it is not possible to determine the various circumstances in the health of the parent which may give rise to scrofulous growth in the child, much less to explain the development of scrofula. I rather allude to them as subjects deserving the investigation of the general pathologist and practical physician. There may be differences of opinion as to the particular condition of the parent which induces the tuberculous constitution in the offspring, and also as to the degree in which this constition may exist in the child at birth."

Let the topics suggested in this quotation be diligently inquired into by those who have the opportunity; and let the initiative be followed up, so ably set forth by Dr. Walshe in the model paper from which the previous statements have been quoted.

In the experience of Dr. Pollock, the disease was hereditary in 362 or 30·16 *per cent.* of all the cases. Of the remote causes of tuberculosis he writes:—

> "Of all of these, doubtless the most important is hereditary predisposition; for, in its absence, how many struggle through influences and errors which destroy others in early life, and attain a considerable longevity? Not all the predisposing causes united could, in a given instance, induce consumption with certainty, without some subtle agent to precipitate, concentrate, and shape those elements of disease into tubercle. . . . In the absence of more accurate knowledge than science yet possesses, we are inclined to refer the actual precipitation of the disease which we call tubercle to the influence of hereditary predisposition acting in conjunction with, or occasionally even in the absence of, all or any of the other remote agents which give rise to phthisis. . . . If to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease. . . . Finally, the forms of disease, those modifications on which depend its rapidity, development, and duration, are most distinctly reflected in families. How often do we witness child

after child carried off at the same age by the same variety of tubercular disease. Tuberculosis, though not present in the parents, may have an ancestral origin generations back."

The concurrent opinions of the entire profession now fully establish the fact that the tuberculous predisposition is capable of transmission from parent to offspring.

The scrofulous constitution has been observed to manifest itself in the child under the following circumstances:—

1. At birth tubercles may exist in one or more organs (CHAUSOIER, OEHLER, HUSSON, BILLIARD).

2. The scrofulous cachexia, already defined and described, may rapidly show itself.

3. By the rapid occurrence of tubercles commencing very soon after birth, subsequent to the gradual appearance of symptoms of the scrofulous cachexia.

4. By a disposition to the various forms of dyspepsia, whose characters have been already noticed (page 16, *ante*).

Race has an influence in the production of phthisis. In this country the tendency of the Creole and Negro to phthisis is notorious. But it is notorious also that the Creole and the Negro, when removed beyond certain limits of the land of their nativity, become tuberculous in other lands besides Great Britain. In the West Indies some of the black races are by no means exempt from this disease, and the Creoles die of phthisis in large numbers in Martinique (RUFZ, NOTT, MORTON). This is the more unlooked for, because as children they live almost in the open air, bathe daily, or still more frequently, and are singularly cleanly in their persons.

"Among the predisposing causes of phthisis," writes Laennec, "I know of none more certain than the *depressing passions*, especially when they are profound and long indulged; and this perhaps is the cause of the greater prevalence of this disease in larger towns, where bad habits and bad conduct are more common, and often the cause of those bitter regrets which neither time nor consolation can assuage." He adds, "I had under my own eyes for ten years a most striking example of the influence of melancholy in the production of phthisis. There existed in Paris for that space of time a nunnery of a new foundation, and which had not been able to obtain from the ecclesiastical authorities anything but a temporary tolerance, on account of the severity of its rules. The alimentary regimen of the nuns, although extremely severe, was still not beyond the bounds of nature; but the spirit of the rules of the nunnery, directing the mind to the most terrible rather than to the consoling truths of religion, as well as compelling the inmates to resign themselves in everything to the will of the abbess, produced effects as sad as unexpected. These effects were the same in all. At the end of two months' sojourn in this house the *menses* became suppressed, and in a month or two afterwards symptoms of phthisis appeared. As the nuns had not been allowed to take the usual vows, I entreated that they would leave the house; and all who followed this advice recovered. But during the ten years I was physician to this establishment the members were renewed twice or thrice, with the exception of the superior, the *tourière*, the sisters who had the care of the garden, of the kitchen, and of the infirmary, or of such as had more frequent intercourse with the city, and consequently greater distraction. The rest died of phthisis."

These circumstances now noticed—namely, hereditary predisposition, or the influence of parental cachexia generally, of various sources, especially syphilis, the influence of *race*, and of depressing passions—all co-operate, where they exist, in establishing the scrofulous cachexia; but the efficient cause seems to be "the impoverished nutrition resulting from *impure air*, and an *improper quantity*, *quality*, or *assimilation of food;* and so long as misery and poverty exist on the one hand, or dissipation and enervating luxuries on the other, so long will the causes be in operation which induced this terrible disease" (BENNETT).

It is, therefore, *the constitutional state* previous to the actual occurrence of local scrofulous affections like tubercle which it is of the utmost importance, if possible, to recognize; and the question has been proposed, "Whether or not a predisposition to scrofula can be anatomically or otherwise demonstrable?" There can be no doubt that an assemblage of phenomena, more or less well expressed and obvious, are characteristic of the scrofulous constitution. These phenomena are mainly due to impaired nutrition of the fundamental parts of the frame, and are associated with imperfect growth, especially of the skeleton, the nervous system, and the intestinal tract—imperfections capable of hereditary transmission to a remarkable extent. Of the structural characteristics of scrofulous persons, none are so significant as the weakness of cell-growth apparent in imperfections of the areolar tissue, as seen in the smooth, soft, delicate, blanched, and transparent skin. The nails are generally defective, elongated, and remarkably incurved. The mucous membranes are thin, pale, delicate, attenuated, and easily expanded. The debility of their function has been already fully noticed. The vascular system is thin, the vessels are transparent, more distensible, and less contractile than in the robust and healthy; and the visible tenuity of the vascular coats is sometimes rendered apparent in the tendency to hæmorrhages from slight causes. The veins are conspicuous, and appear to be more numerous under the skin. The bones are imperfectly grown; and there is often a greater want of symmetry than usual in the skeleton.

The weight of the man is light compared with his size and his age; and on these points in healthy men there ought to be a due concurrence. So important is the information capable of being derived from accurate observations in this direction, that the following tables are given for the purposes of comparison. (See pages 25 and 26.)

Growth, expressed by stature and weight, is thus most marked between the ages of fourteen and sixteen years (p. 26). Its rate is as much as three inches in height during that time; and about ten inches from the age of eleven to eighteen; and it goes on to be marked between eighteen and twenty-five years of age, to the extent of about two inches.

There are also ample physiological reasons of the most cogent kind which clearly show that great care is necessary in the physical training

GROWTH OF THE HUMAN BODY FROM EIGHTEEN TO THIRTY YEARS OF AGE, INDICATED BY WEIGHT AND HEIGHT: AVERAGES TAKEN FROM 100 OBSERVATIONS AND UPWARDS AT EACH AGE. RECORDS TAKEN FROM OBSERVATIONS UPON 4,800 CRIMINALS AT ALL AGES (*Statistical Society's Journal*, March, 1862, by J. W. Danson).

Age.	WEIGHT.												HEIGHT.								
	Average.		Maximum.		Minimum.		Maximum over Average.		Maximum under Average.		Maximum over Minimum.		Average.		Maximum.		Minimum.		Maximum over Average.	Minimum under Average.	Maximum over Minimum.
	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.	Feet.	Inches.	Feet.	Inches.	Feet.	Inches.	Inches.	Inches.	Inches.
18	8	10·79	10	13	6	6	2	2·2	2	4·79	4	7	5	4·34	5	11	4	10½	6·66	5·84	12½
19	9	4·11	12	8	7	4	3	3·89	2	0·11	5	4	5	4·94	5	11½	4	11	6·56	5·94	12½
20	9	5·58	12	8	7	13	3	2·42	1	6·58	4	9	5	5·11	5	11	5	1	5·89	4·11	10
21	9	5·02	12	0	7	3	2	9·	2	2·	4	11	5	5·57	5	11¼	5	0½	5·68	5·07	10¾
22	9	12·41	13	2	7	0	3	2·59	2	12·41	6	1	5	6·17	6	1	5	0¼	6·83	5·92	12¾
23	10	2·95	12	12	7	12	2	9·05	2	4·95	5	0	5	6·17	6	1	4	11	6·83	7·17	14
24	10	2·	12	12	7	12	2	10·	2	4·	5	0	5	5·94	6	1	4	9	7·06	8·94	16
25	10	5·65	13	8	8	2	3	2·35	2	3·65	5	6	5	6·30	6	0	4	11	5·77	7·30	13
26	10	1·06	13	8	6	12	3	6·94	3	3·06	6	10	5	6·28	6	1¾	4	9	7·07	8·78	16¼
27	10	4·75	13	10	7	12	3	5·25	2	6·75	5	12	5	6·38	5	11¾	5	1	5·37	5·38	10¾
28	10	2·62	13	2	7	7	2	13·2	2	9·62	5	9	5	6·65	6	1	5	1	6·35	5·65	12
29	10	5·53	13	12	8	4	3	6·47	2	1·53	5	8	5	7·02	6	0½	5	1¼	5·48	5·52	11¼
30	10	1·55	14	1	8	1	3	13·45	2	0·55	6	0	5	6·36	6	1	5	0¾	6·64	5·51	12¼

ANALYSIS OF MR. MACLAREN'S ANNUAL MEASUREMENTS OF 168 BOYS AT THE GYMNASIA OF THE MAGDALEN COLLEGE SCHOOL AND THE ST. PETER'S COLLEGE, RADLEY, OXFORD, IN 1862 AND 1863.

Number of Observations.	Year of Age.	STATURE.		WEIGHT.		Increase of Stature during the Year.	Increase of Weight during the Year.
		Shortest.	Tallest.	Lightest.	Heaviest.	Average.	Average.
		Ft. In.	Ft. In.	St. Llbs.	St. Lbs.	Inches.	Lbs.
5	9-10	4 0	4 10¼	3 11	4 7	·5	4
7	10-11	4 3⅛	4 7	4 6	5 11	·8	3·7
14	11-12	4 3½	4 11⅜	4 2	6 5	1·1	5
28	12-13	4 4⅜	5 5½	4 8	7 11	1·6	5·9
28	13-14	4 5⅜	5 7¾	4 9	9 10	2·2	7·9
23	*14-15	4 5⅜	5 7½	4 5	9 6	2·5*	8·1
33	†15-16	5 0½	6 0	5 8	10 4	2·2	10·6 †
19	16-17	4 8½	5 10½	5 10	10 10	1·3	8·3
7	17-18	5 3½	6 0¾	7 13	11 8	·6	5·5
4	18-19	5 5½	5 6½	8 8	9 10	·5	5

HEIGHT, AND CIRCUMFERENCE OF CHEST OF 1,270 YOUNG PERSONS, RECORDED BY DR. HARRISON, OF PRESTON (*Edin. Med. and Surg. Journal*, 1835, p. 425).

Age.	Number Examined.	HEIGHT.			CIRCUMFERENCE.		
		Average.	Tallest.	Lowest.	Average.	Great.	Small.
		Ft. In.	Ft. In.	Ft. In.	Inches.	Inches.	Inches.
11-12	210	4 2¾	4 11	3 10½	25½	30	22
12-13	203	4 5	5 0	3 11	26	29	21
13-14	192	4 6½	5 3	4 0	26	32	23
14-15	197	4 7	5 3	4 0	26½	32	21
15-16	186	4 10	5 6	4 1	27½	32	23
16-17	131	5 0¾	5 7	4 4	28	34	24
17-18	151	5 0¾	5 7	4 3	27½	34	23

of young persons.‡ The main reasons are to be found in the co-relations which obtain amongst the following elements, and which ought to be maintained at the healthiest standard, namely,—(1.) Age; (2.) Weight; (3.) Stature; (4.) Development and growth of the skeleton; (5.) The vital capacity of the chest; (6.) The growth of the muscles in relation to the bones; and the progressive increase of muscular force with advance of years.

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* Year of greatest increase in stature.

† Year of greatest increase in weight.

‡ I. Compare the average height of *full-grown* men at twenty-five years of age, as given by the following observers:—

Quetelet,	5 feet 5·27 inches	= 65·27 inches.	Average, 66·8.
Danson,	5 „ 6·30 „	= 66·30 „	
Boyd,	5 „ 7· „	= 67· „	
Liharzik,	5 „ 8·89 „	= 68·8 „	

II. The average height of the *growing lad* at eighteen years of age:—

Boyd,	5 feet 0·5 inches	= 60·5 inches.	Average, 63·003.
Liharzik,	5 „ 4·17 „	= 64·17 „	
Danson,	5 „ 4·34 „	= 64·34 „	

There are individual peculiarities connected with the skeleton which at once ought to excite suspicion and suggest careful training. These are,—(1.) Narrowness of the thorax, especially at the expansion of the false ribs—a condition, when combined with functional incapacity or inefficiency in the acts of respiration of most characteristic significance. The vital capacity of the lungs, as determined by (Hutchinson's) spirometer, compared with age, weight, and height, ought to be observed and recorded in all physical examinations. Hutchinson's observations show that men from five to six feet high have a vital capacity of lung ranging from 174 to 262 cubic inches, in an ascending scale according to height and age; and whenever the quantity of air is 16 *per cent.* deficient, there is reason to suspect some local affection of the chest. Dr. Graham Balfour has subsequently followed up these investigations with the spirometer. He has especially examined how far a capacity *under* the average may be taken as an indication either of a tendency to pulmonary disease or of a feeble constitution, rendering the man liable to a higher rate of mortality than that to which men of or above the average are subject. He found that the loss to the service by consumption was much greater among the men having a "*vital capacity*" *under the average* than amongst men of *average capacity* or *above* it; and although the proportion of deaths did not differ materially amongst those three classes, yet the invaliding was *four times as high* among men *under the average* as among the others. A "vital capacity" *below the average* may therefore be considered as indicating a generally feeble organization, less capable of resisting the deteriorating influences to which a soldier is exposed, ("Contributions to the Study of Spirometry," *Med.-Chir. Transactions*, vol. xliii.)

Such functional incapacity is further indicated by the "breathing being shorter," with less "breath motion." The *EX*piration is quick and forcible; and there is a minimum quantity of air taken in by ordinary *IN*spiration. Such lessened respiration tends of itself to induce accumulation of mucus in the air-cells, and thereby to set up inflammation. Everything which tends to impede or to interrupt or obstruct the regular, complete, and continuous performance of the respiratory acts has a most prejudicial effect upon the lungs (especially of "growing lads"), favouring the accumulation of growing material in the air-cells, which may eventually degenerate and form a cheesy mass in all respects resembling tubercle. Life not only depends on breathing, but the energy and the vigour of life are in a great measure ruled by the capacity and the free play of the breathing organs (SIBSON, 1844).

Dr. Walshe has very justly directed attention to the prevalence of anæmia amongst female servants in London, who are obliged to go up numerous and long flights of stairs very often in their daily labours; and is of opinion that the suspended inspiratory acts have much to do with the peculiar state of the blood that ensues ("Clinical Lectures," *Lancet*, 1849). Dr. Pollock, on the other hand, however, protests against anæmia being considered a frequent antecedent or manifestation of phthisis. He considers it a condition least frequently associated with phthisis. In none of 125 cases of anæmia was there any sign of tubercle; and although the girl with extreme anæmia is always supposed to be consumptive, the diagnostic points are:—(1.) The build of the anæmic patient is generally antiphthisical, and, in spite of her colour and delicate appearance, there is neither loss of flesh to any great extent nor fever. (2.) Mobility, percussion, and, tone of respiratory sounds are normal, even when cough, dyspnœa, and hæmoptysis may have existed for months.

The blood-changes characteristic of anæmia Dr. Pollock considers antagonistic to tubercle; and cases of anæmia very rarely proceed to tubercle of the lung. Anæmia wastes those organs and tissues dependent on an abundant supply of red blood; phthisis wastes all tissues by furnishing them with impure blood. The phenomena of anæmia are insufficiency; the phenomena of phthisis are impurity of blood (POLLOCK).

It is very important to remember, in regard to training animals and young persons, that they have much more extent and range of lung than are required in the quiet every-day pursuits of life. Less lung is used when the animal lies down or sleeps, or is depressed; and, on the contrary, walking, running, wrestling, the force of the passions, each or all of them bring the greatest amount of lung into action or use. In short, the amount of lung in use is an ever-varying quantity; and just as much lung *may come to be used as* A HABIT as the listlessness or vigour of the moment requires. There is every range, every variety, till the top of the wind, the top of the speed (the full vital capacity) is attained (SIBSON).

Practically, therefore, the more fully the lungs are judiciously used, the more is their capacity nursed; and conversely, the less they are used and expanded, the more useless they are likely to become, if not absolutely diseased. Under a judicious system of training, an undeveloped man, even although he may be feeble, narrow-chested, and sickly, may yet become active, full-chested, and healthy. We have numerous examples of this among the boys in our training ships for seamen. The over-fed, short-winded pugilist, rower, or cricketer, may in a few weeks be changed, by training alone, to the firm-fleshed, clear-skinned, long-winded winner of the boxing fight, the foot race, or the rowing-match.

It is this want of use that probably renders the apices of the lungs more liable to the growth of tubercle in them than in any other part of their substance. It is a portion of the lungs which has less play or expansion than any other portion, and is apt to be bound down by the surrounding parts. The apices are, therefore, the parts most likely to remain in a quiescent state of non-expansion, especially when acts of respiration are inadequately performed, either owing to the constrained position of the body in certain trades, or from habitual stooping of the body from listlessness of habit or want of vigour in the system. This quiescent state of the air-cells is favourable to the growth of tubercle; and thus the apices of the lungs are the most common seat of tuberculous growths (Reviewer in *Medical Mirror*, vol. i., p. 638). The effects of want of exercise and of impure air are thus most potent agencies in causing phthisis. "The much greater prevalence of phthisis in most of the European armies (French, Prussian, Russian, Belgian, and English) can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault." In all the places where phthisis has prevailed, in the most varied stations of the army, and in the most beautiful climates, the only common condition was the vitiated atmosphere which our barrack system everywhere produced; "and, as if to clench the argument, there has been of late years a most decided decline in phthisical cases in these stations, while the only circumstance which has notably changed in the time has been the condition of the air" (Parkes, l. c., p. 91). Indeed, the air is rendered so impure by respiration, that while an atmosphere so vitiated has a most injurious effect upon the health, contamination of the air has been so great, where lung diseases abound, as to give rise to the idea that phthisis appeared to be propagated by contagion (BRYSON, PARKES).

It is therefore within the power of the medical officer to direct the physical training of young persons so that the apparently sickly and the short-winded may in time be developed into the wiry and active young man, long in wind, sound in body, and lithe of limb—a result which, however, can only be attained by judicious feeding, careful exercise throughout the development of the body, and by the gradual nursing of the breathing powers. The opposite of this is seen in the breaking down of recruits at a very early period of service in the army—within the third year—a result likely to be greatly obviated by the *gymnasia* which are now being introduced by Government for the physical training of recruits before and after they are instructed in drill.

The Austrian runners are another class who furnish instances of breaking down by over-exertion in running at ages unsuited for their strength. They seldom live above three or four years, and gradually die of consumpton (*Remains of Mrs. Trench,* p. 72).

In all physical training the condition of the heart must be considered not less than the lungs, seeing that its movements respond to the movements of the chest and the lungs. They are members of one great system; and in nursing one we nurse the other; for by giving tone and health to one we must give tone and strength to the other (SIBSON). If, on the contrary, the development and gradual training of the lungs are not successful, the lesions which become developed are of a peculiar character, and are apt to be thought very lightly of; because, in the first instance, they are so little capable of appreciation by a single observation; and, secondly, because the lesions are insidious, and take some time before they reach a stage to be apparent. Frequent observations, extended over a considerable interval of time, are necessary to appreciate the wasting of delicate tissues which sets in, and which progresses so insidiously, and creeps on almost imperceptibly, but with a result so sure "that day by day and grain by grain the mortal part wastes and dies away." It is this progressive atrophy which it is so important to detect. Time is necessary to do this, and some certain mode of detecting the gradual decay. For this reason it is absolutely necessary to have the means of weighing men, and so to determine the ratio of the weight of the person to his age, to his height, and to his respiratory and other functions. These data are absolutely necessary to be known and recognized before any conclusions can be arrived at as to those healthy or normal physiological states with which weight has to do. It may, indeed, be stated generally that every organ and function has a physiological relation to weight and age; and, as a general rule, weight increases with the height and age, and there are physiological limits beyond which the range may not extend.*

Loss of body weight is therefore *exceedingly significant*, if PROGRESSIVE, *as indicating a persistent atrophy*—the *grain* by *grain decay* which is frequently the earliest appreciable sign of disease. The first manifestation results from absorption of the fat and wasting of the muscles of the body. The appetite may be good and the secretions regular, yet emaciation below the lowest average normal to the individual steadily goes on—a condition more marked and more perilous in those who have been previously stout than in others. The loss of weight is regularly progressive, and necessary to be distinguished from transient loss of weight. In senile phthisis a long period of emaciation commonly precedes positive evidence from physical signs. Sudden and great loss of weight is always serious. It indicates, in the early stages, rapid pulmonary lesions; and generally the most rapid reduction of weight is in males (POLLOCK).

The most rapid loss of weight observed by Dr. Pollock was a loss of 56 lbs. in three months; and in twenty-eight cases the following table shows the progressive reduction in weight:—

Period.	No. of Cases.	1 stone.	1½ stone.	2 stone.	2½ stone.	3 stone.	4 stone.
In 5 weeks .	1 had lost. . . .	—	—	1			
„ 2 months	4 „ (respectively)	—	1	2	—	1	
„ 3 „	7 „ „	1	2	1	—	1	1
„ 4 „	3 „ „	—	—	2	—	1	
„ 6 „	3 „ „	—	1	1	1		
„ 8 „	1 „ „	—	—	—	—	1	
„ 9 „	1 „ „	—	1				
„ 10 „	3 „ „	—	1	1	1		
„ 12 „	5 „ „	1	—	1	—	2	1
Total cases,	28						

The lowest weight in health of one of these persons was 10 st.; he lost in five weeks 1½ st. The rest in health averaged 12 st.; and one was 14 st. 10 lbs., and he lost 3 st. in eight months. Another, whose weight in health had been 13 st., was reduced to 10 st. in three months; 20 out of 28 observed, were between twenty-five and thirty-five years of age; 6 were in the first stage; 10 in the second; and 12 in the third of phthisis when examined, showing that the period of deposit was exceedingly short, and the softening and destructive processes accelerated.

General Treatment of the Scrofulous Cachexia.—From what has been already stated in illustration of the pathology of this malady, it is to be observed that the general treatment now in most repute is founded on the doctrines—(1.) That scrofulous local lesions will heal of themselves if the nutrition of the system can be maintained and the continuous growth of tubercle arrested; (2.) That the periods of frequent temporary arrest of all the general and local symptoms of disease ought to be diligently taken advantage of to improve and preserve health by hygienic means; (3.) That the efforts of the practitioner should be directed to the digestive rather than to the pulmonary system; (4.) That the kind of morbid nutrition in the body generally, and altered morphological change in the tissues of the organ where the deposit takes place, appear to be chiefly due to excess of albuminous and deficiency of fatty elements in the chyle.

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* Henry Pooley & Co., 89 Fleet Street, E.C., supply suitable weighing machines at a cost of £3, 15s., and upwards.

The treatment, therefore, to be pursued must be essentially reparative of the waste of tissue generally; corrective of what has been unfit in the individual diet and mode of life; and, lastly, supplementary of the elements of nutrition which have been deficient.

During the past *thirty* years in Germany, and *twenty-three* years in this country, the treatment of scrofulous affections has gradually but steadily become more and more firmly based on those pathological doctrines which Dr. Bennett, of Edinburgh, was mainly instrumental in first elucidating, and in earnestly recommending to the notice of the profession generally in this country. The view here taken regarding the nature of scrofula leads to the belief that—(1.) The blood is impoverized through the preliminary dyspepsia which precedes the growth of tubercle; (2.) That in *pulmonary phthisis* the growth of tubercle results from the exudation of lymph and of new growth which is consolidated primarily in the air-vesicles; (3.) That the successive formation and softening of these tubercles lead to ulcerations of the pulmonary and other tissues, and promotes wasting of the body generally.

It has been now fully shown, especially by the observations of Dr. Bennett, in the first instance, confirmed by the extensive experience of the physicians at the Brompton Hospital for Consumption, in London, that such treatment as is directed to remove the mal-assimilation of food frequently checks the tendency to the repeated occurrence of tubercles, while those which previously existed remain harmless; that general symptoms and physical signs may disappear completely; and that even extensive excavations in the pulmonary tissue may heal up and cicatrize. According to the testimony of Dr. Wood, of Pennsylvania, the fatal results have not only been postponed, but the death-rate from phthisis has diminished in the principal cities of the United States since such principles of treatment have been adopted.

The indications of general treatment are therefore—

First, *To improve the faulty nutrition, which is the cause of the scrofulous cachexia, and of the exudations assuming the characters of tubercle.*

Second, *To subdue the fever which attends the growth and changes going on in the tubercle-nodules, and to favour the absorption either of the entire exudation, or of such portions of it, that what remains may undergo such changes as are consistent with its future harmless existence in the organs or other parts where it may have grown.*

Third, *To prevent the recurrence of fresh exudation by careful attention to hygienic regulations, especially during the intervals of apparent return to health.*

To fulfil the first of these indications it is of all things important that fatty matter be assimilated in large quantities, and it appears that such are most readily absorbed and assimilated when in the fluid condition. The substance of all others found most beneficial has been cod-liver oil. At the Brompton Hospital more than 600 gallons of this oil are used annually. There, on a great scale, its merits have been tested and compared with the effect of treatment conducted on general principles, and irrespective of its use. The result has been to confirm, in the estimation of the medical profession, the great value of the remedy in the treatment of phthisis, when appropriately administered, and combined with the use of such other measures as any special circumstances in the individual patient may require (THOMPSON). The general opinion of the profession with regard to cod-liver oil may be summed up in the statement originally made by Dr. Bennett,—namely, that "it rapidly restores the exhausted powers of the patient, improves the nutritive functions generally, and stops or diminishes the emaciation. The night perspirations subside, the cough is quieted, and the expectoration is diminished after the oil has been used for a few weeks. A very constant and well-marked favourable change under its use is to be noticed in the diminution of the pulse, which gradually but steadily has been observed to come down at the rate of five or six beats weekly; and during the same period the weight of the body has been known steadily to increase at the rate of half a pound to a pound. In females it is a significant and highly favourable symptom when the catamenia return after the oil has been used for some time. The usual dose adopted by the late Dr. Thompson, at the Brompton Hospital, is *one* or *two tea-spoonful twice a day* at first, and gradually increasing the quantity to *half an ounce three times a day.* Dr. Bennett recommends somewhat larger doses—namely, for an adult, *a table-spoonful three times a day*, which may often be increased to *four* or *even six* with advantage. When the stomach is irritable, however, *a tea* or *a dessert-spoonful is enough to commence with.* It appears from the observations of Dr. Thompson that no additional advantage is obtained by pushing the oil beyond the limits of the doses adopted by him at the Brompton Hospital, from the fact that where its use has most obviously increased the weight of the body, to the extent in one instance of a pound per week for twenty-one weeks, only three pints had been taken during that time. The kind of oil used, as far as coarseness or fineness is concerned, seems not to affect the beneficial result in any material degree. Some patients even prefer the coarse to the fine oil. The experience of some is, moreover, favourable to combining the oil with *liquor potassæ* as an emulsion; and as it appears that undue acidity prevails as well in the stomach as in the intestinal canal, the addition of the alkali ought, on theoretical grounds, to be advantageous.

It seems also that, when cod-liver oil was first used as a medicine, more than fifty years ago, in the treatment of rheumatism, it was then ordinarily combined with an alkali. It may be taken, however, unmixed, or it may be floated on milk, or nitro-muriatic acid mixtures, or on lemonade, soda-water, lemon juice, or on a saline draught during effervescence, when such combinations are suited to the patient. Creosote has been recommended to be added, as it is said to render the stomach more tolerant of the remedy. The following formula, quoted from Dr. Thompson's *Clinical Lectures on Pulmonary Consumption*, yields a palatable mixture, which ought to be combined as an emulsion:—

"An ounce and a half of cod-liver oil, four drops of creosote, two drachms of compound tragacanth powder, and four ounces and a half of aniseed water. Of this mixture an ounce may be taken thrice daily."

The more direct and immediate action of cod-liver oil upon the blood has been attempted to be ascertained by Simon, Snow, and Thompson. They record an increase of blood-corpuscles and a diminution of fibrin under its use; and from the researches that have been made in animal chemistry regarding the blood in phthisis, a deficient proportion of blood-corpuscles is observed to be a most constant peculiarity. But phthisis is not the only disease in which this occurs, as shown by the following table (SIMON):—

AVERAGE PROPORTION OF SOME CONSTITUENTS OF THE BLOOD.

	Albumen.	Corpuscles.
In Health,.............	76	130
In Pneumonia,.........	80	122
In Phthisis,............	100	78
In Rheumatism,......	100	74
In Diabetes,.............	105	80
In Bright's Disease,...	103	50
In Chlorosis,............	72	56
In Carcinoma,...........	45	55

Rheumatism and diabetes present the greatest similarity in these states of the blood to phthisis; and they are diseases for which cod-liver oil has been used with advantage.

Cod-liver oil, therefore, is indicated, where it can be taken, in all those diseases in which the blood-corpuscles are deficient, where nutrition is impaired, and where fat is not readily assimilated.

Besides cod-liver oil, other animal fats and oils, where they can be taken and assimilated, are sure to be followed with benefit. Hence milk rich in fatty matter, such as asses' milk, and milk drawn from cows at a short interval after the greater part of their milk has been withdrawn, and which is known in Scotland as the "afterings," are found to be followed by improvement where they are persevered in and are assimilated. So also has it been with cream and butter. Dr. Bennett instances the partial success occasionally of caviar, bacon, pork, mutton chops, and the marrow of the bones of oxen; while Dr. Thompson instances the good effects he has obtained from the use of oil obtained from the foot of the young heifer (neat's-foot oil). The administration of any of these remedies is quite consistent with doctrines now taught regarding the pathology of tuberculosis, and it is useful to know their individual value, in order that in particular cases one may fall back upon their use where a change may be desirable.

It has been considered that some of the good effects of cod-liver oil may be due to the biliary elements with which it has been incorporated. This view is not supported by the experiment of adding ox-gall to other animal oils not derived from livers, as no beneficial results have been observed to follow. But as the active principle of the gastric juice has been now successfully isolated by chemistry, and has been successfully used to aid the digestion of food in the stomach, might not some principle be obtained from the liver which might aid the assimilation of fatty substances when mixed with the intestinal juices?

In fulfilment of the second indication mentioned (p. 31), the propriety of abstracting blood has been much discussed.

It has been already seen that febrile symptoms of a very severe kind sometimes attend the exudation and consolidation of tubercle. It is also a more or less frequent clinical observation that pneumonia, bronchitis, and pleuritis, in acute or chronic forms, are intercurrent attendants on the deposit and future changes of tubercles in the lungs. "Hence," as Dr. Bennett justly observes, "there are all kinds of intermediate changes between the simple and tubercular exudations constantly going on in the progress of a case of pulmonary tuberculosis. The phenomena of phthisis, pneumonia, pleurisy, and bronchitis, in their acute or chronic forms, may appear together, and be inextricably mingled, or they may succeed each other at intervals." Thus tuberculosis, both as a constitutional and as a local disease, is scarcely ever free from exacerbations, the various local and constitutional states acting and re-acting on each other. While, therefore, on the one hand, the system requires an increased and well-directed supply of nutritive materials, on the other hand, there are constitutional states of excitement, depending on local irritation, which require to be subdued, and even demand antiphlogistic treatment. Whatever theoretical view may be taken as to how the exudation may most readily be absorbed, all physicians are now at one as to the propriety of preserving the general strength, of effecting elimination of effete material, and of meeting antiphlogistic indications rather by diaphoretics, diuretics, emetics, and purgatives, than by abstracting any considerable amount of blood, either at once or at repeated intervals. In fact, it is now observed that the administration of appropriate diet, and abstinence from lowering remedies, with cod-liver oil, while they correct the general nutrition, may be so regulated as to subdue the constitutional irritation by a perseverance in their use for a period of not less than four or six weeks. Moderate general bleedings in acute phthisis, as well as local bleedings during the exacerbations of chronic phthisis, undoubtedly confer a temporary relief in the diminution of local pain and general febrile re-action, and allow a more free respiration to be performed, if respiration should be impeded by congestion of the lungs. Dr. Bennett, however, is opposed to bleeding. His object is simply to favour excretion by means of antimonials; and, on subduing the re-action by their means, he again proceeds with the nutritive mode of treatment to fulfil the first indication. Nevertheless, it cannot be doubted that the condition of some cases demands blood-letting; but, as Sir James Clark observes, "The employment of general blood-letting in consumption requires great judgment and circumspection. The more general error is the abstraction of too great a quantity of blood at a time—treating the disease as if it were a purely inflammatory one, and forgetting that the inflammatory symptoms are merely consecutive upon tubercles, and that the constitution of the consumptive patient is little capable of replacing the blood too lavishly drawn." Simply to remove or diminish congestion, the condition of the patient and nature of his constitution being considered, "blood may be abstracted," he continues, "with advantage at any stage of consumption when the symptoms require it." The experience of Dr. Wood leads him to teach similar doctrines. Topical counter-irritants furnish the best means of subduing constitutional irritation in chronic forms of the disease. These may be in the form of setons, issues, succession of blisters, tartar emetic ointment, or croton oil rubbed on the chest in any of its regions. Amongst these the frequent use of dry cupping ought not to be omitted.

In fulfilling the third indication, the real power of the science of medicine may be demonstrated. It is by well-directed hygienic measures, successfully and efficiently carried out, that the real strength of the physician may be put forth to prevent the recurrence of fresh exudation; and his hygienic exertions towards the patient are to be redoubled during the temporary intervals of apparent return to health. It is now almost an axiomatic truth, that of all things which deteriorate the constitution on the one hand, and influence pulmonary congestion on the other, none are so detrimental as impure and deficient air, together with frequent variations of temperature, and changes from sudden heat to chilling cold. These latter vicissitudes may be considered characteristic of the physical climate of Great Britain and Ireland. "The conditions of preventive treatment which have seemed most useful are nutritious food and proportionate great exercise in the free and open air. So important has this last condition proved to be, that it would appear that even considerable exposure to the weather is better than keeping phthisical patients in close rooms, provided there be no bronchitis or tendency to pneumonia or pleurisy" (Parkes, l. c., p. 445).

Tonic treatment consists essentially in the adoption of those means which promote or stimulate the healthy nutrition of the body. Its elements exist in fresh air, abundant exercise, sufficient repose, and judicious diet. A hygienic code applicable to the tuberculous cachexia has been recently laid down by Dr. Richardson, in the second volume of the *Sanitary Review,* and very recently in a special work *On the Hygienic Treatment of Pulmonary Consumption.* It is derived from these elements of tonic treatment; and as it puts well-known truths, too little appreciated, in a formal and more important aspect than is wont, its precepts are here quoted.

I. *A supply of pure and fresh air for respiration is constantly required by the tuberculous patient.*

As it is known that if *one per cent.* of carbonic acid exists in a room, the air is unfit for a healthy person, it is therefore much more so for a consumptive one. The temperature of a room ought to be equally maintained at from 55° to 56° Fahr., ventilation and heating being effected by open fire-places. A single room ought not to perform the two offices of a bed-room and a sitting-room. The sleeping-room ought not to afford less than 1,000 feet of space; and if larger, so much the more healthful will it be. In connection with these statements, while it is objected, for obvious reasons, with much justice, to the treatment of tuberculous patients in special hospitals, there is much on the other hand that might be improved in all our hospitals, with reference to the arrangement of the patients, to secure to them fresher air than they generally obtain. "Constant though impereceptible movement of the air is the point to be attended to"—*i. e.*, thorough ventilation. All who are able to be out of bed ought to have their meals in a common room, which is not used for any other purpose, and is apart from the wards or dormitories.

II *Active exercise in the open air is imperatively demanded by the tuberculous patient.*

In the words of Dr. Jackson, "He must be made to feel that the *risk is in staying in the house,* and not in going out of it." But the skin must be perfectly protected; and while a chill, or inclement weather, is to be avoided, the patient must go out in all seasons, without being too fastidious about the weather, walking exercise being persevered in as much as possible. "The best climates for phthisis are perhaps not necessarily the equable ones, but those which permit the greatest number of hours to be passed out of the house" (PARKES). Next to diet exercise in the open air is, of all things, the most important: it should be carried as far as the vigour of the patient will permit. It should not be done rashly, but boldly; and, if possible, the patient ought to have faith in it; for without this he is not likely to pursue it so far as he can, and then he will not derive from it all the benefit which it can afford (JACKSON).

III. *It is important to secure for the patient a uniform, sheltered, temperate, and mild climate to live in, with a temperature about* 60° *and a range of not more than* 10° *or* 15°; *where also the soil is dry, and the drinking-water pure and not hard.*

The classic work of reference on this topic is that by the late Sir James Clark. If it is possible to give a practical abstract of his extensive and valuable experience, it may be done somewhat as follows:—

1. After the functions of the digestive organs and skin have been re-established in improved action, the patient who labours under a tuberculous cachexia may derive benefit by a residence in a mild or temperate climate (such as has been defined), conforming to all the hygienic and medicinal treatment already mentioned.

2. When symptoms, however slight, indicate that tuberculous deposit has located itself in the lung, removal to a mild climate, especially if effected by a sea voyage, under favourable circumstances, may still be useful as a means of improving general health, of lessening the chance of intercurrent inflammatory affections of the pulmonary organs, and even of arresting the further progress of the disease. The nausea, squeamishness, or even sickness, which with some are always more or less associated with a sea voyage, are beneficial to cases of incipient tuberculosis. The effects of such nausea tend to increase the natural secretion and elimination from the pulmonary mucous membrane; so that minute portions of tubercular exudation, commencing to consolidate in the air-vesicles, are effectually, gradually, and gently passed out with the motion of the pulmonary mucus in the expectoration. The sensation

of nausea tends to subdue any local vascular irritation; and the unceasing motion of a sailing vessel tends to keep up a constant exercise which is advantageous to the patient.

3. When extensive tuberculous disease exists in the lungs, little benefit is to be expected from a change of climate; and a long journey will most certainly increase the sufferings of the patient, and hurry on a fatal termination.

4. There are cases, however, of chronic consumption which may derive benefit from residence in a mild climate,—namely, cases in which the deposit is limited to a small portion of the lungs, and little systemic irritation prevails; or in cases in which the disease has ceased to extend in the lungs, but where a long time is required to complete repair.

IV. *The dress of the scrofulous patient ought to be of such a kind as to equalize and retain the temperature of the body.*

Under this topic waterproof coats, boots, and shoes are to be condemned. Flannel ought invariably to be worn next the skin in all seasons; and in winter a chamois leather vest may be required over the flannel.

V. *The hours of rest should extend from sunset to sunrise.*

VI. *Indoor or sedentary occupation must be suspended; but outdoor employment in the fresh air, even in the midst of snow, has been and may be advantageous.*

VII. *Cleanliness of body is a special point to be attended to in the hygienic treatment of tuberculosis.*

VIII. *Marriage of consumptive females, for the sake of arresting the disease by pregnancy, is morally wrong and physically mischievous.*

IX. *The medicinal treatment must be adapted to the site of the local deposits and the general nature of the particular case. Iron and iodine in various forms are the most useful remedies;* but medicine is utterly powerless and useless unless the hygienic means now insisted upon are carried out to the uttermost.

RICKETS.

LATIN EQ., *Rachitis;* FRENCH EQ., *Rachitisme;* GERMAN EQ., *Rhachitis* —Syn., *Englische Krankheit;* ITALIAN EQ., *Rachitide.*

Definition.—*A constitutional disease of early childhood, characterized by an unhealthy state of the system, which precedes for several weeks or months a peculiar lesion of the bones, manifested by curvature of the shafts of long bones and enlargement of their cancellous extremities. Some of the solid visceral organs exhibit also peculiar lesions. The growth of the bones is characterized by irregularity, by non-solidification of their growing layers, and by the progressive formation of medullary cavities in the old bone, thus rendering the bony laminæ thin and brittle* (VIRCHOW). *In the solid visceral organs, such as the spleen and liver, there is generally lardaceous disease.*

Pathology.—From the great vascularity and infiltration of the affected bones, some have regarded the disease as expressing some form of inflammation; more especially as pain accompanies the lesions from the first. But the course and the constant results of the disease do not favour this view. Another theory (based on the fact that the urine of rachitic children is often extremely rich in *lactic acid*, and contains as much as from four to six times of *phosphate of lime* as normal urine contains) holds that the calcareous salts taken up by the food cannot be deposited in the terminal and peripheral layers of bone, being held in solution by the lactic acid in the blood and excreted by the kidneys.

But the actual changes in the bones do not support this view; these consisting of—(1.) Proliferation of the cartilages and epiphyses, and of the periosteum—the sources of the normal growth in length and thickness of the bones. (2.) The cartilaginous and fibrous tissues ossify more densely, and at a later period than in the normal growth of bone. The tissues remain abnormally soft for a length of time; after which they become abnormally hard (NIEMEYER). Virchow considers the retarded ossification due to a diminished supply of chalky salts. The most probable hypothesis regarding the cause of rickets is that which refers to inflammation of the epiphysal cartilages and periosteum, and local disturbance of circulation hindering the deposit of calcareous salts (NIEMEYER).

Active decomposition also goes on in the stomach and intestines of rachitic children.

Symptoms.—The earliest recognition of the cachexia associated with rickets is rarely apparent before the fourth month of infant life; and usually between the fourth and twelfth months. It does not in general declare itself until the child first begins his attempts to walk, or until he shows suffering during the first dentition; and at first the progress of the disease is so very slow as almost to be imperceptible. The number of cases happening in the first or second years of life very greatly exceeds that of other periods; and there is a period of at least six months during which a marked series of deranged actions succeed each other, and which eventually culminate in the condition known as *rickets.* Many of the phenomena of these deranged actions are common to other diseases; but some are characteristic, and when they occur in sequence are sufficient indications of the specific characters of rickets.

The symptoms may be arranged into four classes:—(1.) Those which are common to many diseases—symptoms which might arise from deranged digestion, from improper food, or from scrofula, and which are often referred to the "irritation of teething," or to the so-called "infantile remittent fever." These phenomena always denote the precursory or incubative stage of rickets; (2.) Those which at once mark the nature of the disease, render its diagnosis easy, and which enable us to predict that the bone affection will show itself; (3.) The stage of characteristic deformity; (4.) Phenomena of favourable or of unfavourable import, inasmuch as they may characterize a period of restoration to health, of irremediable atrophy of the body, or of approaching dissolution.

During the precursory or incubative period, the most ordinary symptoms of impaired general health are those which indicate gastro-intestinal irritation. The bowels are irregular in their action; sometimes confined, but more commonly there is diarrhœa (*intestinal catarrh*), with tumidity or enlargement of the abdomen. The stools may be of a dirty brown or leaden colour, and of a most offensive odour. There is great proneness to decomposition of the contents of the intestines. In some respects the odour therefrom is peculiar in its resemblance to rotten or half-decayed meat. Appetite is feeble, or entirely lost, and digestion is difficult. The child becomes dull and languid, sad or peevish; febrile irritation prevails; the skin is hot; and the temper irritable. Although drowsy, it sleeps but little. It is thirsty, and will drink large quantities of water. If it has begun to walk, it is "taken off its legs." It lies about, and is unwilling to play, or to be amused, or to indulge in any kind of action. It prefers to sit or to lie; and it appears to be feeble or indolent, and is unable to use exertion of any kind. The transition from health to these phenomena is always gradual and slow; but there are at least three sets of phenomena which, according to Sir William Jenner, being superadded to these, are characteristic of the approach of rickets. These symptoms, therefore, stand by themselves in the second class as pathognomonic of this affection.

(*a.*) The most remarkable is *profuse perspiration of the head, or of the head and neck*, and upper part of the chest. It arrests the mother's attention, and she seeks medical aid. She will tell the physician that the perspiration stands in large drops on the child's forehead—that it runs in streams down the face; and it is especially when the child sleeps that such copious perspirations of the head occur; but they are not unfrequent when the child is at the breast, or even when only resting its head on the mother's arms. A little increased exertion, or a little increased temperature, may induce such excessive perspiration. Such perspirations are extremely weakening and colliquative during sleep; and when they occur the superficial veins of the scalp are generally large and full; the jugular veins are much dilated, and sometimes the carotid arteries may be felt strong and pulsating (COPLAND, JENNER).

(*b.*) Another characteristic feature of the disease is seen in the desire and in the efforts of the little patient to be cool, particularly at night. The child kicks the bed-clothes off, or throws its naked legs on to the counterpane; and this even in cold weather.

(*c.*) There is also, thirdly, general tenderness. The child cannot be moved without its uttering a cry; pressure on any part of its body is followed by evidence of suffering. It ceases to play and to move, but lies with outstretched limbs as quietly as possible, for all movement produces pain; and it will cry at the approach of any one who has been accustomed to move it in play.

As the disease progresses the child becomes staid and steady in appearance. It assumes a pensive, aged, and languid aspect. Its face grows broad and square; and when placed on the mother's arms, it sits (as she says) "all of a heap." The spine bends, and the muscles are too weak to keep the spine erect. Its head thus seems to sink between its shoulders, and its face appears turned a little upwards. Before the general cachexia has lasted long, the bone deformity begins to attract attention, and usually the lesion of the bones is out of all proportion in severity to the enlargement of the ends of the long bones; and the younger the child, the softer usually are the bones. The consequences of the bone disease thus become superadded to the general cachexia; and as the disease progresses, the muscles lose their power and begin to waste. The child cannot support itself; and if it has commenced to walk before it becomes the subject of extreme rickets, it loses its power of walking. Intellect is invariably deficient. The teeth are retarded in their development, and they fall from their sockets early. The back, arms, and sides of the face are often covered with downy hair. In short, as Sir William Jenner observes, "the general aspect of the ricketty child is so peculiar that when the crooked limbs, the large joints, and the deformed thorax are concealed, you may even detect its ailment at a glance. Its square face, its prominent forehead, its want of colour, its large staring and yet mild eyes, its placid expression, and its want of power to support itself like other children of its age, in its mother's arm, all conspire to form a picture which has no like in the gallery of sick children," (*Med. Times*, l. c.) *Laryngismus stridulus* is a frequent result of rickets.

Treatment.—Ventilation of the room in which the child lives is of first importance. *Milk* diluted with *lime water* (about a fourth part), and a tea-spoonful or two of cream added, is the best of food. Sugar ought not to be added to the milk. *Liebig's food for children*, and *Parrish's chemical food* are both valuable agents in the dietary. About once a week a dose of *rhubarb, soda*, and *calumba*, in equal parts, should be given, followed next day by a tea-spoonful or more of *castor oil*. *Prepared chalk* and *soda* may also be given twice or thrice a day. When the febrile disturbance is subdued, the child should live as much as possible in the open air. *Vinum ferri* ought then to be given in the following formula:—

℞. Vin. Ferri *ƒ* ʒi.-ʒii.; quiniæ sulph., gr. i.; acid sulph. dil. ♏i.-ii.

This form is especially useful when the skin is flabby, covered with perspiration and *anœmia* well marked. Or small doses of the *syrup of the phosphates of iron, quinine, and strychnia*, or *syrup of the phosphate of iron and lime* may be given along with the food, or just before meals. *Cod-liver oil* is of essential service; but the stools ought to be examined daily; and if any of the oil passes by stool its dose ought to be diminished, or its administration suspended for a time. (For more details on *Rickets* and its treatment, consult the admirable lectures of Sir William Jenner, in *The Medical Times* of 1860, vol. i.)

CRETINISM.

Latin Eq., *Cretismus;* French Eq., *Crêtinisme;* German Eq., *Cretinismus;* Italian Eq., *Cretinismo.*

Definition.—*A condition of imperfect development and deformity of the whole body, especially of the head. It is endemic in the valleys of certain mountainous districts, and is attended by feebleness or absence of the mental faculties and special senses; and is often associated with goitre.*

Pathology and Phenomena of Cretinism.—The condition of idiocy named *cretinism* (and associated with goitre in many districts) is of great interest; but the relations of the two are not yet clearly understood. The idiocy of *cretinism* is associated with deformity and imperfection of the bodily organs, the brain, in common with other parts, participating in the imperfection and deformity. The affection of the mind varies from mere obtuseness of thought and purpose to the most complete obliteration of all intelligence.

Three *varieties* are to be distinguished:—

"(*a.*) *Complete Cretinism*—Synonym, *Incurable Cretinism,—Cretinism characterized by idiocy, deaf-dumbness, deficiency of general sensibility, and absence of the reproductive power.*

"(*b.*) *Semi-Cretinism,—A degree of cretinism in which the mental faculties are limited to the impressions of the senses and the bodily wants; the general sensibility is obtuse, the head is badly formed and drooping, the speech is rudimentary, and the reproductive powers are feeble or absent.*

"(*c.*) *Incomplete Cretinism*—Synonym, *Curable Cretinism,—A degree of cretinism in which the mental faculties, though limited, are capable of development; the head is moderately well formed and erect, the special senses, the faculty of speech, and the reproductive powers are present.*"

Dr. Guggenbühl, of Zurich, was the first to recognize the fact that the mental state of *cretins* could be improved by improving the growth and condition of the body. In 1842 he succeeded in buying the mountain of Abendberg, which encloses the plain of Interlaken, and there he established an hospital for these unfortunate children. The infant cretins, removed from the low close valleys (in which the malady too often finds the circumstances most congenial for its development), are there fed and trained in "the free, dry, cool, and bracing air of the open but sheltered and sunny slopes of the Abendberg."

With but few exceptions, *cretins* are goitrous; and it has been said that when both parents are goitrous for two generations in succession, the offspring, being the third generation, are sure to be cretins (WATSON).

The cretin is found chiefly in the valleys of the Pyrenees and the Alps, in the mountains of Syria, in the hilly parts of China, and in the Himalaya regions; but the disease is not confined to the lower valleys of Switzerland, or to those other mountain districts of the Old and New World which resemble it in physical conformation. All over Europe the victims of this disease may be seen; and Virchow found, in his official inquiry into this subject, that no less than 133 decided cases were living in the villages of Lower Franconia; and in Germany, Sweden, Norway, England, and even in London, isolated cases of *cretinism* are to be met with.

The stature of the *cretin* is diminutive; his head is of great size, but flattened at the top, and spread out laterally; while the countenance is vacant and void of intelligence. The nose is flat, the lips are thick, and the tongue is large. The skin is dark-coloured, coarse, and rough. The abdomen is sunken and pendulous; the legs are short and curved.

Virchow's dissection of the heads of *cretins* has led him to conclude that the primary abnormality of the brain commences with the growth of the bones of the *basis cranii*, and especially with the *sphenoid* and the adjoining parts. In the normal state the basiler part of the occipital bone, the sphenoid and ethmoid, with their intervening cartilages, form a portion of an arch; while the same parts in a *cretin* are early ossified into the form of a rectangle, early union of the bones taking place, with various lesions of the intervening cartilages. With such early union, arrest of growth occurs at that part of the skull; but various compensatory developments continue in other parts. Hence the *prognathous* face, and the sinking of the root of the nasal bone. Irregular and partial union of the sutures at an early age is a frequent morbid condition of the insane cretin, associated with an atrophic condition of the *gyri* below the site of union. The oblique downward direction of the orbit in *cretins* is brought about by the compensating growth of the skull generally, and more especially of the *malar*, the *frontal*, the *temporal* bones and *zygomatic* arches, in consequence of the deficient development of the *sphenoid* bone. The stunted development of the bones at the base of the skull gives a very short distance between the front and middle part of the cranium; while the diminished growth of the nasal septum and of the jawbones gives a *prognathous* form of face alike to the cretin, the negro, and the monkey.

It may generally, therefore, be concluded, from the cumulative nature of the evidence, that a poison exists in association with lime and magnesia in geological formation, whose action induces undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for blood-vessels (KÖLLIKER, VIRCHOW); and it is fair to connect the unusual quantity of lime taken into the system with such premature and abnormal ossifications. Wherever chemical examination of the water used by the inhabitants of the different places where goitre and cretinism prevail has been made (as it has been especially in India), it has always been found to contain a large quantity of carbonate of lime; whereas the water derived from the clay-slate rock, and which was drunk by the inhabitants who did not suffer from *goitre*, contained none. Such observations as those described, and especially those of M'Lelland and Greenhow, show that neither the atmosphere, the elevation above the sea-level, the physical aspect of the country, nor locality, have anything to do with the production of goitre; but they prove almost to demonstration that the affection is due to some specific action of the drinking-water which flows from rocks of a particular geological formation named magnesian limestone. The circumstances under which these affections were found by M'Lelland to exist in the low burning plains of Bengal, formed a striking corroboration to his observations in the hills of Kemaon.

Cretinism and goitre are very prevalent in different parts of the district of Goruckpore. The soil of the district is of two sorts. One, to which the natives give the name of *Bhat*, characterizes the lands bordering the river Gunduk and its branches. This soil is remarkable for the large proportion of calcareous matter which it contains. One specimen, on analysis, yielded upwards of twenty-five *per cent.* of carbonate of lime. Cretinism and goitre are very prevalent in the villages built upon this soil. In some of them 10 *per cent.* of the population are affected; and of the children in the villages where goitre prevails 10 *per cent.* are *cretins.* The dogs and cats of these villages are also often affected with the disease. On the other hand, the lands on the banks of the Gogra consist of a soil to which the natives give the name of *Bangar.* It is much less retentive of moisture than the *Bhat* land, and requires irrigation for the production of winter crops. This Bangar soil is very siliceous, and contains scarcely any lime. Goitre and cretinism are unknown in the villages built upon this soil (*Brit. and For. Med.-Chir. Review,* Jan., 1861).

The natives of Oude ascribe their goitres to drinking certain waters; and they adduce cases to prove that by partaking of the water of certain wells they get the disease, and by deserting those wells they sometimes become cured of it (GREENHOW). Thus almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of goitre. Remarkable instances are known wherein the exchange of well for rain-water, for drinking purposes, has been followed by the best effects, and even by the disappearance of goitrous tumors. Dr. Greenhow states that in Oude, where the water of wells believed to be injurious in consequence of their excessive impregnation with lime has been given up, and other water used instead for drinking, great benefit has been felt, and goitres have decreased in size, even though the subjects of them have continued living in the same village as before. He was assured also, by several of his patients in Oude, that certain wells were known by them to be deleterious, and that the natives of the villages avoided them accordingly, having learned to do so from experience. He tested the water of the wells most shunned by the natives, and found it to contain a great excess of lime; and he concludes, from his own investigations, in connection with others, that the use of drinking-water containing lime is the main cause of goitre. How it acts on the system is as yet unknown.

Symptoms.—Although it seems that some cases of cretinism are congenital, yet Dr. Tuke, from personal observation, and from the Report of the "Sardinian Commission," believes (*op. cit.,* p. 104) that there is no pathognomonic sign by which cretinism can be recognized at birth, but that a certain combination of symptoms permits the prognosis in childhood of the future development of cretinism. After the fifth or sixth month he describes the infant cretin as presenting the following symptoms:—"The development of the body proceeds very slowly; the child, though weak, is remarkably stout, and appears swollen; the colour of the skin is somewhat dusky, sometimes yellow, sometimes natural; the head is large, the *fontanelles* widely separated, and sometimes all the sutures disjointed. The expression is stupid; the appetite is voracious; and much time is passed in sleep. The belly is swollen; the extremities are generally attenuated; the neck is thick, but not always goitrous; teething is not completed for many years, is generally accompanied by an offensive salivation, and frequently by convulsions. Usually the child cannot stand before its sixth or seventh year; and it is then that it begins to articulate certain sounds, supposing it has not been deaf from birth. The voice is hoarse and shrill, and words are spoken with difficulty." His stature is diminutive. His head is of great size, but flattened at the top, and spread out laterally; and the dissections of Virchow show that the parts at the base of the cranium are early ossified in the form of a rectangle. Early union of the bones takes place, and with such early union arrest of growth occurs at that part of the skull, while various compensatory developments continue in other parts. Hence the *prognathous* face, and the sinking of the root of the nasal bone. Thus the character of the face in the cretin, as Dr. Tuke observes (*op. cit.*, pp. 105-6), remains unchanged from puberty to old age. The eyes are generally affected with strabismus; the zygomatic arch is large, and the mouth of large size; the lips thick, and the lower one hanging down. The lower jaw is small, retreating, and its angle very obtuse. The predisposition to cretinism appears to be hereditary; and, as noticed by Dr. Watson, it has a close but ill-understood connection with goitre, so that, with few exceptions, cretins are goitrous; and he also observes, that "when both parents are goitrous for two generations in succession, the offspring being in the third generation, are sure to be cretins" (Lect. xliii., vol. i., p. 788). Fodère makes an observation to the same effect.

Treatment.—The indications of treatment are those which suggest improvement in all hygenic measures for the prevention of the disease. Once established, the condition of the cretin can only be ameliorated by institutions similar to those founded by Dr. Guggenbühl of Zurich. The condition is beyond medicinal remedies.

DIABETES—*Syn.*, DIABETES MELLITUS.

LATIN EQ., *Diabetes*—Idem valet, *Diabetes mellitus;* FRENCH EQ., *Diabète*—Syn., *Diabète sucré;* GERMAN EQ., *Diabetes*—Syn., *Zucker harnruhr;* ITALIAN EQ., *Diabete*—Syn., *Diabete melito.*

Definition.—*A constitutional disease obviously produced through errors in the processes of assimilation either in the stomach, in solid organs, or in the blood, and characterized especially by an excessive discharge of urine, more or less constantly saccharine, excessive thirst, and associated with progressive emaciation of the body.*

Pathology.—From the time of Charles II. of England, when Dr. Thomas Willis first observed the saccarine character of diabetic urine, no disease has had its nature more inquisitively examined, and with more interesting and instructive results. The abnormal state of the urine naturally at first led the inquiry towards the kidneys. They have been industriously examined, both as to their structural and functional relations, but without elucidating the nature of the change in the urine. Dr. Matthew Dobson, of Liverpool, in 1779, first established by experiment the fact that the sweetness was due to the presence of sugar. The next step in the inquiry was the detection of sugar in the blood of the diabetic patient. Ambrosiani, of Milan, in 1835, and Dr. Charles Maitland, in 1836, obtained crystals of pure sugar from the serum of the blood, and a large portion of fermentable crystallizable syrup. The late Dr. Robert Macgregor, of Glasgow, in 1837, confirmed these observations by experiments, followed by those of Dr. G. O. Rees, of London, and Dr. Christison, of Edinburgh. Thus inquiry regarding the essential character of the disease was removed from the urine and the kidney to the blood; and research took a new direction, so as to ascertain, if possible, the source of the sugar in the blood and in the urine.

Although this disease has hitherto found a nosological place under diseases of the kidneys, the researches of Bernard, Parkes, Pavy, Ringer, and others, very clearly show that amongst them it is misplaced. If the disease is to be regarded as a local one, it should rather come under *hepatic* than *nephritic diseases;* but the weight of evidence clearly shows that *diabetes mellitus* belongs to the *constitutional* class of diseases.

The unusual discharge of urine in this malady was originally ascribed by Mead to a morbid state of the liver and bile; but subsequently nutritive and assimilative functions connected with the digestive canal were considered by Cullen, Home, and Dobson to give rise to the morbid state. That the process of digestion and assimilation in the stomach was the source of the evil has been hitherto the prevailing theory regarding the nature of this disease. The belief received confirmation especially by the ingenious experiments of the late Dr. Robert Macgregor, of Glasgow, who ascertained that sugar was found in the stomach of diabetic patients during the process of chymification, even when no saccharine matter had been swallowed. Sugar has now also been detected in the saliva, the sweat, and in the stools. To the ingenious M. Claude Bernard, of Paris, the science of medicine is indebted for the elucidation of the nature of the normal generation of sugar in the animal economy (glycogenesis); and thus we have been provided with data for the comparison and study of its morbid generation in *melituria.* He has shown us that one of the natural functions of the liver is to generate sugar; and thus the sagacious speculations of Mead, which referred the phenomena of diabetes to a morbid state of the liver and bile, are now not only curious, but interesting and instructive. While our knowledge regarding the formation of sugar by the liver, and its physiological relations to the animal economy in health and in disease, have been especially illustrated by Bernard, in France, it is to be observed that Dr. George Harley, of University College, and Dr. Pavy, of Guy's Hospital, London, have confirmed many points, and especially elucidated the interesting doctrines of Bernard. Much is still unsettled as to the significance of the phenomena; but the following statements will convey a summary of the present position of the inquiry regarding *glycogenesis,* as set forth in the *Medico-Chirurgical Review* for January, 1857.

From the experiments just referred to, the fact was considered as an established one, that the food, and more especially its amylaceous and saccharine elements, had the power of forming sugar, which then passed into the blood and urine from the alimentary canal. The experiments of Bernard and others, however, have demonstrated that the animal organism has the power of forming sugar altogether irrespective of the nature of the food; and that sugar exists in a certain part of the circulation—namely, from the hepatic veins to the pulmonary capillaries—both in carnivorous and herbivorous animals. The liver is found to be this sugar-producing organ; and it is the only organ of the body which, in the normal state, is found to be impregnated with sugar—a fact established by Bernard, by observations on a large number of animals in almost every department of the zoological series. In man he examined especially the liver of five executed criminals, also that of a man who was killed by a gun-shot wound, and that of a diabetic patient who died suddenly from pulmonary apoplexy. In four of these he determined the absolute and the relative amount of sugar in the liver. The total weight of the three healthy livers was 4·205 grammes, which yielded of sugar a total of 66·074 grammes; so that the average weight of each liver being 1,401·2 grammes, the weight of sugar yielded was 22·037 grammes, to compare with the liver of the diabetic case, which weighed 2,500 grammes, and yielded 57·50 grammes of sugar. He shows that the relative quantity of sugar varies little when the system is in a normal condition, very seldom exceeding 4 *per cent.* He especially demonstrated that sugar was secreted in the liver, and entered the blood from that organ; for, by a comparative analysis of the blood of the portal vein as it enters, and the blood of the hepatic veins as they emerge from the liver, he found sugar in the latter, but not in the former. Sugar is not only found in the liver of adult animals, but it has been found in the livers of the human fœtus, the fœtal calf, and the unhatched chick—thus proving that it does not merely accumulate there as a product from the digestive canal.

Further, it has been determined that the sugar is undergoing perpetual destruction and renovation. It can be observed to disappear, and its further formation may be prevented under the following conditions:—(1.) By causing an animal to die slowly by starvation, or by dividing the pneumogastric nerve; (2.) When the function of the liver is disturbed by severe, and especially by acute diseases.

The following is the course described by Bernard as that taken by the sugar secreted in health by the hepatic cells:—

> "It passes with the blood of the capillaries into the hepatic veins, and thence into the *vena cava ascendens.* It is at the point of discharge of the last-named vessel that the blood is the most strongly saccharine; it then becomes mixed with the blood from the lower parts of the body, and passes up to the right auricle, where the sugar undergoes a new dilution from its admixture with the blood of the *vena cava descendens.* From the right auricle it passes into the right ventricle, and thence to the lung. In the whole of the route from the liver to the lung the blood is constantly saccharine, but the amount of sugar varies extremely, and is least at the greatest distance from the liver. In the lung the sugar, being brought into contact with the air, and mixing with the whole mass of the blood, sometimes completely disappears.
>
> "These two organs, then—the liver and the lung—stand in an inverse relation to one another, in so far as the saccharine matter is concerned. In a fasting animal, for example, the blood which arrives at the liver contains no trace of sugar, while that which leaves it is distinctly

saccharine. Inversely, the blood which arrives at the lung contains sugar, while that which leaves it contains no traces of this constituent. The sugar in this physiological state remains hidden between the liver and the lung, and this is the reason why its existence and formation within the animal body were not earlier discovered. The analysis of blood drawn from superficial veins would fail to detect it under these conditions" (*Med.-Chir. Review*, p. 32, January, 1857).

But under some conditions even in health, and therefore called "physiological conditions," sugar may be found in the blood beyond the lungs. The activity of the glycogenic function being increased with an augmented flow of blood to the liver, such as that which takes place after a meal, it is found that *four* or *five* hours after the commencement of intestinal digestion the production of sugar in the liver attains its maximum. For three or four hours at this time the production of sugar exceeds its destruction, so that "at this period of digestion we find sugar in all the vessels of the body, both arteries and veins; and we find it in the renal arteries, but in too small quantity to pass into the urine. This active and increased flow of blood through the liver thus displaces the sugar previously found there, and projects it into the circulation, and at the same time acts as a stimulus to the liver, which is still further excited by the nervous system under the influence of digestion. This increase of sugar is thus altogether independent of the nature of the food."

The cases of so-called "intermittent diabetes" are partly explicable by this interesting observation, in which the urine of digestion is saccharine, while no sugar can be detected at other periods, as in old persons observed by Bence Jones (*Med.-Chir. Trans.*, vol. xxxvi., p. 401).

The influence of diet is thus far remarkable, that the formation of sugar diminishes when fatty kinds of food are used—a fact supposed to be explained by the circumstance that the fats are absorbed directly by the lacteals, and thus do not affect the portal blood. It is also observed that in health the ingestion of amylaceous or saccharine matter does not augment the quantity of sugar in the liver, nor in the animal economy generally, although in cases of diabetes the use of these saccharine substances commonly causes a great and immediate augmentation of the sugar in the urine.

It does not yet appear clear how the sugar is destroyed in the blood in health. The destruction has been ascribed—(1.) To the oxidation or combustion of sugar in the lungs; (2.) To its combustion effected through the agency of an alkali; (3.) To the influence of extreme division in the blood, through which it may be converted into lactic acid by a simple molecular change.

Connected with the formation of sugar in the liver, Bernard shows that two substances are concerned—(1.) One soluble in water; (2.) A substance slightly soluble in water, and which remains fixed in the hepatic tissue after all blood and sugar have been removed by prolonged washing. While, therefore, Bernard's observations showed that the blood which left the liver by the hepatic vein contains a peculiar substance of a saccharine nature, which does not exist in the blood brought to the organ in a state of health by the portal vein, the more recent researches of Dr. Pavy, of Guy's Hospital, have shown that the liver does not actually form sugar, but a substance that becomes sugar almost immediately on coming in contact with albuminous matters. "It is especially destined as a *pabulum* or fuel for the combustion process, being usually eliminated from the blood in the form of carbonic acid and water during its passage through the lungs, so as not to pass into the systemic circulation unless either its quantity be unusually great or its elimination interfered with by imperfect respiration. It appears to be elaborated by the converting power of the liver, either from materials supplied by the food or from the products of the waste of the system (Carpenter, p. 308; *Animal Physiology*, Bohn's edition).

Connecting all these observations together—namely, Parkes, Lehmann, Bernard, Pavy, Basham—may it not be suggested, as a topic of investigation, whether or not the elaboration of this material by the liver is not arrested in Bright's disease and increased in diabetes, in the active stage of which disease it ultimately comes to be eliminated by the kidney as sugar in the urine?

The ultimate result of Bernard's investigations shows that the increased formation of sugar by the liver, and its presence in the blood, is the result of some exciting cause which, acting by reflex action, conveys the stimulus to the *medulla oblongata*, whence it is propagated by the spinal cord and filaments of the great sympathetic nerve to the liver, and so excites its *glycogenic* function. This doctrine is supported by the following experiment of Bernard:—

> "When we prick the mesial line of the floor of the fourth ventricle, in the exact centre of the space between the origins of the auditory and pneumogastric nerves, we at the same time produce an exaggeration of the hepatic (saccharine) and of the renal secretions; if the puncture be effected a little higher, we very often only produce an augmentation in the quantity of the urine, which then frequently becomes charged with albuminous matters; while, if the puncture be below the indicated point, the discharge of sugar alone is observed, and the urine remains turbid and scanty. Hence it appears that we may distinguish two points of which the inferior corresponds to the secretion of the liver, and the superior to that of the kidneys. As, however, these two points are very near to one another, it often happens that, if the instrument enters obliquely, they are simultaneously wounded, and the animal's urine not only becomes superabundant, but at the same time saccharine" (*Med.-Chir. Review*, l. c., p. 42).

He found also that in cutting the pneumogastric nerves the secretion of sugar was stopped, but that it still took place when the floor of the fourth ventricle was irritated, after the division of the pneumogastric. Any irritation conveyed through this nerve may thus induce the diabetic state. The following are the general circumstances under which the phenomena of *melituria* become developed:—Any agents or conditions which cause a suspension of the functions of animal life, while the purely nutritive or organic functions remain intact, may bring about *melituria*. Thus the Indian *worari* poison acts; so does apoplexy produced by a blow on the skull. Local irritation of the liver itself, as Dr. Harley has shown, may also induce the condition. By the injection of alcohol and ether into the *vena portæ,* Harley was able to induce diabetes. The internal use of arsenic and quinine has also been said to have induced saccharine urine; and thus it is not improbable that irritant substances absorbed from the bowels by the mesenteric veins may sometimes bring about the morbid state.

In spite of all these theories, the immediate cause of diabetes still remains obscure, and its beginnings are a mystery.

According to Griesinger, diabetes occurs much more frequently in males than in females—nearly in the proportion of three to one. It occurs most frequently between the ages of thirty and forty in males, and in females between those of ten and thirty. The disease shows sometimes a hereditary origin and transmission. The usual recognized exciting causes are exposure to cold and wet, concussions of the body, immoderate use of sugar, new wine and fruit, great and continued mental exertion, mental depression, alcoholic intoxication.

Morbid Anatomy.—No constant lesion is found. There is no remarkable lesion in any part of the nervous system. Sometimes the floor of the fourth ventricle is diseased and sometimes healthy—nothing is constant.

The pancreas is very often hypertrophied. The walls of the stomach are thickened, chiefly through muscular hypertrophy, with the mucous membrane thickened and softened.

The lungs are almost always tuberculous, or contain caseous deposits, and not unfrequently there is puenmonia or gangrene.

Of the secondary lesions, the lungs are most frequently affected. In thirty-one out of sixty-four fatal cases, or in nearly a half, tubercle was found (GRIESINGER). But Pavy and Wilks do not believe the lesion to be that of tubercle, but the result of a chronic inflammation, by which the lung tissue is broken down and cavities form.

In about half the cases the kidneys are enlarged, and sometimes are in a state of chronic parenchymatous inflammation, as in some forms of Bright's disease.

The liver usually appears normal, but is sometimes congested with blood. The bile resembles a mixture of rhubarb, and deposits a copious sediment of columnar epithelium and yellow amorphous granular-looking matter.

Symptoms.—The early symptoms of *diabetes mellitus* are obscure. Dr. Prout believed that there is a stage which precedes the formation of sugar, and which is marked by a superabundant and highly dense urine, loaded with an excess of urea. But much uncertainty prevails on this point, and nothing is assured except that the constitution is not greatly affected till the saccharine matter forms and is eliminated by urine. In some very few instances the quantity of urine passed is hardly greater than in health; but more commonly it is in great excess, amounting to eight, ten, sixteen, thirty, and even more pints during the twenty-four hours; so that the patient is incessantly disturbed in the night, and loses his sleep, while the urethra and its orifice become inflamed and sore.

The onset of the disease is thus generally insidious and unobserved. There may be a sense of general discomfort, some emaciation may declare itself, while constant thirst and frequent micturition become well marked symptoms.

At this period the general health begins to give way; thirst is intense, and the patient often drinks many quarts, or even gallons, in the course of the day. The quantity of water drunk has been believed to be less than the quantity of urine passed,—in some instances only as one to four; but this statement, however, is not now believed to be correct. From the careful experiments of Dr. Parkes and others to determine this point, it appears that the quantity of urine is actually *less* than the quantity of water taken in liquid and solid food; and this is more especially seen to be the case if long periods (ten or twelve days) are taken for examination. Water is often retained for some time in the body of diabetics; and if an observation be made in a single day, it may happen that some of the urine retained from the previous day is then poured out. It is such retention of water which may account for the apparent excess of urine over drink (Parkes *On the Urine*, p. 339). The appetite is capricious, but generally excessive and voracious, the skin harsh and dry, and the patient becomes greatly emaciated, and loses sexual desire and sexual power. Almost all the water drunk passes off by the kidneys, and the insensible perspiration is diminished both by skin and lungs. The intestinal excretion of water is also greatly lessened; hence the bowels are costive and the fæces dry and hard. The water is not passed off by the kidneys so soon as in health. If a diabetic person drink water in the morning, the urine may not be increased till mid-day; but if grape-sugar be added to breakfast, the urine is passed as rapidly as in health. In advanced cases the drain upon the constitution is so great that the alveolar processes are absorbed, and the teeth, loosened in their sockets, are apt to fall out. These symptoms are much relieved by medicine, and life much prolonged; but often, when the case appears most favourable, tuberculosis becomes apparent, and the patient sinks under this disease.

The emaciation of diabetes is progressive, and the muscles become atrophied, as well as the adipose tissue. Even the heart is said to suffer. The skin is persistently dry, but on the appearance of fever, perspirations set in.

The digestive organs become weak and feeble, the inordinate appetite diminishes, and food is loathed. Nausea is frequent—a sinking at the pit of the stomach is complained of—the bowels become constipated, and the fæces pale. A short dry cough becomes frequent, the index of commencing disease of the lungs; and the patient generally suddenly sinks from exhaustion, or from coma or convulsions.

Condition of the Urine.—When *diuresis* is considerable, the urine should invariably be examined, and its constituents determined. A faint sweetish odour may be percepible in diabetic urine, comparable to fresh hay or milk. The chamber-vessel should be examined for crystals of sugar which may have been formed (fig. 2). The best evidence, however, is that derived from chemical tests.

A portion of urine, which is usually of a light-straw colour, should be taken, and its specific gravity determined; and if greater than 1·020, it should be evaporated, and if sugar be present we shall have a dark-brown residue, something like treacle. This extract, like the natural sugars, consists of crystallizable matter and of an uncrystallizable syrup; and to separate them Dr. Christison recommends that the extract be agitated with rectified spirit, and the residue boiled in another portion of the same fluid, when, on cooling, the crystallizable sugar will separate in light-greyish grains like grape-sugar. Again, if sugar should be suspected to exist, even in minute quantity, a small portion of yeast should be added to a small quantity of the urine, when, if sugar be present, fermentation will ensue, and each square inch of carbonic acid given off corresponds nearly to one grain of sugar. This test is so delicate that one part of diabetic urine, according to Dr. Christison, may be detected in 1,000 parts of urine of the density of 1·030. The most certain and approved test is that known as "*Trommer's test*," based on the re-action of the salts of copper. A portion of urine in a test-tube is to be treated with *one* or *two* drops of a solution of *sulphate of copper*, and afterwards a considerable excess of *potash* is to be added. The dark-blue solution which results is then to be held over the spirit-lamp, and boiled for a moment, when a yellowish-brown *precipitate* of the sub-oxide of copper is produced. Dr. Roberts expresses a want of certainty in the results of most of those tests. He recommends Fehling's copper solution as the best test. It consists of Sulphate of Copper, gr. viii.; Tartrate of Potash, ʒss.; Liquor Potassæ, ʒi. Some of this test solution is to be boiled, and some drops of the suspected urine added to it. If sugar be abundant, a thick yellowish opacity and deposit of yellow sub-oxide are produced, which changes to a brick-red at once if the blue colour of the test remains dominant. If no such re-action ensues, more urine is to be added, until a quantity equal to the bulk of the test employed has been poured in. The whole must be again heated to the boiling point; and if no change occurs, it is to be set aside without further boiling. As the mixture of the urine and test cools, if no milkiness is produced, the urine may be confidently pronounced free from sugar. No quantity *above a fortieth of a grain* can escape such a test, and any quantity below that does not appear to be of clinical importance. It is important to bear in mind that Fehling's test solution must always be freshly prepared. For the application of further tests, see Dr. Beale's Tables, p. 21.

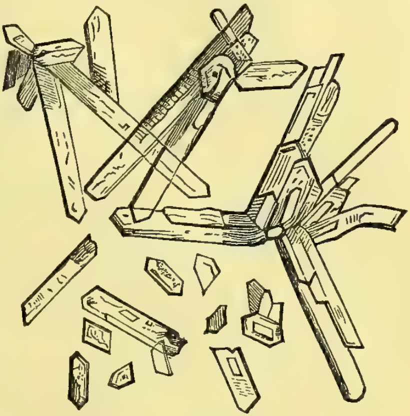

Fig. 2.*

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* Crystals of diabetic sugar from diabetic urine (after **Beale**, p. 150).

The density of diabetic urine, however, is one of the best indications. This fluid varies in density from 1·030 to 1·074 (Becquerel); but on an average, and tolerably constantly, it is 1·040; and when the urinometer stands above 1·030, we may suspect that sugar is present. The quantity of sugar present has been calculated by Dr. Henry, in urine of density 1·020, to be 402 grains in every pint, while at 1·050 it contains 958 grains of sugar—the increment being, as he conceives, one scruple, or nearly so, for every degree of specific gravity between the extremes that have been mentioned. If these data be correct, a person passing sixteen pints of urine daily, of specific gravity 1·050, actually passes nearly two pounds avoirdupois of sugar. But the amount varies greatly, amounting sometimes to *one* pound or *two* pounds, or even two and a half pounds, in twenty-four hours. In a few months patients will pass their own weight in sugar, (Parkes, l. c.) Its amount is mainly influenced, in the first instance, by the saccharine and amylaceous nature of the food, which always augments the amount of sugar. The augmentation is quite perceptible about two hours after food, and continues for four or six hours, if the amount of starch taken has been considerable. It is probable, though it is not absolutely proven, that all the starch eaten is converted into sugar; and in cases accurately observed by Mr. Graham and Dr. Parkes for a considerable length of time, the quantity of sugar excreted by the urine never exceeded the amount of starch; and almost all the starchy food was accounted for by the diabetic sugar (Walshe, Parkes). It has been supposed by some that when the starch only is converted into sugar, it is the earliest stage of the diabetic state; and it is certain that during the progress of some cases sugar begins to be formed from other sources and in other ways, and is no longer derived solely from the starch.

In some cases the sugar may recognize no other origin than the starchy food for many years. Such seems to have been the case of the late Mr. Camplin, of Finsbury Square, who kept his disease at bay for ten or twelve years, (*Med.-Chir. Trans.*, vol. xxxviii., p. 69; Parkes, l. c.) Complete fasting or abstinence from saccharine and starchy food for eight or twelve hours reduces the quantity of urine to the normal amount, and brings down the specific gravity to the usual figure. Sugar can then neither be detected in the urine nor in the blood, as was determined by Dr. Parkes after numerous analyses of blood under such circumstances (*On the Urine*, p. 348).

In the majority of advanced cases of diabetes, sugar is not only produced from starch, but also from *nitrogenous* foods, especially *gluten* and *animal food*. In such cases complete abstinence for a time from food lessens, but does not entirely remove, the sugar from the urine. Perhaps in all such cases the formation of sugar from albuminous food indicates a more advanced or active stage of the disease than when the sugar is formed from starchy compounds only; and it is probable that the amount of sugar from this source increases as the disease advances. Dr. Parkes refers to a case related by Schultze, in which the amount of sugar in the urine when the patient was on mixed diet was *one-third* more than could have been furnished by the starch food alone; and the amount can always be ascertained by keeping the patient strictly on a meat diet, and feeding him with starch from time to time as an experiment (l. c., p. 348).

Thus, three conditions affecting food would seem to be present in cases of diabetes:—

The earliest and fundamental one continues to act throughout, and is distinguished by complete arrest of the normal metamorphic changes of starch and sugar—an arrest probably associated with some substance in the digestive canal which absorbs the sugar. At first it affects the sugar derived from starch food, then it affects the sugar derived from nitrogenous food. Dr. Bence Jones suggests that in some cases the changes may pass one step beyond the conversion of starch into sugar, and produce "vegetable acid." He believes a disease exists which is thus allied to diabetes, and characterized by a great amount of acid, probably *lactic*, in the system and in the urine. The chief transitional changes of starch enumerated by Dr. Bence Jones are as follow:—Starch into sugar, into vegetable acid, into carbonic acid; and he supposes the arrest to occur at the acid stage, instead of at the sugar stage, in the morbid condition he describes.

The second condition is an abnormal production (probably in the liver) of sugar from nitrogenous food—*i.e.*, an amount of production which is abnormal—combined with arrest in the transformation of sugar normally formed (PARKES). A diabetic patient, kept on the most rigorous meat diet, has been known to pass a quantity of sugar corresponding to *two-fifths* of the entire meat food, or *three-fifths* of the *albuminate* contained in the meat (GRIESINGER, quoted by PARKES).

A third condition may be also distinguished—namely, one in which the *tissues* themselves, and especially the muscles of the body, contribute to the morbid formation of sugar. This occurs under circumstances of extreme inanition, when almost no food is being supplied to the body, and yet a constant quantity of sugar is eliminated. Traube, Parkes, and Ringer have each recorded such cases. In one of Dr. Parkes's cases, the blood, after seventeen hours' fasting, was found to be still very rich in sugar (PARKES, l. c., p. 350). It is, however, a question whether this is an abnormal formation of sugar merely, or a further and more advanced stage of the disease. There are also cases which may be noticed here, in which the patients continue to lose ground although the quantity of sugar lessens, and in which the substance known as *inosite* or *muscle-sugar* is found in the urine (fig. 3). It increases in quantity as the sugar lessens, and at last as much as eighteen and twenty grammes of pure *inosite* have been procured from

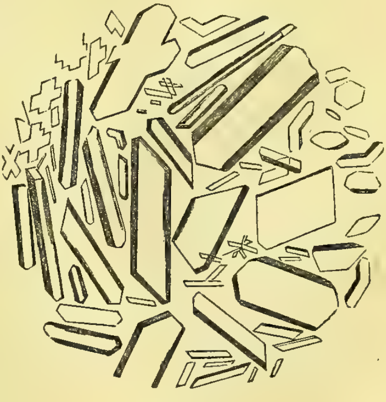

Fig. 3.*

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* *Inosite* or *muscle-sugar*, crystallized partly from alcohol and partly from water (after FUNKE).

the day's urine. The *inosite* may be obtained in the form of colourless prismatic crystals, which are efflorescent. It does not reduce the oxide of copper to the state of sub-oxide, as is the case with *diabetic sugar* and *grape-sugar;* and it is said to have not quite the same composition as the latter substance, but is represented by C_2 H_2 O_2; so that one atom of *grape-sugar* would thus represent six atoms of *inosite.* It may be detected by evaporation of the suspected fluid nearly to dryness in a platinum basin, when, if a little ammonia and chloride of calcium be added, a rose colour is produced, especially if the mixture be again concentrated by evaporation. M. Hohl records a case of diabetes in which, while the proportion of sugar gradually diminished, the *inosite* gradually increased in amount till upwards of 300 grains were passed in the twenty-four hours (PARKES, BEALE). The observation is one of great interest in connection with the pathology of this remarkable constitutional disease.

Professor Sidney Ringer, of University College, has made some observations of great interest, to show the amount of *urea* and of *sugar* respectively furnished by the tissues of the body and by nitrogenous food. (1.) During inanition one series of observations showed an enormous disintegration of tissues (48 grammes of urea and 105 grammes of sugar being passed in twenty-four hours), the relation between the urea and the sugar being tolerably constant. In the second series, when nitrogenous food was taken, the urea increased about the third hour after food, and reached its maximum about the fifth hour, after which it continued to diminish, and reached the inanition amount in the eighth hour. The sugar followed the same rule, and almost in an exact ratio; but the urea was in slight relative excess to the sugar, showing that the nitrogenous food raised the urea slightly more than it did the sugar. During inanition the urea was to sugar as 1 to 2·235, while after nitrogenous food the urea was to sugar as 1 to 1·9. It thus appears certain, as Dr. Parkes observes, that there is some close connection between the amounts of urea and of sugar in such cases as diabetes. The amount of urea may be double or even treble the normal amount, and that to an extent much greater than can be accounted for by the food taken, and due probably to some peculiarity in diabetes, causing heightened metamorphosis of tissues, such as might arise from the excessive action of oxygen on them (PARKES, l. c., p. 342). The amount of sugar is still further increased if diabetic patients take more water than their thirst demands; and it is probable that the urea is also increased, although exact experiments are wanting.

On standing, diabetic urine soon begins to ferment, with the appearance of *lactic, butyric, acetic,* or *formic acids,* and develops the yeast plant; and during this fermentation the urea entirely decomposes. Dr. Christison gives the following formula for ascertaining the amount of solid matter in *diabetic urine:*—"Multiply the excess of the specific gravity over 1·000 by 2·33, the result is the number of parts of solid matter in 1·000 of urine."

Diabetes is "chronic in its course, usually lasting from one to two years, though it may run on for six, eight, or ten years. Becquerel mentions the case of a boy, nine years old, who died in six days, and

Roberts a child of three years, who died in three weeks. Of 100 fatal cases collected by Griesinger, the duration was:—

Under 3 months,	1
Between 3 and 6 months,	2
“ 6 ,, 12 ,,	13
,, 1 ,, 2 years,	39
,, 2 ,, 3 ,,	20
,, 3 ,, 4 ,,	7
,, 4 ,, 5 ,,	2
,, 5 ,, 6 ,,	1
,, 6 ,, 7 ,,	2
,, 7 ,, 8 ,,	1
Undetermined,	12

The most common *complication* is tuberculosis, it occurring in one-half of the cases of diabetes protracted to the first year (Roberts). It is often quite acute. “A low and fatal type of inflammation of the lungs, pleura, or peritoneum is not infrequent after the disease has lasted some time.” In every tissue of the body there exists a tendency to asthenic inflammation, apt to run into abscess, diffuse suppuration, sloughing, phagedenic ulceration or gangrene. Boils, in successive crops, and even carbuncle, may appear in the course of the disease. In one of Dr. Roberts's cases, boils were an initial symptom. Spontaneous gangrene of the lower extremities, with obstruction of one or more arteries of the limb, is not infrequent in diabetes (Marchal de Calvi). Defective vision is met with in a certain number of diabetic patients. Cataract occurs in some long-standing cases; its frequency is variously stated. Of 225 cases collected by Griesinger, there was cataract in 20, but of his own 7 cases, it was present in 3; von Gräefe states the proportion as 1 in 5; Bouchardat, 1 in 38; of 45 cases treated by Roberts, there was cataract in only 1; and Garrod says that in the large number of cases of diabetes he has treated he has never once seen it. Diabetic cataract is nearly always soft, though examples of the hard kind have been reported by Wilde, von Gräefe, and Guersant. Roberts thus describes it:—“It comes on generally after the diabetic state has lasted eighteen months or two years; but it has been known to appear in six months. Its course is rapid; the two eyes may become completely cataractous in a few days; sometimes it is developed more slowly.” It begins in one eye—generally in the right,—but soon involves the two. Drs. S. Weir Mitchell, of Philadelphia, and B. W. Richardson, of London, have endeavoured to show that it was due to physical imbibition by the lens of the saccharine matter of the aqueous humor; but there is no proof of this. Hepp and Fischer both failed to find sugar in cataractous lens removed from diabetic subjects, and there are other reasons against the reception of this theory, particularly the infrequency of its occurrence, the long delay of its appearance, and its being occasionally unilocular.

Dimness of sight, from supposed functional disorder of some of the internal structures of the eye (amblyopia) happens, according to Bouchardat, in about one-fifth of the cases of diabetes. Fauconneau Dufresne found the sight more or less affected in 20 out of 162 cases. Generally it is slight, temporary, and often recurrent. It is rarely a permanent affection, and when so, ends in total blindness, and betokens a speedy fatal termination. In such cases the retina is said to be atrophied (CLYMER).

Prognosis.—The ultimate issue of every case of diabetes is probably fatal; at least the number of cases in which the urine is rendered permanently natural is extremely small, and many of them, at the moment the disease seems to have yielded, die of phthisis. Even when the presence of the saccharine principle has been so far conquered that it alternates with lithic acid deposit, or that lithic acid becomes the prominent feature, the circumstance is anything but favourable, for such individuals generally die of some sudden and overwhelming attack of internal inflammation or of apoplexy (PROUT). On the other hand, where the source and nature of the mal-assimilation can be discovered, and either rectified or held in check, and if the patient will submit to regimen, a favourable result may be hoped for in some cases. A "not inconsiderable number, however, recover completely, and many more attain to a state of conditional amelioration—that is, an amelioration which is conditional on the observance of a certain diet and regimen." Dr. Trousseau says:—"By hygiene and a proper regimen, aided by the action of certain remedies, wisely and prudently used, we may hope to cure a few and to relieve a large number of diabetic patients, if the disease has not reached the last period." The younger the patient the less is the chance of ultimate recovery. Dr. Roberts states that all the cases under twenty which he has seen have eventually died of the disease. In all persons the symptoms may continue for years, with a tolerable state of health. In corpulent persons the prognosis is more favourable than in those of spare habit. Saccharine urine, without excessive urinary secretion, is less serious than when the secretion is very great. "*Cœteris paribus*, the longer the disease has existed, the more unfavourable the prognosis; *cœteris paribus*, also, the greater the general severity of the symptoms, the less is the hope of amendment. Cases which can be traced to mental anxiety and traumatic lesions appear to be somewhat more hopeful than those for which no tangible cause can be assigned" (ROBERTS). Great diminution in the amount of, or the total disappearance of, sugar in the urine, when saccharine and amylaceous articles of diet are not used, is a favourable sign; also a soft, perspirable skin, with a moderate appetite, or the symptoms generally remaining stationary. Albuminuria, thoracic and intestinal complications, and permanent cataract or amblyopia, are always fatal (CLYMER).

Treatment.—There are few diseases in which the treatment has been more varied than in *diabetes mellitus*. Every conceivable medicine has been given, but exact quantitive determinations of the effect of them on the sugar have rarely been made. The emaciated state of the patient presents an insurmountable obstacle to bleeding. Nevertheless, this mode of treatment has often been practised, and as much as 160 to 170 ounces of blood have been taken in a few weeks. The late Sir Henry Marsh found blood-letting of service when the disease was recent, and the strength of the patient still maintained. He found that it promoted the action of diaphoretic remedies; and especially when followed by the tepid bath. He found leeches to the epigastric region of benefit, when internal heat with fulness and tenderness prevailed there, with a "gnawing" feeling about the stomach. In the face, however, of so high an authority, the pathology of diabetes, as given in the text, does not sanction general blood-letting as a mode of cure.

Mercury, alike with *lead*, *antimony*, *zinc*, *silver*, and *copper*, are indifferent as to their influence on the formation of sugar (PARKES). Opium has been given to the extent of 100 grains in the twenty-four hours; but with an equal want of success, although it does seem to lessen the sugar, probably by lessening the appetite and hindering the taking of food (PARKES). The whole *materia medica* has been exhausted in search of a remedy for this disease. The metals, the fixed and the volatile alkalies, the vegetable and mineral acids; the astringents, purgatives, tonics, diaphoretics, and diuretics, have in their turns been administered, and each has perhaps afforded some relief; but the disease has proceeded, and finally, it may be said, nearly every patient dies whose treatment is left entirely to drugs. Dr. Prout, who considered it merely as a form of dyspepsia, conceived that each case requires a different treatment. In the early stages of the disease, the late Drs. Bullar, of Southampton, have found great benefit from the *tincture* of the *muriate* of iron, now called the *tincture of the perchloride of iron.* Dr. Camplin speaks of the *citrate of ammonia* in the effervescent form, generally combined with the *citrate* of iron, as more useful than any other medicine; while bitters and alkaline remedies did him great service at one period of his attack.

Opium combined with ipecacuanha is eminently useful as a sedative, especially in the form of Dover's powder; while exercise, warm clothing, friction of the surface, hot bathing, and diaphoretics, improve the cutaneous functions. The functions of the skin must always be inquired into, and must be kept active. The nitro-muriatic baths, and the internal administration of the acid, might also be employed with some prospect of benefit in cases requiring acid tonics. This is more generally the case in the young, and in the early stages of the disease, in which the *tincture of the perchloride of iron* is also of service, prescribed in the *infusion of quassia* or of *calumba.* But each case requires to be made a special study, considering that many, or at least several, organs may be concerned in the disordered working of the system (PROUT, CAMPLIN). The *mistura ferri composita* is another of the iron preparations which has been found of service. The administration of alkalies, as recommended by Miahle and Contour, is also occasionally successful.

The little benefit derived from medicine induced Dr. Rollo to try the effects of an entirely azoted or animal diet; and out of nineteen cases, two are said to have been cured by this means. A full and generous diet is unquestionably useful in these cases; but the patient soon gets disgusted with mutton or beef, or both, for breakfast, dinner, and supper: he consequently nauseates a meat diet, and abandons it altogether. A diet of salt fish has been attempted; but the patient in a short time so loathes it that it has to be given up. A mixed diet, therefore, if contra-indicated by some theories, is at least the best to adopt in practice, and is consistent with the remarks made under pathology, if duly regulated and aided by other means. It will be evident, however, that those vegetables which contain a large quantity of saccharine matter should be avoided in some degree, as potatoes, grapes, or other very ripe fruit, and, *à fortiori*, sugar itself.

"There are therefore," as Dr. Camplin justly observed, "certain fixed broad principles upon which the disease is to be treated." In all cases the various influences of the stomach, liver, skin, and kidneys on the nervous system and on each other, ought to be sought out and determined, and the basis of treatment arranged accordingly. *It is necessary to abstain from all amylaceous food, as well as from every solid and liquid containing sugar, or any substance readily convertible into sugar.* Fat meat and eggs may be taken, if biliary derangement is not induced by them, and fish is a most important article with which to vary the monotony of the dietary. Milk also may be indulged in occasionally, as it is not found that the sugar it contains is readily converted into glucose. Its influence, however, requires watching. It is desirable to vary the food as much as possible during the day, taking the lighter kinds in the later meals. When soups are taken, they ought to be really good, and flavoured with aromatics or onions, to the exclusion of carrots, turnips, and pease. They may be thickened with some bran *finely powdered.* Lettuces Dr. Camplin found to agree well, when eaten sparingly with oil and vinegar, or with a little salt only, if the vinegar is likely to disagree. Pickles in small quantities may be permitted to convalescents. If cocoa agree, it may be taken, prepared *from the nibs* only. With regard to drinks: if milk is found to agree, it may be used as a drink, combined with half its bulk of lime-water, or in the form of what is known as "buttermilk" in Scotland and in Ireland, but which in England is generally given to pigs, not yet being sufficiently appreciated by natives of the country south of the Tweed. Dr. Camplin eventually found it necessary to abstain from all alcoholic drinks; but, where they are found desirable or necessary, a selection may be made from those wines and spirits which are freest from sugars. Of these, clarets may be chemically considered the best, then Burgundy. The so-called "fruity wines" must be entirely interdicted; and of all alcoholic beverages *weak* brandy and water is the safest. The amount of brandy must be always *measured* and taken as directed by the medical attendant. A *tea-spoonful* in a tumblerful of water is generally sufficient for an ordinary dinner drink; and Dr. Camplin candidly and feelingly observes, from his own experience, that no diabetic need expect to recover or continue well who cannot exercise self-control, and make up his mind to be temperate *in all things.*

Seeing that under this system of diet the patient is deprived of the use of ordinary bread, Dr. Camplin devised a form of bread prepared solely from *bran;* and the great value of *bran cakes* as a substitute for bread in cases of *diabetes* has now been established by the experience of so many individuals that its use ought to be insisted on. The bran used should be *thoroughly washed,* so that it may be as free from starch as possible; and *finely powdered,* so that it may not irritate the susceptible mucous membrane of the intestines. Such carefully-prepared and finely-powdered bran may be obtained from Mr. Batchley, of 362

Oxford Street, London, near the Pantheon; also of Mr. Donges, Gower Street, London, North. But if it is desirable to prepare the powder at home as it is required, a special mill and sieve for this purpose are necessary, and may be obtained of Messrs. Evans Brothers, 54 Bricklane, Spitalfields, London, E. (Dr. Camplin's *Monograph on Diabetes.*)

The *formula* for bran cakes is thus given by him:—"Take a quantity of wheat bran (say a quart). Boil it in two successive waters for a quarter of an hour, each time straining it through a sieve; wash it well with cold water on the sieve, until the water runs off perfectly clear. Squeeze the washed bran in a cloth, as dry as possible, then spread it thinly on a dish, and place it in a slow oven. When it is perfectly dry and crisp it is fit for grinding into fine powder.

"The bran thus prepared is ground in the mill for the purpose, and must be sifted through a wire sieve of such fineness as to require the use of a brush to pass it through, and what remains on the sieve must be re-ground till it is sufficiently soft and fine.

"To prepare a cake, take of this bran powder three or four ounces, three new-laid eggs, one and a half or two ounces of butter, and about half a pint of milk. Mix the eggs with a little of the milk, and warm the butter with the remainder of the milk; stir the whole well together, adding a little nutmeg and ginger, or any other spice that may be agreeable. Bake in small tins (pattipans, which must be well buttered), in a rather quick oven, for about half an hour. The cakes when baked should be a little thicker than a captain's biscuit.

"These cakes may be eaten with meat or cheese for breakfast, dinner, and supper, and require a free allowance of butter; and the cakes are more pleasant if placed in the oven a few minutes before being placed on the table.

"When economy is an object, when a change is required, or if the stomach cannot bear butter, the cakes may be prepared as follows:—Take of the prepared bran four ounces, three eggs, about twelve ounces of milk, with a little spice and salt, to be mixed and put into a basin (previously well buttered). Bake it for about an hour; the loaf may then be cut into convenient slices and toasted when wanted; or, after slicing, it may be re-baked, and kept in the form of rusks.

"Nothing has yet been discovered of equal utility to these bran cakes, combining, as they do, moderate cost with freedom from starch, and sufficient pleasantness as an article of food" (Camplin *On Diabetes*, third edition).

Consistently with the experiments of Bernard, "cod-liver oil holds out some prospect of a natural plan of treatment by its use."

Dr. Pavy recommends ground almond powder, made into biscuits, rusks, and bread, with eggs, as a substitute for ordinary bread. Mr. Hill, 60 Bishopsgate Street, London, makes such biscuit.

An abstinence from water lessens the formation of sugar; but it probably accumulates in the body, so that when fluid is again given, an excessive elimination of sugar occurs (RINGER, GRIESINGER); and patients become extremely depressed and ill if water is withheld from them, probably from the impregnation of the body with sugar (PARKES). Coffee lessens the sugar, but increases the urea. Rennet, as recommended by Dr. Gray, of Glasgow, at first lessen the sugar and water;

but they afterwards increase again. Warm baths lessen the amount slightly; so does bicarbonate of soda. Hence the recommendation of Miahle and Contour.

When the diabetic symptoms subside, congestions, especially of the head, are apt to supervene. Such congestion Dr. Camplin found to subside gradually under the use of *citrate* of *ammonia* and small doses of *colchicum wine*.

The great difficulty in the treatment of diabetes is to manage the dyspepsia and impaired digestion, and, at the same time, to diminish and keep in check the formation of sugar. Warm flannel ought to be worn next the skin in all cases, and residence in a warm climate will often be of service as an aid to the means of cure employed.

"However surely," writes Dr. Clymer, "an exclusive animal diet may lessen, or entirely remove, the sugar in the urine of a diabetic patient, it is certain that it cannot long be tolerated." In this opinion Niemeyer, Trousseau, and many other physicians now concur:—"Under its use, the appetite fails, and a loathing of all food soon happens. The necessity of a mixed diet for man is as necessary for him when suffering from diabetes as in a state of health. In this disease the nervous system is undoubtedly implicated, and a rigid adherence to animal food alone, were it practicable, would soon be followed by an aggravation of the nervous troubles. There is, from the very beginning, and throughout the course of the disorder, a strong tendency to devitalization, and this too must be guarded against. Fortunately the sugar in the urine may be kept down, and at the same time the general strength of the system maintained, by a properly adjusted diet of mixed food. Along, then, with the carnivorous dietary, whose importance is not to be undervalued, certain vegetables may be permitted, not only with impunity, but with advantage; and these are cabbage, cauliflowers, onions, spinach, water-cress, sorrel, endive, lettuce, &c. Trousseau has found no ill consequences from eating acid fruits, as strawberries, gooseberries, cherries, and he has allowed apples, pears, and grapes. He even suffers his diabetic patients to eat a small quantity of bread, if they greatly crave it; for, as he remarks, there are many persons who are unable to make a meal without it. Such a regimen is more likely to keep the disease stationary, and secure the general comfort of the patient, by upholding his strength, than by confining him to one kind of diet, which his stomach soon revolts against, and which must result in innutrition, general debility, the development of intercurrent affections, and sooner or later death. Enforced daily exercise in the open air, when possible, just short of fatigue, is as of much importance in the treatment of diabetes as diet. Gymnastics should be practised. Trousseau says that he has repeatedly seen, during the hunting season, diabetic patients, abroad with their gun and dog, cease both to drink and urinate to excess, and regain their strength, and even their virile powers. A suit of flannel or buckskin should be worn next the skin. Warm alkaline baths should be frequently taken, and an occasional Turkish bath will be found serviceable, if it produces no disposition to boils; or daily packing with the wet sheet may be tried, carefully watching the effects. The whole body should be hand-rubbed daily. Cream, so long as it agrees, may be permitted, and cod-liver oil is in many cases well borne, and would seem, as a nourisher, to do good. The alkaline theory has been proved to be false and absurd by Cl. Bernard and Poggiale; still the benefit of small doses of the alkalies, not continued, however, longer than a week or ten days at a time, is incontestable. They are best given in the form of the Vichy, Marienbad Kreutzbrun, or Carlsbad Spoudel water. Legroux thought he derived benefit from the administration of arsenic. Dr. Richardson, of London, has recommended the respiration of oxygen, and Bérenger-Féraud has shown that during its use there is great diminution in the amount of sugar in the urine (*Bul. de Thér.*, t. lxvii). The ethereal solution of the peroxide of hydrogen (ozonic ether) has been given with asserted benefit in half drachm doses in an ounce of distilled water. Dr. T. K. Chambers has prescribed iodide of potash on purely empirical grounds. On no pretext should any form of alcoholic drink be permitted. The preparations of iron are indispensable, and should be varied from time to time. To lessen the thirst and craving for food, small doses of opium are useful. By the use of the mixed diet recommended, particularly if fatty articles be taken, the obstinate constipation, so common in the earlier stages of the disorder, is often obviated without a recourse to drugs. Should this not be the case, castor oil in capsules, or rhubarb and podophyllin, must be prescribed.

"This mode of treatment, which secures a proper but varied diet, one that will not disgust the patient, but will nourish his body, though it may not remove the symptom glycosuria, will, in a large number of cases, give the diabetic a fair share of conditional health, so that, as Dr. Trousseau remarks, he would not know he had sugar in his urine, if an occasional chemical analysis did not remind him of its presence" (American edition).

PURPURA.

LATIN EQ., *Purpura;* FRENCH EQ., *Purpura;* GERMAN EQ., *Purpura;* ITALIAN EQ., *Porpora.*

Definition.—*A disease not unusually attended by fever, characterized by purple spots of effused blood, which are not effaced by pressure, and are of small size, except where they run together in patches.*

Pathology.—By some this disease has been considered identical with scurvy, and been named the "*land* scurvy." Others have considered it simply a disease of the skin—the *purpura simplex* of Willan and Bateman. Authors generally have spoken of the disease under the name of *petechiæ sine febre,* because the spots are not connected with continued fever. The disease is very closely related to *hæmorrhages* as a morbid state on the one hand, and to *scurvy* on the other.

Two varieties are distinguished, namely:—

(*a.*) *Simple,* and

(*b.*) *Hæmorrhagic;* which latter is thus defined by the College of Physicians of London—"*The disease when accompanied by hæmorrhage from a mucous surface.*"

It has not been determined what is the nature of the morbid state essential to purpura. In some cases the urine has been observed to contain an excess of albuminous matter, with a deficiency of urea (COMBE).* The blood has been found deficient in coagulating power.

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* An interesting case may also be referred to, related by Dr. Goodfellow in his admirable Lectures on *Diseases of the Kidney*, p. 74.

An examination of the blood in two cases of purpura, by Dr. Parkes, showed that, with a somewhat diminished proportion of the solid constituents in general, there was a remarkable increase in that of iron. A peculiar source of danger attends this disease in the occurrence of extravasation of blood into the internal organs. The lungs, the brain, the liver, and the alimentary canal are the most frequently affected. Purple spots and effusions of blood are to be found on the serous membranes, as in the *arachnoid*, the *pleura*, the *pericardium*, the *peritoneum;* and occasionally the blood lies coagulated in the cavities enclosed by these membranes. It has also been found in the bladder and in the calyces and pelvis of the kidney (CRAIGIE).

Instances occur in which blood oozes, or is discharged more or less copiously, from the mucous membranes without affection of the skin; and to this class of cases, as a form of disease now under consideration, are to be referred those cases of profuse or fatal hæmorrhage from slight causes, recorded under the name of "*hæmorrhea*," which have been ascribed to a *diathesis* termed the *hæmorrhagic*, and which is supposed to be hereditary, (*Edin. Med. and Surg. Journal*, vol. xxv.) When the disease is protracted the patient becomes of a sallow complexion, waxy coloured, and dingy; anasarcous swellings, gangrenous and bad sores appear on the feet and legs, general anasarca prevails, and he dies exhausted. Dr. Craigie considers that the appearance of purple spots in such cases is connected with more or less disease of the heart, especially with hypertrophy or dilatation of the ventricles. The following observation by Dr. Watson puts the pathology of purpura in an interesting and practical light:—

> "I have adverted to one peculiar source of danger in purpura—the hazard that blood may be effused in some vital organ, where even a slight amount of hæmorrhage suffices to extinguish life. Dr. Bateman states that he had seen three instances in which persons were carried off, while affected with purpura, by hæmorrhage into the lungs. During the course of one week, in the year 1825, I was present at two inspections in the dead-house of St. Bartholomew's Hospital, illustrative of the same point in respect to another vital organ, and involving a question in forensic medicine. The subjects of examination were both of them women of middle age, who had been brought into the hospital covered with purple spots and bruise-like discolorations, and suffering hæmorrhage from the mucous membranes. Each of these women declared that the apparent bruises were marks of beatings received from her husband. One of them became suddenly hemiplegic a little while before she died. Of the manner of dissolution in the other case I am not sure. In both instances a considerable quantity of blood was spread over the surface of the brain between its membranes; and in one of them blood had been shed also into the cerebral substance, which it had extensively lacerated.
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> "It may be worth mentioning that in one of these corpses there were indications either of unusually rapid putrefaction after death, or (what I think more probable) of some degree of decomposition even before life was extinct. This woman died in the evening, and the body was examined next day, twelve or fourteen hours afterwards. A quantity of fetid gas escaped from the cavity of the abdomen as soon as it was opened, and small bubbles of air were seen to ooze from the areolar tissue of

various parts of the body. Even when incisions were made into the *liver*, air frothed up, as it might do under ordinary circumstances from a section of the lungs" (*Lectures*, vol. ii., p. 866).

Symptoms.—Various symptoms, denoting general disorder of the constitution, precede the appearance of the petechiæ, generally by some weeks, such as languor, which is oppressive, weariness, faintness, and gnawing pains at the pit of the stomach. The appetite is variable, generally weak, but sometimes there is an inordinate craving for food, which, when eaten, is said to lie with a weight upon the stomach. The tongue is yellowish, and coated with a viscid fur, the countenance is sallow or dingy, or has a bloated, pale appearance, with swelling underneath the eyelids. The purple spots appear first on the legs, and afterwards, without any certain order, on the thighs, arms, and trunk of the body, and their presence is attended with much weakness and great depression of spirits. No degree of pressure alters the colour of the spots, and they are distinguished from flea-bites by the absence of a central puncture. At first the spots are bright red, but in a day or two they become purple, afterwards brown, and when they are about to disappear they assume a yellowish tint. When the disease continues for a long time, all these varieties of colour may be seen on a patient at the same time.

The pulse is feeble, and generally a good deal quicker than natural. Deep-seated pains are felt about the epigastric region, as well as in the chest, loins, or belly. In some instances giddiness and lightness of the head prevail, especially when attempting to move or to stand erect, and there may be even dull pain in some part of the head. Constipation of the bowels, palpitation and irregular action of the heart, with a tendency to frequent syncope, are the most destressing and dangerous symptoms.

Causes.—They are not well known. It is a disease more common in women than in men; and in boys before than after puberty. It has appeared under the long use of a poor diet, much sedentary occupation, watching, mental distress, and anxiety. It has also appeared under very opposite conditions; for example, in persons accustomed to the use of nutritious food and free living, but in whom there is reason to believe the digestive functions and the constitutional powers have been impaired by the persistent abuse of spirits or malt liquors.

Diagnosis is generally between *Purpura* and *Scurvy*. The following are the principal distinguishing features of each contrasted:—(1.) Scurvy is most common late in the winter or early in the spring; purpura in the first seasons of summer and autumn. (2.) In scurvy the gums are uniformly soft, sore, spongy, swollen, and hæmorrhagic: this is no common feature in purpura. (3.) In scurvy tumors of a painful kind form in the extremities, with stiffness or contractions of the joints: such symptoms do not attend purpura. (4.) Scurvy is marked by extreme debility, and is rendered worse by blood-letting and by mercury, and is cured by the administration of lemon-juice: not so with purpura, which may require blood-letting for its cure, is not benefited by antiscorbutic remedies, and is sometimes speedily cleared away upon the supervention of mercurial salivation and hypercatharsis (WATSON and WOOD).

Dr. Graves, of Dublin, has described an *exanthema hæmorrhagicum* in which febrile symptoms ushered in the malady, with an exceedingly dry and brown tongue, and a pulse which conveyed the sensation of small sharp vibrations with each beat—a kind of *wiry trembling*, with an extraordinary *thrill* and *hardness*, "yielding only to the withering influence of approaching death." Bleeding from the intestines and urinary system quickly supervened. These phenomena continued to spread till the whole mucous system of the patient became the seat of copious hæmorrhage. A peculiar exanthematous eruption appeared upon the skin, lasting for several days (five), and never presenting any signs of extravasation. Death followed in about four weeks. The pulse did not exceed its natural frequency (70), and there was no disturbance of the nervous system, such as headache, delirium, or want of sleep; and in the beginning of the disease the strength was not remarkably impaired; but subsequently yielded to loss of blood. The disease seemed almost exclusively confined to the arterial and capillary systems of vessels.

Treatment.—To treat this disease with success, it is necessary to ascertain the circumstances under which it becomes developed in each particular case. *Quinine* or *bark*, stimulants, *mineral acids, nutritious food*, and *wine*, was the treatment adopted by Cullen, Duncan, and Willan. Subsequently this plan of treatment was questioned by Dr. Parry, of Bath, who found that in his cases a full bleeding from the arm was a much more speedy mode of curing the disease. Dr. Harty, of Dublin, confirmed this principle of treatment, and was successful with liberal doses of purgatives, so as to clear out the intestinal canal completely. *Oil of turpentine*, administered in moderate and repeated doses, has also been recommended. Dr. Hardy, of Dublin, recommends the tincture of *larch bark*. He has long used it as a styptic and carminative tonic; and it is "one of the most elegant forms of prescribing a terebinthinate" (MOORE). *Fifteen drop doses* of the tincture may be administered every hour, or *eight* or *ten drops three times a day*, afterwards increasing the dose according to the age of the patient and the necessities of the case.

The treatment of purpura may be comprised in the following measures:—The bowels ought invariably, and without exception, to be first thoroughly and effectually evacuated by means of *senna, aloetics*, or *calomel* and *jalap*. If several effectual doses of either or all of these medicines be not followed by less heat of skin, diminution of the frequency of the pulse, abatement of the internal pains, and a cleaner state of the tongue; if the spots continue to increase in number and size, and the hæmorrhagic oozings do not cease,—blood, according to age, strength, and other circumstances, may be drawn from the arm, while the patient should abstain from animal food in every form, and should subsist on boiled rice with whey, or the light subacid fruits, as grapes, oranges, strawberries, gooseberries, baked apples, or the like. His drink may consist of tamarind-water, or water acidulated with sulphuric acid. Under this plan most cases of the disease will be speedily and readily brought to a favourable termination. If symptoms of local uneasiness continue after the urgent phenomena have disappeared, leeches should be applied in the neighbourhood of the part; and it will be proper to continue the periodical and regular evacuation of the alimentary canal. When the spots have disappeared and the hæmorrhage has ceased, the constitution recruits rapidly under the gradual but cautious use of light soups and fresh fruits and vegetables (Craigie).

SCURVY.

Latin Eq., *Scorbutus;* French Eq., *Scorbut;* German Eq., *Scorbut;* Italian Eq., *Scorbuto.*

Definition.—*A chronic morbid state, characterized by sponginess of the gums and the occurrence of livid patches under the skin, of considerable extent, which are usually harder to the touch than the surrounding tissue. It is ushered in by debility, lassitude, lowness of spirits, attended by fetor of the breath, and the gums swell by irritation, till they overhang the teeth in palmated excrescences. The livid subcutaneous patches and spots appear upon the skin, of considerable extent, especially on the lower extremities, among the roots of the hair. Spontaneous hæmorrhages may take place from the mucous canals; contractions of the muscles and tendons of the limbs occur, with pains, and sometimes superficial ulcerations. An altered state of the albumen of the blood is associated with this condition, and the phenomena are brought about by a deficient supply of the organic vegetable acids, or of the salts of fresh vegetables.*

Pathology and Historical Notice.—Scurvy is mentioned by Pliny as having occurred in the Roman army commanded by Germanicus after a long encampment in Germany beyond the Rhine. It prevailed to a frightful extent in the army of Louis IX., when he was made prisoner in Egypt in 1260. With fish only for fresh provisions, dispirited by being obliged to act on the defensive, and harassed unceasingly by the Saracens, the Christian army was almost annihilated by a most terrible outbreak of scurvy. But it was not till navigation was improved, and long voyages undertaken, that this disease became well known from its general prevalence and formidable character. Vasco de Gama, in his first voyage to the East Indies by the Cape of Good Hope, in 1497, lost 100 men out of 160 by this affection. James Cartier, in his second voyage to Newfoundland, in 1535, speaks of sufferings still more severe. Of 110 people there were not ten whole. "This malady being unknown to us," he writes, "the body of one of our men was opened, to see if by any possible means the occasion of it might be discovered and the rest of us preserved. But in such sort did the malady increase, that there were not above three sound men left. Twenty-five of our best men died; and all the rest were so ill that we thought they would never recover again." A decoction of the leaves and bark of a tree, supposed to be a species of spruce fir, was the remedy which they found restored health and recovery. "It was scurvy which used to decimate our navy, and render long sea voyages almost impossible. It was mainly by scurvy that Anson, in his celebrated voyage of 1740-2, lost within the first ten months nearly two-thirds of his crew, and during the remaining period about half of the survivors. Scurvy continued to prevail with little abatement till 1764. The voyages of Sir R. Hawkins, Hosier, Anson, Drake, Cavendish, Dampier, Byron, and of numerous other navigators, furnish similar details, and show how recklessly the lives of sailors were sacrificed (COPLAND). But at last it was against scurvy that Cook had attained his triumphant success, when, in 1775, after three years' absence, he brought back a healthy crew, which, out of 112 men, had lost only one by disease. During the sixteenth, seventeenth, and earlier part of the eighteenth century, the disease was endemic * in towns, fortifications, camps, and armies. "Many thousands were often cut off within a few months in single armies and garrisons; and it is probable that more seamen perished from scurvy alone than from all other causes combined, whether sickness, tempest, or battle" (WOOD). In this country and in America it is now very little known; and many have never seen the disease, unless they have lived in seaport towns.

Dr. Clymer states, in the American edition of this work, that scurvy has always prevailed in the United States Army to a considerable extent, and under the same circumstances—a deprivation of fresh vegetables. From 1840 to 1859, inclusive (excluding the years 1847-48—the period of the Mexican war—no reports having been made during that time), there were 4,935 cases of scurvy reported and 52 deaths, in an aggregate strength of 187,144 men (*Statistical Report, U.S. Army.* By authority. 1860.) In the column which marched on the city of Mexico, the men, for some time previous to their landing at Vera Cruz, during the siege, and afterwards, could obtain no vegetables; and the writer is informed, on reliable authority, that on their arrival at Jalapa, although there had been plenty of fresh beef, there was scarcely a man who did not have a scorbutic taint. During the late civil war, scurvy, in some form or another, was generally prevalent in the American armies. Though the statistics of the first two years show an extremely small number of cases, "unparalleled in the history of armies," it is not a true exhibit of the actual prevalence of the disorder. There were reported in the first year, 1,328 cases and 9 deaths, and in the second year, 7,395 cases and 90 deaths; to which may probably be added 304 cases of purpura and 31 deaths (*Circular, No. 6, Surgeon-General's Office, War Dept.*, 1865). It first appeared to any extent in the Army of the Potomac, at Harrison's Landing, Va., in July, 1862; and from that time forward, until the end of the war, in 1865, it continued an increasing and formidable disorder. "It occurred in all the armies subjected to hardships, especially in the West; and its worst and most fatal manifestation was made after the war had closed, and during the occupancy of the frontier of the Rio Grande by the twenty-fifth Army Corps," and in consequence of the want of fresh vegetable food (*U. S. Sanitary Commission Memoirs*).

"What has been written of scurvy," he continues, "in the British army, during the Crimean war, is equally applicable to our own during the late war. The returns convey but a faint conception of the disastrous part which it [scurvy] acted among the troops; for though it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was widespread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fevers and affections of the bowels" (*Medical and Surgical History of the British Army*, 1858).

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* It is a mistake to call scurvy *epidemic*, as has sometimes been done. The term cannot be applied with propriety to diseases other than those which we have seen to depend upon the presence of specific poisons which enter the blood by impalpable media, and common, therefore, in a greater or less extent, to all classes of the community.

"In the first years of the war scurvy did not prevail amongst the Confederate troops apparently to any extent; for with a monthly mean strength of 160,231 officers and men, and with 1,056,349 cases of sickness and wounds entered upon the field reports in nineteen months, from January, 1862, to July, 1863, only 2,203 cases of scurvy are recorded; and in 398,641 cases of sickness and wounds entered upon the the hospital reports, there were only 2,068 cases. But it progressively increased, with a diminishing commissariat and increased hardships (J. JONES, *in San. Com. Memoirs*). The same authority, in his report on the diseases, &c., of the Andersonville prison, states that scurvy, arising from sameness of food and imperfect nutrition, caused, either directly or indirectly, nine-tenths of the deaths amongst the United States army prisoners confined there."

Morbid Anatomy.—The days when scurvy was most prevalent were not those in which many *post-mortem* examinations were made; and our earliest knowledge of the morbid anatomy of this disease is derived principally from Poupart and Lind. They tell us that in those cases in which flux or dysentery is absent the intestines have been found perfectly sound, however copious the hæmorrhage from them may have been. The principal effects of the disease were observed in all cases in the cellular tissue of the extremities. The quantity of congealed blood effused in that part, even where no stain or mark could be perceived on the skin, was quite astonishing. "It often lies," says Lind, "in large concrete masses on the periosteum, while the bellies of the muscles of the legs and thighs seemed quite stuffed with it, often an inch in thickness." Haspel and Marmy confirm these accounts of Lind when they describe laminar masses of black grumous effusion in the subcutaneous and inter-muscular connective tissue, compressing and breaking down the primitive fasiculi. He often found water effused into the cavities of the chest and abdomen, and no less frequently blood,—the quantity of blood effused in all parts sometimes amounting, in his opinion, to no less than a fourth part of that contained in the whole body. Poupart gives some further particulars, and says that on moving the limbs of some scorbutic patients a noise is heard; and that on examining the joints the epiphyses had entirely separated from the bones; and in other cases, that the cartilages of the sternum had separated from their bones; and bones that had united after being broken very often separated again at the site of fracture. He says that if we squeezed the ribs which had begun to be thus separated from their cartilages, "there came out abundance of corrupted matter, so that nothing was left of the rib but its bony plates." The mesenteric glands were usually enlarged; the spleen, often three times bigger than natural, fell to pieces, as if composed of coagulated blood. In two cases examined at St. Thomas's

Hospital by the late Dr. Robert Williams, patches of ecchymoses were found under the pericardium covering the heart, and also under the arachnoid membranes covering the brain.

In some endemics of scurvy there is a marked tendency to the effusion of dark liquid blood into the serous cavities, and of the synovial membranes, the most common site being into the pericardium, then the pleura, and next the peritoneum (KARAWAGEN). The spleen is often enlarged, distended with blood, and soft. The texture of the muscular system is also soft and flabby. The blood appears to be deficient in red particles (BUSK, BECQUEREL, RODIER, ANDRAL, FRICKS), and does not impart a stain to the lining membrane of the heart and great vessels. A fluid or dissolved condition of the blood has also been noticed, appearing often as "a mere gore, not separating into crassamentum and serum, and putrefying soon. It appears to be starved of some essential ingredient." It flows with difficulty from the vein, and, after standing some hours, deposits a thick, muddy sediment, which subsides from a reddish serum; and in the last stage of the disease it becomes quite black (ROUPPE). There does not, however, appear to be any evidence of deficient powers of coagulation in scorbutic blood; and so long ago as 1699, Poupart noted the large coagula found after death in the cavities of the heart. Three analyses of the blood were also made by Mr. Busk, before 1840, in all of which the quantity of fibrine was found to be above its normal standard. Stoeber, in 1845, came to the same conclusion; and so did Andral, on repeating analyses which at first led him to an opposite conclusion. Two facts of great importance, if confirmed, have been observed by Chatin and Bouvier. They have observed that the *albumen* of the blood does not coagulate under a temperature of 74° Cent., or 165·2° Fahr.—that is, from 5° to 8° Fahr. above the normal standard; so that the albumen of the blood undergoes some change in scurvy which increases its solubility. They also observed that the force of cohesion of the *fibrine* was so much lessened that they were unable to isolate it thoroughly from the red corpuscles, and it was this attraction of the fibrine and the red corpuscles which the early writers on scurvy attempted to describe by the terms "*agglutinated blood*," "*viscid and thickened crassamentum*" (Parkes "On the Pathology and Treatment of Scurvy," *Brit. and For. Med.-Chir. Review*, Oct., 1848). Thus all the phenomena of scurvy, and the conditions under which it becomes developed, point to the blood as essentially altered. What the alteration definitely consists in has not been yet determined; but it appears that the condition of the blood in scorbutus does not consist in the want of a due proportion of either of its three prominent constituents. The proportion may in some cases be deranged; but it appears that in all cases there is a deficiency or altered quality of some essential ingredient. The following statements by Drs. Copland, Watson, and Wood contain a summary of the opinions of the chemical pathologists of the present day who have specially investigated the nature of scurvy by analysis of the blood:—

Dr. Christison supposes that scurvy arises from the want of vegetable albumen or animal casein in the food; that it is the deficiency in the quantity of azotized aliment, and consequent insufficient nourishment of the body, which is the cause of scurvy. Dr. Garrod believes that the malady is caused by the absence of *potash,* and that potatoes and other antiscorbutics owe their virtues to the *potash* they contain. The following are his conclusions:—(1.) That in all scorbutic diets *potash* exists in much smaller quantities than in those which are capable of maintaining health; (2.) That all substances proved to act as antiscorbutics contain a large amount of *potash;* (3.) That in scurvy the blood is deficient in *potash,* and the amount of that substance thrown out by the kidneys is less than that which occurs in health; (4.) That scorbutic patients will recover when *potash* is added to their food, the other constituents remaining as before, both in quantity and quality, and without the use of succulent vegetables or milk; (5.) That the theory which ascribes the cause of scurvy to a deficiency of *potash* in the food is capable of rationally explaining many symptoms of that disease.

These propositions of Dr. Garrod's require much further investigation; and they are not supported by the most elementary and important facts in the history of scurvy. Both soda and potash are constant constituents of the animal body, and it appears that they are not capable of replacing each other. For example, we always find the potash to exist in large quantities in the ash of muscle, soda in very small quantities (Berzelius, Liebig). In the ash of the blood we find the relation reversed. It appears, also, that the muscular system requires the presence of potash; and we should therefore expect to find that where there is a deficient supply of this base, the effect would soon be manifested in the functions of that system. This we do not find to be the case in scurvy. Without any amount of wasting of the body, we may find marked muscular debility; but men in an advanced stage of *scorbutus* have been known to do severe labour, till even the approach of fatal syncope from the weakened action of the heart; and this, perhaps, is one of the earliest symptoms of the disease. "Opposed also to this theory," writes Dr. Wood, "are the facts that nitrate of potash has often failed to cure the disease, while it has been cured by the addition of pure citric acid; and that, after failure under treatment with the salts of potash, recovery immediately commenced when the patient was allowed to eat fresh vegetables." Trotter also always held the opinion that the real antiscorbutic principles in fruit and vegetables were the vegetable acids, particularly citric, oxalic, and malic acids; and to test the accuracy of his opinion, he made, in 1800, a series of comparative trials between *lemon juice* and pure *citric acid.* Both these remedies were furnished to eight or ten ships, and reports were then obtained of their effects. The result was that *citric acid* was found the most efficacious. Sir William Burnett, also, was in the habit of supplying convict ships with *citric acid* and *nitrate of potash,* as well as with *lemon juice,* in order that comparative trials might be made of the relative value of these remedies; and the official documents bearing on this point were carefully examined by Dr. Parkes, who considered that nothing could be more convincing than the evidence they contained, showing that the efficacy of *citric acid* was clearly proved, while *nitrate of potash* was shown to be inferior in curative power, (*On the Pathology and Treatment of Scurvy,* l. c.) Dr. Clymer mentions that the Ottawa lumberers living on pork salted with nitre suffer severely. Dr. J. O. Grant found in one *shanty*, out of thirty-six of those men, that twenty-five were affected with scurvy. The astonishing effects of *fresh* lemon juice have been evidently underrated by Dr. Garrod; and it is more probable that its virtues are due to citric and other acids than to potash. Dr. Aldridge contends for the influence which should be ascribed to a deficiency of phosphorus, sulphur, lime, and the alkalies, in occasioning scurvy. That something may be owing—a part merely—to the causes contended for by Dr. Aldridge is not improbable. But it is unnecessary to pursue this subject any further than very briefly to state, that one of the most evident changes from the healthy condition is seated in the blood, which is altered in composition either by the addition to it of some ingredient or ingredients, or by the absence of something which ought to exist in it; and the deficient ingredient may be one of the ordinary constituents of the blood, or it may be some principle or element entering into their composition. This deficiency is due to the absence of certain articles of diet; and the disease is known by experience to be at once cured by supplying those articles.

Symptoms.—The earliest are a change of colour of the skin, particularly of the face and eyelids. This is sometimes the first and only sign of the disease. The skin round the orbit may be puffed up into a bruised-coloured swelling. At the same time there are vague, wandering, rheumatic-like pains in the limbs, weariness, depression of spirits, and a longing for fresh vegetables and fruits. With these pains, however, there is no fever; the pulse is soft and natural, and the temperature of the body lower than usual, and great disorder of the respiratory function may supervene. The countenance appears pale or yellow, and bloated; there is great depression of the physical powers, followed by swelling of the gums, which become soft, spongy, and hang over the teeth in large fleshy-like palmated masses, very much disposed and readily excited to bleed; and the edges of the gums are purple where they are in contact with the teeth. Several old observers state, and Fauvel confirms the observation, that in old people without teeth these vegetations do not occur, and the gums remain comparatively unaffected. In one case he noticed that a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum immediately became level and firmer, while the other symptoms preserved their intensity. The swollen and fungus gums, as Dr. Parkes observes, have always appeared to be much more intense in cases occurring on shipboard; and it is not improbable that they are affected peculiarly because they are exposed to pressure and attrition; sometimes they are severely ulcerated.

All parts suffer from pressure in cases of scurvy—the merest rub causes an ecchymosis, the slightest possible blow produces an extensive bruise, and the gums may suffer most from the causes already noticed. A small eruption (like flea-bites) of a purple hue is next seen on the lower extremities; and about the same time the muscles of the leg or thigh become hard and painful, and in a day or two the skin over the pained part becomes first yellow and then purple. This discoloration forms patches sometimes as big as the palm of the hand, and may extend over half the leg and thigh. The popliteal regions are a frequent site of this pain and discoloration, sometimes attended with œdema, especially of the ankles and feet. The discoloration is especially apt to show itself in the situation of old cicatrices and injuries. "These extravasations are both *superficial and deep-seated.* The *superficial* are,—(1.) *Dermic,* and (2.) *Subdermic.* The *dermic ecchymomata* vary in size from small, dark-coloured spots to large blotches; they may be distinct, or may come together at some point of their circumference. The anatomical site of the spots is the hair follicles or sudoriferous canals, and their structure hinders the diffusion of the sanguine serosity. The epidermis becomes dry, rough, and raised in scales, resembling the skin of a recently-plucked fowl (H. LARREY), and the sudatory function is diminished or suspended. Subsequently desquamation takes place. The solid part of the blood may, however, remain infiltrated in the meshes of the derm, forming brown spots, which may last for months, the epidermis over these being smooth and shining."

"In the *subdermic ecchymomata* the blotches are larger and of variable depth. They may invade the whole limb, but the most common site is the ham, groin above and below Poupart's ligament, the thigh, calf, and pterygo-maxillary region. If they compress superficial veins, they cause œdema and pain. They produce swellings in the flexures of the joints, most often in the ham or shin, in the pterygo-maxillary region, and bend of the elbow. They are hard, but pit on persistent and firm pressure. When on the shin and circumscribed, they may be mistaken for syphilitic nodes. Resolution is their usual termination, their hue changing from dark purple to green and yellow. When the dark colour persists with a greenish-yellow border, it is evidence that the effusion is very thick. Should their termination be by ulceration, a sort of indolent boil forms, with a deep-red base and black summit, with œdema of the adjacent connective tissue. If the surface is galled, there is constant weeping of a sero-sanguinolent fluid; the tumor flattens a little; and a small, dark, ulcerated central point, with wine-red edges, appears: this is a gangrenous mass, which gains little by little, until an ulcer of variable size is established. The most frequent site of these scorbutic ulcers are the calves of the legs, the buttocks, thighs, sacrum, shoulders, arms, and more rarely the chest and abdomen. The *deep-seated extravasations* are,—(1.) *Sub-aponeurotic;* (2.) *Parenchymal,* into the muscles, kidneys, heart, lungs, &c. These extravasations into the muscle-tissue never end in suppuration, but the tissue becomes atrophied, and undergoes a sort of gelatinous degeneration; resolution is very sluggish. When the effusion is beneath a resisting aponeurosis, as the *fascia lata,* there is no alteration in the integument immediately over it, but the swelling and discoloration happen at remote points, as the ham, about the knee, &c. The source of these extravasations would seem to be the softened capillary vessels, permitting the leakage of altered blood. There is no apparent solution of continuity except an ulcer exists" (MARMY, quoted by Dr. Clymer).

The tongue is white, the breath fetid, and the stools generally pale. As the disease advances, all these symptoms are aggravated. The loss of physical power increases, the purple spots have a tendency to ulcerate, and the ulcers are distinguished from all others by their putrid fungoid appearance and great tendency to bleed; old sores open, and the callus of broken bones has been even dissolved and their ends separated. Profuse hæmorrhages frequently take place from the mouth, nose, lungs, or bowels, sometimes from *bullæ,* which form and burst (discharging blood) on the mucous surface. The teeth become loose, so that they either fall out or may be taken out by the finger and thumb. *Ptyalism* is not uncommon in scurvy, with swelling of the parotid and sub-maxillary glands. The pulse hurries on to 120 or 140; and at length the patient sinks from diarrhœa or dropsy, and with effusion so sudden that he perhaps has walked a short distance and then died in a quarter of an hour afterwards.

The duration of the disease is generally many weeks, and sometimes under the most favourable circumstances many months, the patient recovering his strength very slowly. When the disease follows ague, obscure or irregular remissions or intermissions of febrile symptoms are observable, and more or less enlargement, with pains in the region of the spleen, is often detected. From the commencement of the disease the alvine evacuations are more or less disordered, but the change may not attract attention. At first costiveness prevails, but subsequently the stools become much more frequent and very offensive. Diarrhœa, with colicky pains, supervenes, and, with more or less attendant hæmorrhage, rapidly sinks the patient beyond hope of remedy. Dysentery frequently supervenes upon the scorbutic state, more especially when dysentery is epidemic; and this combination of maladies is sometimes termed "*scorbutic dysentery.*" The tongue, pale at first, becomes broad, flabby, and indented at the edges of the teeth. The great poverty of the blood causes the mucous membrane of the tongue and inside of the lips and cheek, as well as of the skin, to have a peculiar paleness, which contrasts remarkably with the appearance of the gums. The eyesight is frequently weakened, and night blindness (hæmeralopia or nyctalopia) has frequently been observed as a commencing and concomitant symptom; and a most distressing state is the inability to sleep at night.

"The tendency to *swoon* in the most severe cases is sometimes so great that the slightest motion, or the erect posture, or even any trifling exertion, may be followed by fatal syncope. It is stated in the account of Lord Anson's voyage, that many of the men, although confined to their hammocks, ate and drank heartily, were cheerful, and talked with much seeming vigour and in a strong tone of voice; and yet, on their being the least moved, although it was only from one part of the ship to the other, and that in their hammocks, they have immediately expired; and others, who have confided in their seeming strength, and have resolved to get out of their hammocks, have died before they could reach the deck. And it was no uncommon thing for those who could do some kind of duty, and walk the deck, to drop down dead in an instant, on any endeavour to act with their utmost vigour" (COPLAND).

There is an intercurrent chest affection which is apt to occur during cold and damp weather in scorbutic patients.

"It begins," writes Dr. Clymer, "with slight rigors, followed by feverishness, and accompanied by lancinating pains in one or both sides. There is dyspnœa, and a feeling of constriction in the chest, as if a cord were drawn tightly round it. This condition is commonly caused by intrapleural effusion of blood, but sometimes blood escapes into the pulmonary tissue; in the latter case, the expectoration becomes dark and sanious, and has a fetor resembling that of pulmonary gangrene, but which is due to decomposition of the sanguine fluid. Cold sweats, increasing dyspnœa, anxiety, and a frequent, thready pulse, precede death, the constant termination. Sometimes there is neither pain nor cough, but rapid increase of the breathlessness, and sudden death. The physical signs of the lung lesion are often wanting, though sometimes there may be dulness, mucous rhonchi, and bronchial respiration (HASPEL). Chest dulness on percussion in scurvy may sometimes be due to sanguineous effusions into the thoracic muscles" (HASPEL, BUZZARD).

With our knowledge now of the phenomena of embolism, may not the suddenly fatal end of such cases be due to the morbid condition of the fibrine of the blood already referred to?

Diagnosis.—The scorbutic state of the skin is to be distinguished from *flea-bites, bruise, typhus fever,* and from *purpura hæmorrhagica.*

In purpura the blotches occur suddenly in persons previously to all appearance in good health; while the dingy hue of the skin is wanting, also articular effusions, and pains in the limbs.

Prognosis.—In the present day, when the patient can command medical care and proper diet, scorbutus, though tedious, is seldom fatal. When these, however, have been wanting, the mortality has been terrible. Lord Anson, it should be remembered, in his voyage round the world, lost above 200 men, and at last could not muster more than six fore-mast men in a watch fit for duty. At the commencement of our last war with France, on the fleet returning from sea, it often happened so many men were landed ill of scurvy that even Haslar Hospital, large as it is, could not contain them, and many were lodged in the chapel, others in tents, while others died in the boats before reaching the shore.

Cause and Conditions under which Scorbutus is Developed.—In the Middle Ages scurvy prevailed to so great an extent that it was said to be epidemic among the inhabitants of the low countries of Holland, Friesland, Brabant, Pomerania, Lower Saxony, and, indeed, all countries from the 50° to the 60° of north latitude. This has been attributed to the absolute want of winter food for the cattle, so that it was necessary to kill them on the setting in of the frost, and either to salt or dry the flesh. Food was deficient and of improper quality; and hence the large stores of salt provisions found in the larder of the elder Spencer in the days of Edward II., even so late in the spring as the third of May. Six hundred bacons, eighty carcases of beef, and six hundred of sheep, was his abundant supply. In all these countries, however, in proportion as agriculture has advanced, and a succession of green crops has enabled the farmer to kill his best and fattest meats in winter, and in proportion as fresh vegetables have been introduced at our tables, together with a liberal use of wine and beer, so has this disease disappeared. The former universal prevalence of scurvy in the Navy, and its almost entire disappearance in the present day, necessarily has reference to a particular cause—the too exclusive use of salt provisions and absence of fresh vegetables. "In 1797 the victualling of the Navy was changed, greatly improved, and strictly regulated; and immediately consequent to the change the health of the seamen improved strikingly. Scurvy, typhoid fever, dysentery, and putrid ulcer, which up to the period of the change produced great havoc, became comparatively rare in occurrence and light in impression." Since 1797 the improvements have consisted in giving cocoa instead of gruel for breakfast, issuing salt meats at a much earlier period after being cured, the supply of better articles, and in greater abundance by one-third, the substitution of tea in the afternoon instead of spirits; but, chief of all, the use of fresh vegetables as often as possible; and, with every improvement in these respects, there has been, as a general result, a further improvement in health, till these four forms of disease, at no distant date so destructive, are scarcely known except by name.

It is now, however, completely established that salted meats are not more productive of scurvy than fresh meats, so far as concerns a monotony of diet. The experience of the Russians in 1720 and 1736, of the French in 1750–60, and of our own regiments at the Cape in 1836, sufficiently establishes this point. Nevertheless, salt meat has generally formed a large and important part of the food taken by the sufferers in all great outbreaks of scurvy.

Besides the injurious effects of cold and moisture, as well as impure air, combined with the conditions already noticed as tending to favour the development of scurvy, it is now well known that exposure for a lengthened period to the pernicious influences of a malarious district greatly aids in developing scorbutus. By observations especially devoted to this subject, I have determined that, amongst our troops who had been in Bulgaria during the war with Russia in 1854, there were two and a half *per cent.* of admissions for scurvy amongst them more than among those who served in the Crimea only; and that the deaths among them reported from this disease were also greater by three *per cent.* than among those troops who served in the Crimea, (*Glasgow Med. Journal*, July, 1857; and *Trans. of the Royal Med.-Chir. Society*, vol. xl.)

Recent combined researches have shown that scorbutic diseases were developed amongst our troops in the Crimea under the following conditions:—(1.) Deficiency of absolute nutriment; (2.) Improper adjudication of the *nutrient* and *respiratory* principles of the *diet*—its monotony; (3.) Bad quality of the diet, and improper cooking, or none at all; (4.) Exposure to cold, combined with imperfect clothing, and labour beyond the strength of the best-fed men; (5.) The persistent pernicious influence of residence in a paludal district (Bulgaria). But these circumstances are only to be regarded as the *occasional antecedents*, any one of which, or all combined, can never, *per se*, originate the disease. LOOKING TO THE HISTORY OF SCURVY, IT WILL BE SEEN THAT THE INVARIABLE AND INDISPENSABLE ANTECEDENT OF THAT DISEASE HAS BEEN A DEFICIENCY OR ABSOLUTE WANT OF FRESH VEGETABLE FOOD. Privation of vegetable food is its one essential cause. There is no other invariable antecedent; and there are sufficient reasons why it may not always be followed by scurvy. It is this antecedent which is the *vera causa* of *scorbutus;* and the most successful methods of prevention and of cure are in accordance with the hypothesis which assumes it to be the cause (BUDD, CURRAN, LAYCOCK, PARKES). "The giving of vegetable food is its one essential counteractive" (SIMON).

On these questions Dr. Clymer gives the following interesting summary, in the American edition of this book:—

"In 1846, the potato crop failed in Great Britain and Ireland; and in the following year there was much scurvy amongst all classes. There was an outbreak of the disorder in Scotland, especially amongst the artisans and the labourers on the railways, in 1846–47, described by Drs. Christison, Ritchie, and Lonsdale (*Edin. Mon. Jour.*, 1847), and the general fact with regard to the food of all was, that it failed in variety and in the quantity of its animal constituents. None had tasted potatoes after the harvest of 1846—a period of seven or more months—nor fresh vegetables; but animal food, fresh and salted, was taken in large quantities, as well as pea-soup, suet puddings, bread, and oatmeal. At Workington, a seaport town of 7,000 inhabitants, there was no case of scurvy, turnips having been used in large quantities. Dr. Curran (*Dublin Quar. Jour.*, 1847), describing the disorder as it appeared in Ireland, says:—'In no single instance could I discover that green vegetables or potatoes had formed any part of the regular dietary;' grains, tea or coffee, flesh and fish, being the food. Dr. Shapter observes that in Exeter 'the only difference in the usual diet of the sufferers consisted in the absence of the potato,' and that many of them had abundance of the necessaries of life, except fresh vegetables, (*Med. Gazette,* vol. iv.)

"In the Crimean war, the allied armies suffered severely from scurvy. In the British army it first appeared in Bulgaria, where the diet was poor and the supply of vegetables scanty. When it arrived in the Crimea there was an abundance of grapes, cabbages, &c.; and though the ration was inferior, the disorder disappeared; but as winter set in, and vegetable food could no longer be got, it began again. When the supply of fresh vegetables and lime-juice became more constant, it gradually disappeared, and there were but few cases during the second winter.

"The French army suffered still more, no less than 23,000 cases of scurvy being recorded (SCRIVE). Good but lean fresh meat was issued, at first twice, then five times a-week, with rice, and occasionally dried vegetables—chiefly pease, beans, and lentils—in small proportions, and bread irregularly. In the spring, with vegetation, the number of cases decreased; but in July, with a parching sun, the disease reappeared, and in the course of three months, the finest and warmest in the year in that region, no less than 5,000 cases of scurvy occurred (BUZZARD). The Sardinian army, on its arrival in the early part of the summer of 1855, was largely affected with scurvy, which was checked by the issue of fresh vegetables. Dr. Buzzard, who was a staff-surgeon in the army of Omar Pasha, says, that 'loss amongst the Turks from this disorder was still greater, and that the original force which formed part of the expedition from Bulgaria to the Crimea was almost entirely swept off by disease, of which scurvy formed an important element' (Reynold's *System of Medicine,* Art. 'Scurvy,' vol. i., 1866). During the winter of 1854–55, of Omar Pasha's troops, as many as 1,000 were sent away monthly, all suffering severely from this disorder. During the summer of 1855, this army was encamped near Balaklava; and Dr. Buzzard remarks that, though 'their food was very imperfect, they were supplied with onions, and consumed large quantities of water-melons,' and no cases of scurvy appeared, nor could he detect any scorbutic taint in patients suffering from other diseases. But during the winter all vegetable supplies ceased, and 'shiploads of sick were brought to Trebizond, all of whom were severely afflicted with this disease'—in some cases 'developed to an extent which recalled the terrible descriptions of the disorder contained in the narratives of our early voyagers.' This was explained by the fact that, besides the absence during the winter of fresh vegetables, they had not had a sufficient quantity of food, their diet consisting 'entirely of biscuit, a little rice, haricot beans, and "yagh" (a coarse butter made from mutton fat), and there was 'absolute starvation.' In 1836, over 100 cases of scurvy occurred in the 75th regiment while quartered at Caffreland, when not an ounce of salt provisions was issued, when the men had no harassing duties, and were abundantly supplied with fresh meat. The Hottentot troops doing duty with them were served with the same rations, but sought out for themselves pumpkins, melons, wild fruits, and esculents, and entirely escaped; as did also the 27th and 72d regiments, encamped eighteen miles distant, fed with the same rations, but supplied with vegetables in addition. Dr. Buzzard states that scurvy is common in North Wales, where fresh meat and milk are abundant, but where there is little or no garden produce, and that he has met with many cases amongst the poor of London, who have eaten no meat of any kind for weeks, but had lived on tea, bread, and butter. At the close of the Punjaub campaign (1848–49), the troops had abundance of excellent fresh meat and bread, but no fresh vegetables, yet suffered severely from scurvy; and in the Himalayan stations, during the second Burmese war, under the same circumstances, scurvy was prevalent amongst the troops (*Med. and Surg. Hist. of the British Army*, vol. ii., 1858).

"Scurvy prevailed to a great extent in the United States army during the civil war. This was not owing to any deficiency in the ration, or to the want of liberal provisions for fresh vegetables by the government and private organizations, or the neglect of the medical department in issuing antiscorbutics. All these were most generously provided. But there were times when they could not be obtained, or used; when the men were obliged to live on marching rations; or when, through the ignorance or negligence of commanding officers, they were not distributed. Scurvy, under these conditions, was the invariable sequence. At one time, in the Army of the Potomac, when 'symptoms of scurvy began to appear,' and there was a general 'low vitality of the men,' Dr. Letterman found that he had rightly attributed it to 'want of fresh vegetables;' for, 'while large supplies of potatoes had been issued, the troops received in some cases a very small quantity, and in others none at all' (*loc. cit.*, p. 106). Again he says (pp. 109–10),—'This favourable state of the health of the army, and the decrease in the severity of the cases of disease, are in a great measure to be attributed to the improvement in the diet of the men by the issue of fresh bread and fresh vegetables, which has caused the disappearance of the symptoms of scurvy that in January began to assume a serious aspect.' The testimony of Dr. F. Hamilton, with respect to the Army of the West, is to the same effect (*loc. cit.*)"

Treatment.—The early history of navigation, as it records the greatest ravages of scurvy, so does it also record the best antidote to the disease. Of four ships which sailed from England in the beginning of April, 1609, for the establishment of the East India Company, they were all so severely visited by scurvy as to have lost nearly one-fourth of their crews when they arrived at the Cape of Good Hope. The crew of the Commodore's ship was not attacked. This immunity arose from three table-spoonfuls of lemon juice having been served daily to each of his men. But notwithstanding this evidence of the success of lemon juice in preventing scurvy—evidence the most conclusive—this valuable remedy and preventive was altogether slighted for a hundred and fifty years afterwards (Copland). Lord Anson's people, in 1740, on reaching the Island of Tinian, were recovered principally by eating oranges; and that noble, brave, and experienced commander was so convinced of their usefulness that before he left the island he ordered one man from each mess to lay in a stock for future security. Sir Charles Wager's people, also, were terribly afflicted with scurvy in the Baltic. Sailing, however, in the Mediterranean, and having heard how effectual oranges and lemons were in the cure of this disease, he took on board at Leghorn a large quantity of them, ordered a chest each day to be brought on deck, and allowed the men, besides eating what they chose, to mix the juice with their beer, and to pelt each other with the rind, so that the deck was strewed with the fragrant liquor. By these means he brought his men home in good health.

In the year 1747 Dr. Lind made some comparative trials between this and some other modes of treatment (as vinegar, sulphuric acid, and tamarinds) on board the "Salisbury," at sea. As a general conclusion from his experiments, he affirms that orange and lemon juice, or more properly the citric acid obtained from all the species of the botanical genus *citrus*, or the natural order of fruits called *hesperidæ*, are greatly more efficient than any other remedy in the cure of scurvy.

Notwithstanding this strong opinion of Dr. Lind, the Navy continued to suffer severely from scurvy for half a century, till the Admiralty gave a general order for the supply of lemon juice. This salutary measure was accomplished by a representation from the Medical Board of the Navy, in the year 1795, when Lord Spencer was First Lord of the Admiralty, after a trial made on board the "Suffolk," of seventy-four guns. This ship sailed from England on the 2d of April, 1794, supplied with a quantity of lemon juice sufficient to serve out two-thirds of a liquid ounce daily to every man on board, and this was mixed with their grog, with two ounces of sugar. She arrived at the Madras roads on the 11th September, after a passage of twenty-three weeks and one day, without having had any communication with the land, without losing a man, and having only fifteen on the sick list. Scurvy appeared in a few of the men during the voyage, but disappeared on an increased dose of lemon juice being administered. "Let this fact," says Sir Gilbert Blane, "be contrasted with the state of the channel fleet in 1780, when Admiral Geary's fleet returned into port, after a ten weeks' cruise in the Bay of Biscay, with 2,400 men ill of scurvy; and let the state of this fleet be contrasted with that of the channel fleet in 1800, which, by being duly supplied with lemon juice, kept the sea four months without fresh provisions, and without being affected with scurvy."

In 1780 the number of cases of scurvy received into Haslar Hospital was 1,457; in 1806 *one* only; and in 1807 also *one*. While it is notorious that many medical men have never seen the disease, it is, as Dr. Budd has assured Dr. Watson, by no means rare in the hospital ship at Greenwich, which is often full of cases of scurvy; most of the patients so affected having just arrived in *merchant ships* from a long voyage; and some rumours are now abroad to the effect that scurvy is more frequent in the merchant service than heretofore—and, if so, the least that can be said is, that it is a disgrace to the merchant princes of a nation like Great Britain.

With regard to the prevalence of scurvy among sailors in the merchant service, Dr. Barnes (Physician to the "Dreadnought" hospital ship) reports in 1864 to Mr. Simon that "of the entire number of cases admitted during the last twelve years into the Sailors' Home at Poplar, nearly half are, at the time of their admission, suffering more or less from scurvy, and of these perhaps a twentieth part seriously diseased with it; that of the entire number of cases admitted during the last twelve years into the hospital ship 'Dreadnought,' cases of scurvy have formed a twenty-fourth part; that to these must, of course, be added sufferers who are not taken into such establishments—a large but uncertain number of cases taken into the low lodging-houses of the water-side; but that this is for London alone, and is not nearly all due to London shipowners; that of eighty-six cases of scurvy treated in the 'Dreadnought' during 1863, only fourteen came from ships which had issued from the port of London, while thirty-one came from foreign ships, twenty-one from Liverpool ships, eight from Sunderland ships, and twelve from Glasgow and other British ships; that Liverpool ships, besides furnishing to London a large proportion of the scurvy which is treated here, convey probably a much larger quantity of the disease to their own port of departure; that at all events, during 1863, fifty cases (all probably severe) were admitted at the Liverpool hospitals, and during 1862 a dozen cases at the Glasgow and Greenock hospitals; that shipowners of Liverpool and other northern ports, and of Hamburg and America, are those who exhibit the greatest amount of disregard of the safety and health of their crews. The plight in which the poor sailors from certain services are admitted is pitiable to witness. Disabled by hardship, semi-starvation, and ill-usage of every kind, they are cast out with the same indifference with which a worn-out block would be thrown overboard." When it is remembered that the security of this country has on several occasions been imperilled by the disablement of the Royal Navy through scurvy, it may be presumed that the same cause will imperil the safety of our merchant ships. And there can be no doubt that many ships have actually foundered at sea because the crews were so prostrate from scurvy as to be unable to handle them when overtaken by severe weather. It has been the custom to inquire what proportion of the crews were disabled from scurvy. As might be supposed in the case of a disease resulting from a cause operating upon the entire crew, this proportion is often very large. Thus there were recently admitted on board the "Dreadnought" twelve cases of severe scurvy from one ship; two others were known to be seriously affected; and the entire complement of officers and men was only nineteen, leaving but five men in all able for duty to work the ship. The proportion of crew disabled has ranged from 20 *per cent.* to 70 *per cent.;* and it is certain that scurvy ships have rarely a hand to spare. Deprive such ships of a fourth, a half, or two-thirds of their force, and the peril of a ship, cargo, crew, and passengers, in stress of weather, is obvious; and it must not be forgotten that where scurvy has prostrated a large part of the crew, the vigour of the remainder is sure to be sapped, so that there may not remain a single sailor before the mast in a state of thorough efficiency (Simon, in *Sixth Report on Public Health,* p. 19).

The disease, "so fatal when left to itself, is cured with the greatest facility. Symptoms apparently the most grave and serious vanish as if by magic, and without leaving behind them any serious injury to the constitution. The sanious discharge from scorbutic sores has been known to change colour and to become healthy in a few hours after the commencement of treatment. In pure cases of scurvy the blood, and the blood only, is at fault," (PARKES, l. c.) "Lemon juice," writes Dr. Watson, "is really a specific against scurvy, whether it be employed as a preventive or as a remedy. It supplies something to the blood which is essential to its healthy properties." The potato seems to be no less efficacious as a remedy and preventive (BUDD). The reader will find a most interesting account of the efficacy of potatoes and of onions in Dana's *Two Years before the Mast*—a book well worth reading. The antiscorbutic principle, whatever it may be, is in greatest amount in unripe fruits; it lessens gradually as they ripen; and if the juice be obtained, it disappears when fermentation occurs. When lime juice becomes musty, a mucilaginous principle is developed at the expense of the citric and malic acids; and the *per centage* of citric acid gradually decreases (SIR WILLIAM BURNETT, PARKES). Good *lemon* juice seems to be more effectual, however, than pure *citric* acid; probably from its containing *malic* and *tartaric acids,* besides *citric,* and from the *citric acid* being in the form most easily absorbed and decomposed by the digestive organs of man. The Materia Medica gives numerous analogous examples of the superior efficacy of a medicine in its *natural combinations* (PARKES). This is all we can yet say, however, regarding the actions of either of these means of cure, notwithstanding the researches of the chemists of the present day. Moreover, it is sufficient: and with such remedies at command, the prevalency of scurvy in *merchant vessels,* or in any Navy, ought not to exist. "The one thing wanted in order that scurvy should be entirely banished from the *mercantile marine* is proper provision for the dietary of the crew,—such provision as is enforced in *emigrant ships,* where each person's weekly allowance must have in it at least 8 oz. of preserved potatoes, and 3 oz. of other preserved vegetables (carrots, onions, turnips, celery, and mint), besides pickles and 3 oz. of lime juice; where, also, there is considerable variety of bread stuff; and where, on two days in the week, preserved (not salted) meats must be given. With such a dietary as this, the details of which might be varied, provided its principle were adhered to, the occurrence of scurvy would be impossible. And even with dietaries inferior to that just described, scurvy would not occur as it does, if but the provisions of the 'Merchant Shipping Act' (17 and 18 Vict., c. 104) were obeyed,—that whenever a crew shall have been consuming salt provisions for ten days, lime juice or lemon juice and sugar shall be served out at the rate of half an ounce each per day; and if, during the voyage, the opportunities which offer themselves were fairly used for getting new supplies of fresh animal and vegetable food; but owners disobey the law, and captains neglect opportunities to counteract the results of this disobedience. Owners, notwithstanding the law, will send forth their ships on long voyages without any provision of lime juice, or with lime juice insufficient in quantity, or with lime juice of which the quality is bad; and captains, with half their crews more or less disabled, are known to run past St. Helena or the Western Isles, when a few hours' delay would obtain sufficient provisions to repair the mischief occasioned by first neglect" (Simon and Barnes's *Sixth Report on Public Health*, 1864, p. 20).

It is perhaps hardly fair to attribute the improved health of the Navy entirely to the introduction of a daily allowance of lemon juice, considering that the quantity of the diet was greatly increased, and its quality greatly improved, contemporaneously with this addition. It is gratifying, however, to see how largely these combined measures have improved the health of the Navy, and rewarded the cares of those who superintend it; for during the nine years preceding these changes the sick seamen sent to the hospitals were 1 in 3·9, while in the nine succeeding years the proportion was only 1 in 8·4; so that not only has scurvy almost disappeared from ships of war and naval hospitals, but the efficiency of the Navy has actually been increased threefold.

Dr. Parkes advises that the following measures be adopted in time of war, or in prolonged sojourn on board ship, or at stations where fresh vegetables are scarce:—

"1. The supply of fresh vegetables by all means in our power. Even unripe fruits are better than none, and we must risk a little diarrhœa for the sake of their antiscorbutic properties. In time of war *every* vegetable should be used which it is safe to use, and when made into soups all are tolerably pleasant to eat.

"2. The supply of dried vegetables, especially potatoes, cabbages, and cauliflowers; turnips, parsnips, &c., are perhaps less useful; dried pease and beans are useless. As a matter of precaution these dried vegetables should be issued early in the campaign, but should never supersede the fresh vegetables.

"3. Good lemon-juice should be issued daily (1 oz.), and it should be seen that the men take it.

"4. Vinegar ($\frac{1}{2}$ oz. to 1 oz. daily), should be issued with the rations, and used in the cooking.

"5. Citrates, tartrates, lactates, and malates of potash, should be issued in bulk, and used as drinks, or added to the food. The easiest mode of issuing these salts would be to have packets containing enough for one mess of twelve men, and to instruct the men how important it is to place them in the soups or stews. Possibly they might be mixed with the salt, and issued merely as salt" (*Pract. Hygiene*, 2d ed., 1866, p. 466.)

ANÆMIA.

LATIN EQ., *Anœmia;* FRENCH EQ., *Anémie;* GERMAN EQ., *Anaemie*—Syn., *Blutarmuth;* ITALIAN EQ., *Anemia.*

Definition.—*A special morbid state in which there is either a relative diminution of the mass of blood* (ANDRAL), *with the general composition of the blood differing from the normal standard; or in which the mass of blood is diminished, and the liquor sanguinis is watery, poor in albumen, and containing an excess of salts. These conditions, co-existing with relative deficiency of the red blood-corpuscles* (VOGEL), *and a diminution of the urine-pigment* (PARKES), *constitute anœmia.*

Pathology.—The term *anœmia* literally means absolute *deficiency of blood*—a condition of existence obviously not possible. A diminution in the quantity of blood, with an alteration in its composition, almost never occurs alone, but is generally a morbid state resulting from many exhausting morbid processes peculiar to such wasting constitutional diseases as *scrofula* and *cancer.* In many of these diseases the blood-mass is evidently diminished. We have indications of this diminution in the small pulse, in the pale bloodless appearance of the countenance and surface of the body generally, especially seen in the lips and gums, and in the small blue collapsed veins, particularly obvious by contrast on the pallid skin. In such cases one would never think of drawing blood to know whether or not its constitution was changed; but in cases where the opportunities for examination have occurred, the blood-corpuscles have almost always been found relatively diminished; and accordingly the College of Physicians of London define anæmia simply as "*deficiency of red corpuscles in the blood.*" The causes which lead to this diminution are obscure; and at one time the spleen and glands have been held as concerned in bringing about the disease by some abnormal exercise of their functions.

The result of this morbid condition of the blood chiefly betrays itself—(1.) Upon the *vascular system* generally; and (2.) Upon the *metamorphoses of tissues.*

The blood-vessels contract in proportion to the diminution of the blood-mass. The *arteries* contract generally; and the pulse, whenever it can be felt, is found to have become small and tense. The *capillaries* also contract; the skin and mucous membranes becoming pallid and comparatively bloodless. The *heart's* action in extreme cases becomes irregular, and the whole circulation generally is disturbed.

As regards the metamorphoses of tissues, the muscles and the nervous system appear to suffer first; debility and prostration, both bodily and mental, occur; and in severe cases sensation may be lost, syncope is frequently apt to occur, and even death may result.

The disease seems to go through different changes, according to the causes which bring about the anæmic condition. After mere loss of blood in quantity, for instance, the water and the salts are renewed with most rapidity, the albumen later, then the colourless corpuscles, and last of all the red corpuscles. Hence it is that anæmia is frequently associated with a watery condition of the blood, as well as with a lessening of the number of the blood-cells. It is a matter of the greatest importance in practice to distinguish, if possible, between these conditions, with a view to a rational means of treatment.

Those exhausting diseases which are attended especially with deranged nutrition and sanguification, such as *Bright's disease, carcinomatous diseases, scrofula,* and *suppuration,* lead also to the development of that form of anæmia in which the *liquor sanguinis* is in excess, in which the blood is poor in albumen, containing an excess of salts, and in which the blood-cells ultimately become deficient. A tendency to *general dropsy* or to diarrhœa ensues, nutrition becomes still more disordered, and new formations are apt to become developed.

It is very important to know, in a practical point of view, that every acute disease which occurs in an anæmic individual assumes a peculiar character: a very high degree of debility and prostration ensues, convalescence is protracted, and all severe intercurrent diseases acquire a lingering course.

Symptoms.—In combination with an investigation of the blood, the symptoms and signs of *anæmia* are already read in its pathology. It is only by general symptoms, however, that the physician is able circumstantially to conclude that relative diminution of the cells of the blood co-exists with deficiency of the mass. Great debility is a prominent and striking feature. The skin is of a pale waxen colour; the "whites" of the eyes have a bluish aspect; the mucous membrane of the mouth is colourless; the gums and lips are white; and the tongue is pale, large, and flabby; the pulse in general is about 80, but very feeble, and easily excited—the least stimulus, the least mental emotion or movement of the body, produces great momentary acceleration of the pulse.

The temperature in the axilla is usually normal; although anæmic patients generally suffer from cold extremities. Respiration is hurried on the least exertion. The appetite is bad, and thirst prevails.

When the disease has existed for some time, œdema of the feet and ankles may supervene; and, finally, sweating, in the extreme stage of *anæmia.* It occurs most frequently in the last stages of severe and exhausting sickness of a lingering and prolonged kind, where a high degree of paleness of the skin and mucous membranes exists, with a small weak pulse, collapse of the veins, and a small volume of the heart, spleen, and liver.

Mental depression is usually distressing, the temper variable, and intelligence torpid.

In recent days diagnostic characters of *anæmia* have been recognized in various *murmurs* which may be heard in some parts of the vascular system of anæmic patients. Although the seat, the causes, and the signification of these *murmurs* have been very much disputed, yet the following account may be given of them, based on numerous observations and investigations, especially those of Vogel and of Walshe.

There are three kinds of anæmic *murmurs* which may be distinguished, namely—(1.) *Cardiac murmurs;* (2.) *Arterial murmurs;* and (3.) *Venous murmurs.*

The *cardiac anæmic murmur* gives forth what is usually called a "bellows sound," sometimes intense, sometimes faint, and which generally accompanies the first ventricular sound of the heart. The position where these *systolic* cardiac murmurs is best heard is of some importance, as indicating anæmia rather than disease of the heart. They are heard towards the base of the heart in anæmia, rather than towards the apex, as in organic disease of the valves (WALSHE). The *diastolic* murmurs are really of venous origin; and their intensity is more or less increased by suspension of the respiration.

The *cardiac anæmic murmur* occurs most frequently in true anæmia, especially if cardiac excitement is associated with it; and simultaneously with it we frequently hear *arterial* and even *venous murmurs.* It has been observed by Vogel and others, however, that while the presence of these *cardiac murmurs* may be employed as an auxiliary sign together with others in the diagnosis of anæmia, yet it is not to be concluded from their non-existence that anæmia is not present. Such *murmurs* are not heard in all anæmic patients; and, although present, they are not in every case due to anæmia, because they occur in organic diseases of the heart, such as valvular diseases and endocarditis; and a physician trusting to and acting upon stethoscopic signs *merely*, without a due appreciation of general symptoms, seriously injures the patient, and does an injustice to the science of medicine.

The *arterial anæmic murmurs* are seldom heard. They consist of an intermittent blowing, sometimes soft, sometimes sharp sound, perceived to be synchronous with the beat of the pulse, which gives at the same time a thrill to the finger, so that the *murmur* may be inferred by practice from the nature of the pulse. It is only in the larger arteries in which they are heard, such as the brachial, the subclavian, the femoral, the carotid, and abdominal aorta. They are most frequently heard after great losses of blood; and sometimes also in *chlorosis.* Vogel has heard them during the paroxysms of intermittent fever, while they are absent during the intermissions. They may be heard in typhus fever. No positive conclusion can therefore be formed from them alone regarding anæmia.

The *venous murmurs,* or *hums,* significant of *anæmia,* are *continuous,* humming, buzzing, occasionally musical, singing murmurs, easily distinguishable from the blowing *intermittent* arterial murmurs. They are most frequently heard on the right side of the neck, at the junction of the external and internal jugular vein; but they may also be heard in the femoral veins in their maximum degree of intensity. They may be heard, also, over the course of the superior longitudinal sinus, and at the maximum intensity over the *torcular Herophili* (WALSHE). They are supposed to be produced by abnormal oscillations of the venous valves, or by sharp collision among the blood-discs, passing from sideward veins into a large vessel. When the venous murmurs are strong, they may not only be *heard* but also felt as a gentle thrill, by placing the finger on the part.

These venous murmurs are seldom absent in well-marked anæmia. Nevertheless, anæmia is not to be positively inferred from the mere presence of any one of these murmurs.

The characters of the anæmic murmur are given by Dr. Clymer as follows:—

"(1.) It accompanies the first sound of the heart, which it commonly more or less masks; (2.) It is short; (3.) It is usually soft, but not always, for it may be harsh, though not absolutely grating; (4.) It is exceedingly local, the maximum of intensity being generally in the fourth intercostal space, near the sternum; more rarely limited between the fourth and fifth; beyond these points it is never heard; it follows the course of a line drawn from the point of greatest intensity to the inner portion of the right clavicle. Dr. Potain, in his article Anæmia (*Dict. Encyc. des Sciences Médicales*, t. iv., p. 392, Paris, 1866), says, 'The site of the cardiac anæmic murmur is in the arterial orifices, and especially in the aortic orifice: no one doubts this.' This was the received site of the murmur by all, except Hughes, until quite recently. He, remarking that it cannot be heard below the nipple, and in the axilla, as is the case in aortic murmurs depending upon organic disease, or generally on a level with the ascending aorta, or at the arch, was disposed to place it in the orifice of the pulmonary artery (*Guy's Hospital Reports*, 1851). Quite lately, Dr. Parrot has given some excellent reasons for believing the site of the anæmic cardiac murmur to be in the right auriculo-ventricular orifice (*Archives Gén. de Méd.*, Août, 1866); its maximum of intensity, he observes, is in the fourth intercostal space, near the sternum, corresponding with the right auriculo-ventricular orifice; and its line of propagation is in the course of the superior cava, and not of the aorta; and he further assigns insufficiency of the tricuspid valve as the cause of the murmur."

"The mechanism both of the cardiac and venous anæmic murmurs have generally been ascribed, (1.) To increased friction of blood, whose viscidity is diminished, against the walls of the vessels; (2.) To increased celerity of the blood, and consequent loss of relation between the contents of the ventricles and the area of the openings leading from them; (3.) The passage of thin watery blood through tense veins (OGIER WARD). Dr. Flint has suggested spasm of the papillary muscles, causing insufficiency of the mitral valve, as the cause of the cardiac murmur; and Marey believes it due to diminished arterial tension, and increased rapidity of the ventricular systole.

"Whenever a proper examination can be made of the veins of the neck, particularly at the base, the seat of the two pulsations—and sometimes only one, synchronous with the beat of the radial artery—will be found to be limited to the course of the vein, to be from above downwards, to follow a line which makes an acute angle with the artery, and to cease to be heard immediately on the least compression above the point of pressure; all of which goes to prove that the pulsations are due to a reflux of blood into the right cavities of the heart—in fact, a true venous pulse. Now, Dr. Parrot has proved (*Archives Gén. de Méd.*, 1865), that the pulsations of the external jugular veins, double and single, provided the latter are synchronous with the radial pulse, are a sure sign of imperfect occlusion of the right auriculo-ventricular orifice by the tricuspid valve, at the moment of the ventricular systole. He has subsequently (*loc. cit.*) attempted to establish a relation between the venous murmurs and the cardiac murmur, which happen simultaneously, and he looks upon the latter as caused by the former, 'for,' he says, 'of all the conditions which can give rise to intra-cardiac blowing, no one is more favourable than insufficiency of the auriculo-ventricular valves, since the blood, driven with force and rapidity by the ventricle, passes from a cavity where it is subjected to great pressure, and through a narrow orifice, into the auricle, a thin walled sac, where the compressing force is feeble.' "

One constant urinary character attends both anæmia and chlorosis—namely, a diminution in the urine pigment, which is often reduced to one-fourth or one-sixth of its normal amount. Very generally there is also lessening of the free acidity; and urine which is pale and almost neutral during the whole of the twenty-four hours is almost as good an indication for the use of iron as the pallor of the skin itself. The amount of iron in the urine of anæmia is often very small. The quantity of urine may not be decreased, and may be in some cases large. Its specific gravity is low, (PARKES, l. c.) With regard to other constituents, such as urea and uric acid, the accounts are very contradictory.

Causes.—As an independent constitutional disease, anæmia may be said to owe its origin to three sets of conditions,—(1.) Copious loss of blood, such as by hæmorrhage or venesection, or oft-repeated small losses of blood. (2.) From loss of other fluids of the body besides blood, especially of such as contain albuminous, fibrinous, mucinous, or caseinous substances, such as the excessive secretion of milk in protracted suckling, suppuration, profuse blenorrhœa, leucorrhœa, diarrhœa. (3.) From insufficient and improper nutriment, or from disturbances in the absorption and assimilation of food, and the process of sanguification; or from repeated temporary interruptions to oxygenations of the blood, as by imperfect ventilation of sleeping rooms or coal mines. (4.) It may result from the co-operation of many influences; for example, excessive bodily and mental labour; continued excitement, pain, care, grief, hardships; many acute and chronic diseases, some of which augment the consumption of blood, while others impede its formation—this effect being produced by an acute disease under certain circumstances, when its invasion is intense and its duration prolonged, such as by *scrofula, carcinomatous diseases, diabetes;* from poisoning by malaria—the *malaria-chlorosis* of Vogel, or *paludal cachexia* of Martin, as in the "used-up" condition of our Bulgarian troops, described by Dr. H. Mapleton in *Parliamentary Paper* 247, for 1856, p. 253. "It is the most general of all the conditions incident to tropical invalids" (MARTIN). Anæmia is often the result of such poisons as *lead, mercury, iodine, aniline, tobacco,* and of diseases such as *syphilis, rheumatism, goitre.* Tailors, shoemakers, cooks, firemen of steam-vessels, and female operatives in large factories are the most frequent sufferers from anæmia.

Treatment.—The energies of the physician must be directed to discover and counteract the cause of the anæmia. Nutritious substances must be supplied for diet, in the shape of easily digested meat and broths. The purely tonic treatment, in the combination of air, exercise, and diet, must be carried out as far as practicable. A change of air is absolutely necessary, and generally also of diet. Iron is one of the best medicinal remedies. The astringent preparations are pre-eminently tonic; and are especially useful when the anæmia is associated with or dependent upon inordinate discharges. *Solution of the perchloride of iron,* in the form of *tinctura ferri perchloridi,* in doses of ten to thirty minims in water; or *infusion of quasia* or of *calumba* has properties in common with the numerous salts of iron, and is one of the most reliable preparations. If the anæmia is associated with *diarrhœa*, or *menorrhagia*, or *leucorrhœa*, the *solution of the pernitrate of iron* in similar doses is attended with benefit. A preparation which is recently new to the pharmacopœia—*the syrup of the phosphate of iron*—possesses the general properties of the ferruginous compounds, and is of great service when the *anæmia* is associated with certain forms of *dyspepsia*, or with *amenorrhœa*. It invigorates and increases the powers of digestion, and may be given to the extent of one to three drachms for a dose in water.

Another phosphatic preparation of very great value is that which has been devised by the late Dr. Easton, Professor of *Materia Medica* in the University of Glasgow. Although it is not in the pharmacopœia, and although its mode of preparation had not been published before Dr. Easton kindly sent it to me for publication in the previous editions of this work, yet the combination has become very popular throughout the country as a general tonic in *anæmia* and *cachexiæ* generally. As such, it has been largely used by my colleagues, Professors Maclean and Longmore, amongst the used-up cachectic and anæmic soldiers under treatment at the Royal Victoria Hospital at Netley; and I would add my testimony to its being a most valuable medicine in general practice. The following is the original formula devised by Dr. Easton for the preparation of the *phosphates* of *iron, quinine,* and *strychnia,* in the form of a syrup ("*Syrupus ferri, quiniæ et strychniæ phosphatum*"):—

℞. Ferri Sulph. ʒv.; Sodæ Phosph., ʒvi.;* Quiniæ Sulph., grs. cxcii.; Acid Sulph. Dil., q. s.; Aquæ Ammoniæ, q. s.; Strychniæ, grs. vi.; Acid. Phosph. Dil., ℥xiv.; Sacchar. Alb., ℥xiv.

"Dissolve the sulphate of iron in one oz. boiling water, and the phosphate of soda in two oz. boiling water. Mix the solutions, and wash the precipitated phosphate of iron till the washings are tasteless. With sufficient diluted sulphuric acid dissolve the sulphate of quinia in two oz. water. Precipitate the quinia with ammonia water, and carefully wash it. Dissolve the phosphate of iron and the quinia thus obtained, as also the strychnia, in the diluted phosphoric acid; then add the sugar, and dissolve the whole, and mix without heat. The above syrup contains about one grain phosphate of iron, one grain phosphate of quinia, and one thirty-second of a grain of phosphate of strychnia in each drachm. The dose might therefore be a tea-spoonful three times a day.

"The amount of phosphate of quinia might be increased according to circumstances; and if eight grains of strychnia were employed in place of six, as in the above, the phosphate of strychnia would be in the proportion of the one twenty-fourth of a grain in every fluid drachm of the syrup. I would scarcely venture on a much larger dose. In cases of delicate children, with pale countenances and deficient appetites, I have given, with great benefit, a combination of equal parts of the above syrup and of that prepared by Mr. Edward Parrish, often called chemical food. To children between two and five years of age, the dose of this combination may be a tea-spoonful three times daily.

"The preparation ought to be kept as much as possible from light and air, otherwise the preparation *loses* its characteristic *opaline* appearance, and assumes a fawn colour; a precipitate may be thrown down."

It is prepared by Messrs. Murdoch, chemists, in Glasgow; by Duncan and Flockhart, in Edinburgh; by Randall and Sons, Southampton; and by Savory and Moore, in London.

In some cases the *astringent* preparations of iron are not suitable, and are apt to irritate delicate stomachs, or those in whom any inflammatory local disease exists. For delicate females and children the *saccharated carbonate of iron* is a most valuable preparation, in the form of *mistura ferri composita*, to the extent of one to two ounces for a dose; or in the form of the *pilula ferri carbonatis*, in doses of from five to twenty grains in the twenty-four hours. The *citrate of iron and ammonia* is another remedy which possesses scarcely any astringency, and may often be given in cases of anæmia when the stomach will not bear more astringent preparations. Five to ten grains of this salt may be taken during the twenty-four hours. It is best taken during effervescence, prescribed in *solution of citric acid*, and *not* in *bicarbonate of potash solution.* If it is put into the latter, carbonic acid will be given off, and probably burst the bottle. *Tincture of orange-peel* is the best flavouring agent; but as the salt will not dissolve in the tincture alone, it is necessary to dissolve the salt in water first, and then add the tincture, otherwise the division into doses is impracticable (SQUIRE).

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* A little more phosphate of soda gives a better result—say ℥i.

When it is desirable to continue the use of iron for a long time, as in the *anœmia* of neuralgic affections or *tic-douloureux*, or to give it in large doses, the *magnetic oxide of iron* is the best preparation to administer in doses of five to twenty grains twice or thrice a day in water. The *reduced iron*—the *ferrum reductum* of the British Pharmacopœia—is also a remedy which does not possess the astringent properties of the other preparations, and is one of the most powerful remedial agents in cases of anæmia. *One* to *five* grains may be given several times a day in powder or in pill. It has no taste, and *one* grain is equal medicinally to *five* grains of the citrate of iron. The *citrate of iron* and *quinine*, either in solution or in the form of a pill, is a new and useful preparation, in doses of *five* to *ten* grains three times a day. Such a preparation of iron must be found as will not check the digestion of other food. If loss of appetite and feverishness prevail, the form of the remedy is probably unsuitable, and requires to be changed. Chlorine, in the form of warm hydrochloric acid baths, is highly spoken of by Dr. T. K. Chambers, as an agent of great value, and as a directly restorative medicine in anæmia (l. c., p. 332). The bowels are to be kept regular by four grains of the pills of aloes and myrrh taken at bedtime.

Ferruginous remedies are always of use when we have reason to believe that there is a diminished energy in the formation of blood-cells; and if the urine be pale and almost neutral during the whole of twenty-four hours, it is as good an indication for the use of iron as the pallor of the skin in cases of *anœmia* (PARKES). Dr. Trousseau was of opinion, however, that for *anœmia*, complicated with tubercles, *iron* preparations were unsuitable, as tending to hasten their development. Dr. Clymer has found *arsenic* a valuable remedy combined in the form of *chlorides* of *arsenic*, *iron*, and *quinine.*

CHLOROSIS—Syn., GREEN SICKNESS.

LATIN EQ., *Chlorosis*—Idem valet, *Pallor luteus fœminarum;* FRENCH EQ., *Chlorose*—Syn., *Pales couleurs;* GERMAN EQ., *Chlorose*—Syn., *Bleichsucht;* ITALIAN EQ., *Clorosi.*

Definition.—*A deficiency of the blood-cells, with redundancy of the serous part of the blood, occurring in young girls at the age of puberty, and sometimes in the young of both sexes before the complete development of the distinctive characters of the sexes towards puberty has been effected.*

Pathology and Symptoms.—A very indefinite idea is associated with the name and nature of this disease. Many employ the term precisely in the same sense as *anæmia;* or they limit the term *chlorosis* to those forms of anæmia whose causes are unknown. The original use of the term is expressed in the first part of the definition, as limited to that form of anæmia which occurs in the female at the period of puberty. It is to be classed amongst the "*functional diseases of the female organs of generation in the unimpregnated state.*"

A change in the complexion constitutes the most striking symptoms of chlorosis. A marked pallor of the skin prevails, sometimes perfectly pale, at other times yellowish, greenish, or waxen coloured. The lips and mucous membranes are also pale—symptoms which are the result of the deficiency of blood-cells, and so of blood-pigment. Slight dropsical swellings occur, such as œdema of the feet and ankles, the face and the eyelids, and a bluish halo sometimes encircles the orbit.

The temperature of the body is generally diminished—the breath is cool, the lips, nose, ears, hands, and feet are cold; and chilliness of the body prevails, which is morbidly sensitive to external cold, and seeks warmth. As in other forms of anæmia, the patient suffers from great prostration of strength and debility; she is tired by the slightest exertion, and the weakness sometimes becomes so great as to lead to fainting. The patient is languid, listless, sedentary, and indisposed to exertion. She is nervous, low-spirited, and frequently a prey to irregularities of temper.

Hysteria may prevail with *chlorosis.* The patient frequently suffers from vertigo, headache, often recurrent, *tinnitus aurium*, especially of the right ear, sparks in the eyes, tendency to fainting, neuralgia, spinal irritation, and convulsions, or a "heaviness for sleep." The mind is sometimes morbidly impressed with grief, while despondency and frightful dreams prevail; there is the apprehension of nightmare, melancholy thoughts predominate, mania may become confirmed, and the insanity may assume a suicidal tendency.

The nervous system generally is always more or less implicated.

Disorders of digestion attend the disease. Appetite is diminished or perverted, or even depraved. Acids and highly flavoured foods are craved for, and sometimes such substances as chalk, paper, ashes, coal, plaster of Paris, hair, earth, and even excrements, are desired to be eaten. Substances also very difficult of digestion are longed for, and are sometimes digested better than simpler kinds of food. Constipation frequently and obstinately co-exists; but afterwards diarrhœa may alternate, and lead to *hæmatemesis* or *melæna*, when the evacuations are usually scanty, dark-coloured, and fetid. A singular and peculiar pain of one or both sides, which suffer together or alternately, is a frequent symptom. It is referred generally to the region over the false ribs and the *ilia.* The recurrent nature, the particular situation, and the alternating character of the pain, are peculiar and characteristic. It is not aggravated by a deep inspiration, although it may seem to be so at first.

Other functions are no less disordered—for instance, the respiratory, the generative, and the circulatory.

Respiration is oppressed, or performed with difficulty, and the breath is offensive. Breathlessness prevails, and is experienced especially on any exertion; sometimes also fits of dyspnœa; and sometimes a sonorous cough occurs. Menstruation is generally absent or performed imperfectly, irregularly, and with pain, and the flow is thin and watery, or leucorrhœal. The condition of the menstrual discharge is generally very easily impaired. The catamenia become irregular in their return, inconstant, or of short duration in their flow, deficient in quantity, and pale in colour, and terminating in a state of leucorrhœa. In some cases each return of the catamenia is preceded and attended with much pain in the back and in the region of the uterus. Later in the disease there may be complete *amenorrhœa.*

The conditions of the vascular system are variable. The pulse is slow, feeble, and soft, but sometimes frequent, and always easily accelerated; the heart and great vessels are irritable; palpitation is common; or, more frequently, a sense of fluttering in the *præcordia,* with irregular action of the heart, or imperfect syncope and murmurs, as already described, may prevail. It is to be observed, however, that, apart from mere lassitude and palpitation, patients are sometimes chlorotic for weeks, without any other marked symptoms; and the same complication of intercurrent acute diseases is apt to happen as described under anæmia. The condition of the urine is similar to that which prevails in anæmia.

Causes.—The conditions which bring about this constitutional affection are similar to those already mentioned. The disease is frequent in females between the ages of *sixteen* and *twenty-five* years, is often of long duration, lasting for months, and even for years, with a tendency to relapse after cure.

Diagnosis.—It is especially necessary that the physician should not confound *chlorosis* with *jaundice* on the one hand, or *disease of the heart* on the other, or with *scrofula with incipient deposit of tubercle,* or inflammation within the chest or abdomen.

Treatment is chiefly conducted by change of air and diet, and by medicines. All the functions of the body must be carefully observed, watched, and regulated. The residence of the patient must be a reputed healthy spot in the pure air of the country, rather than the town. Dry frictions of the back and limbs are also recommended, with bathing in the sea, where it can be borne.

The food must be regulated so as not to be too stimulating nor disgustingly bland;—frequent change is demanded, with a due attention to the proper apportioning of nutritive and respiratory elements. The diet should be generous, and carefully apportioned to the powers of digestion. Any symptoms of dyspeptic oppression, impaired digestion, or offensive stools, demand a careful re-adjustment of the dietary. Three meals, or perhaps four, may be taken during the day—the intervals between the meals being equal; and, half an hour before each of the meals, two grains of *capsicum*, with one grain of *quinine*, may be given in a pill. The breakfast should consist of biscuit, dry toast, or stale bread, with fresh butter, and perhaps an egg, and one small cupful—not more—of hot black tea. Five or six hours after breakfast, and after the pill, a dinner of *well-done* meat, such as mutton chops, may be eaten with potatoes; porter, beef-tea, or milk being taken for drink, according to taste and the powers of digestion. The evening meal may be similar to the morning. At first there may be a loathing of all kinds of food in the form of regular or "ordinary diets." In such cases no meals should be prescribed, and no solid food; but a cup of milk, with a third part of lime-water in it, may be given every two hours, prescribed as a medicine, and a pint of beef-tea in divided doses may be taken as a drink during the day. This diet may be gradually added to as the appetite improves.

Trousseau observes that "there are some chlorotic girls who would rather die from inanition than eat ordinary food. We should not hesitate in such cases to make those therapeutic concessions which we are so often obliged to submit to in the practice of our art. I allow alimentary substances, allowed to be very indigestible—caring not for the kind of food so long as it is eaten—as radishes, salads, fruits hardly ripe, highly-seasoned sausage meat, old cheese, vegetables, meats prepared with vinegar, acid drinks, and the like. All I require in this whimsical diet is, that there be sufficient variety. By such means we often succeed in rousing the digestive functions, bring back the sensation of hunger, and gradually lead the patient to proper food.

Change of air and bathing will also greatly aid in restoring health. If the patient lives in a town, she ought to be sent into the country; and, best of all, to the sea-side, where sea-bathing may be prescribed, if the strength of the patient is sufficient. Should the patient have been living in the country, a change of air and scene to a lively town will be no less useful as a remedial agent.

The much-be-quacked use of water, in its varied modes of appliance to particular cases, will be found a powerful restorative agent, if judiciously used by the patient under medical advice.

Of medicines, the preparations of iron have most reputation. They seem to act by promoting the formation of the red blood-corpuscles; and they operate best in those cases in which the blood is rich in albumen. They also act as stimulants to digestion; and, from what has been stated at page 85 *et seq.*, it is not altogether immaterial which of the numerous *officinal* preparations of iron are to be prescribed. Almost every physician has some preparation of iron he fancies better than another; and some of them seem to be aided in their good effects by combination with *carbonate of potash*, such as the *sulphate of iron*, in doses of *three* to *five* grains, or of the *ferri sulphas exsiccata*, in doses of two to five grains, prescribed in pills. The use of ferruginous remedies must be persevered in for months, and the general indications for prescribing the preparations are the same as those given under anæmia, page 85. In pills, the iron preparations may also be combined with *extract* of *nux vomica*, or with *strychnia*, in suitable doses; or the *syrup of the phosphates of iron, quinine, dan strychnia* may be given (see page 86); or the eliminative action of the colon is to be promoted by four grains of the pill of aloes and myrrh taken every night at bedtime, or by a grain of the *watery extract of Barbadoes aloes* with a little *gingerine* as a pill. Simple bitter tonics are useful adjuncts to the chalybeate treatment, such as *gentian, calumba,* and the preparations of *cinchona.* They aid feeble digestion.

Where a high degree of serous plethora exists, and produces violent excitement of the vascular system, palpitation of the heart, and congestion of the head, venesection may be practised. It not only acts as a sedative, but aids the radical cure of the disease, inasmuch as it causes the subsequently administered ferruginous preparations to be borne more easily. Both general and local blood-lettings may be used; but the blood must be taken in small quantities, a couple of ounces at a time being quite sufficient (VOGEL).

Tartarated iron (*Ferrum tartaratum*) is also a useful remedy, and may be prescribed with alkalies, in doses of six to twenty grains, dissolved in water.

GENERAL DROPSY.

LATIN EQ., *Anasarca;* FRENCH EQ., *Anasarque;* GERMAN EQ., *Wassersucht;* ITALIAN EQ., *Idropisia generale.*

Definition.—*An accumulation of serum in the areolar tissue, with or without effusion into the serous cavities.*

Pathology and **Symptoms.**—It is a form of extensive or universal *œdema,* and means an infiltration with fluid serum of the general areolar or connective tissue of the body, to which the name of *anasarca* has been given, from the Greek ἀνὰ, "through," and σὰρξ, "the flesh."

The disease usually begins to manifest itself by *œdema* of the feet and ankles—appearing towards evening, and diminishing, if not disappearing altogether, towards the morning.

Such *œdema* is known from its "pitting" under pressure—that is to say, it retains the impression of the thumb and finger when they are made to grasp firmly each side of the ankle over the swollen part. The fluid surrounding the ankle may have gravitated there; but by and by, as the morbid condition persists, which gives rise to the collection of fluid in the connective tissue, the swollen condition extends from the ankles up the limbs, encroaches on the abdominal and thoracic parietes, and lastly reaches the head and upper extremities. The whole body then exhibits a bloated, turgid appearance, sometimes to a very great extent.

When effusion actually begins in the feet and ankles it is usually the consequence of extreme debility, with anæmia. But the *œdema* may be first observed in the face, especially about and underneath the eyes. Such a form is usual in heart and kidney disease.

Where the connective tissue is loose, the local swelling from fluid is generally the greatest; for example, over the dorsum of the foot and back of the hand, eyelids, scrotum of men, and *labia pudenda* of females.

The lower extremities are often so enormously swollen as to become shining and tense; when the cuticle may rise like a blister, or even giving way, an erysipelatous inflammation may pervade the limb. Sloughs are then apt to form in the connective tissue; open sores follow and become the outlet of excessive discharges of serum, which greatly relieve the patient.

Sometimes, but not always nor necessarily, the anasarca is attended with effusion of fluid into the serous cavities, thereby greatly increasing the danger and often proving fatal.

The emaciation by such cases is often extreme; but is only manifest on the *anasarca* subsiding from any cause.

Diagnosis.—The progress of general dropsy is as a rule continuous, with longer or shorter intervals, when the disease does not seem to advance. It may resemble general emphysema, which is distinguished from anasarca by its crackling underneath the fingers on handling the parts.

The **Treatment** of the disease is regulated by the nature of the disease which causes the dropsical state, and will be considered under *Heart diseases* and *Kidney diseases.*

BERI-BERI.

LATIN EQ., *Beriberia*; FRENCH EQ., *Béri-Béri*; GERMAN EQ., *Beri-Beri*; ITALIAN EQ., *Beri-Beri.*

Definition.—*A constitutional disease, expressed in the first instance by anæmia, culminating in acute œdema, and marked by stiffness of the limbs, numbness, and sometimes paralysis of the lower extremities; oppressed breathing (anxietas in paroxysms); a swollen and bloated countenance. The urine is secreted in diminished quantity. The œdema is general, not only throughout the connective tissue of the muscles, but the connective tissue of solid and visceral organs in every cavity of the body is bathed in fluid. Effusion of serum into the serous cavities very generally precedes death.*

Pathology and Historical Notice.—This obscure but very fatal disease is little known to pathologists in this country. Though common in various parts of India, the territorial range of its endemic prevalence seems limited in a peculiar manner. It has been chiefly met with on the Malabar coast, in Ceylon, and in that tract of country reaching from Madras as far north as Ganjam (HAMILTON). It is principally endemic in that portion of Hindostan called the Northern Circars—a province lying on the west side of the Bay of Bengal, extending from 15° to 20° north latitude. Madras, in north latitude 13° 6′, appears to be the southern limit of the disease in Hindostan. Towards the north of Madras, in the jails of Guntoor, Nellore, Masulipatam, Rajahmundry, Vizagapatam, Chicacole, Bellary, and Cuddapah, the disease is known to prevail. Towards the south it is never seen. It is said to extend from the coast not farther inland than forty to sixty miles.

It is a disease of a peculiar nature; and it has been extremely frequent and fatal amongst all the troops, both Europeans and natives, at Ceylon (CHRISTIE); and although beri-beri is a far more common disease among the natives of India than among the Europeans, yet the rate of mortality is nearly twice as great among Europeans as it is among the natives. Indeed, next to cholera, beri-beri must be regarded as the most fatal disease (judging of the proportion of deaths to admissions) to which Europeans in India are liable (WARING); while Mussulmans appear to be more subject to the disease than Hindoos (MALCOMSON).

Among European soldiers in India the ratio of mortality or of deaths to admissions from beri-beri is above 26 *per cent.*, and amongst Sepoys it is nearly 14 *per cent.;* and large as these ratios seem, they are small compared with the ratio of mortality which obtains amongst the convicts in the Indian jails, where the *per centage* is as high as 36·5 (WARING).

A residence of several months in a district where beri-beri prevails is necessary to its development (CHRISTIE, HUNTER, EVEZARD); and the greatest predisposition to the disease exists when troops have been about eight or ten months in a settlement.

The influence of season in promoting the development of beri-beri seems to be remarkable. Towards the close of the rainy season the admissions to hospitals are far more numerous than at any other period of the year: damp and moisture, combined with cold, seem very favourable to the production of the disease; and the most severe cases at Trincomalee occur during the change from wet to dry weather (RIDLEY), when a strong and hot land wind prevails, when the atmosphere is extremely dry, and when the night temperature is many degrees lower than that of the day. From August to December, when heavy falls of rain occur, with occasional sultry days, and when the alternations of temperature are at the greatest, and the exhalations the most concentrated, then the admissions to hospital for beri-beri are the most frequent (WRIGHT).

The name *Beri-beri* was given by the Malabar physicians, and has been handed down to us by writers on Indian diseases as a name for almost every fatal disease of debility: paralysis of various kinds, reflex paraplegia, dropsy, anasarca, cachexia, scurvy, and anæmic rheumatism, with various diseases of the heart and pericardium, have all at some time been included under the common name of *beri-beri.* It is therefore necessary to give to the term some degree of precision, by a definition which will embrace the leading phenomena of this remarkable disease.

The majority of the phenomena which characterize the well-recorded cases of this disease are undoubtedly referable to *anæmia.* The debility, the cold extremities, palpitation, dyspnœa on exertion, frequent, small, and quick pulse, the bruit occasionally heard in the neck, the scanty urine, the torpid bowels, the deadly pallor of the tongue, all indicate a condition of anæmia (EVEZARD). The disease makes its advances in an insidious manner, as all forms of anæmia do, without any primary or well-marked train of symptoms; and the indisposition appears to be comparatively slight which exists as a stage precursory to the visible invasion of the fully expressed disease (WRIGHT, EVEZARD). The approach of the final and characteristic features of the disease appears to be very gradually brought about, a constitutional state or diathesis is gradually established, and a form of anæmia sets in, combined with the cachectic dropsy of Andral—a condition allied to that of chlorosis in the female (EVEZARD). Many of the more early observers of the disease concur in regarding the fully expressed phenomena of beri-beri as the result of exhaustion and debility (FARRELL, DICK, RIDLEY).

Morbid Anatomy.—More or less fluid is found in one or all the cavities of the chest; most commonly in the pericardium. The areolar tissue of the heart is in some instances loaded with fluid; and hypertrophy of its substance is not unfrequent. The areolar substance of the lungs is loaded with water; likewise the substance of the brain. The ventricles contain an increased amount of fluid; and fluid is effused over the surface of the brain.

The abdominal cavity and the general connective tissue of the body abound in fluid. In general, it may be said that in every case examined after death serous fluid in one or more cavities was found to exist; and the pericardium alone, or in conjunction with some other cavity, was the seat of effusion in above 83 *per cent.* of the cases—the quantity of fluid varying from two ounces to a pint. The spinal cord is variously altered, either by minute effusion of fluid into its substance, or by congestion of its vessels, thereby accounting for the phenomena of paralysis. The unsteadiness of gait and the paralysis of the lower limbs may be also in some measure explained by the mechanism of the spinal canal, combined with the presence of serous effusion in its connective tissue. When it is remembered that the spinal cord more completely fills the vertebral canal in the dorsal region than it does either above or below, it will be understood how symptoms of compression from serous effusion will most early manifest themselves by paralysis or unsteadiness of gait in the lower limbs. The kidneys have been found enlarged, softened, and anæmic; the heart is generally found enlarged, pale, flabby, and softened; the lungs are œdematous, and serous effusion into minute connective texture is the only uniform appearance which exists in every case (WARING).

Symptoms.—Like rheumatism and gout, the disease expresses itself under several forms, of which three may be noticed, namely:—

1. The *acute*, *severe*, or *inflammatory form* of this disease is generally the culmination of the constitutional and local phenomena in a first paroxysm. Numbness, paralysis, and œdema of the extremities are the leading symptoms, followed by dyspnœa, and oppression at the *præcordia.* For a short time previous to any other obvious symptom, the patient, though robust-looking, may not have been able to exert himself in consequence of the partial loss of the use of the lower limbs. This rapidly increases, till he finds that there is inability to walk, accompanied with œdema of the extremities, which very soon passes into general anasarca, affecting the innermost recesses of the textures—if such an expression may be permitted. Febrile symptoms are associated with this acute or arterial anasarca. The skin is hot and dry, the urine is scanty and high-coloured, the bowels are costive, and the stomach irritable. There are rapid and full pulsations of the large arteries, while the pulse may be variable at the extremities, accompanied generally with dyspnœa and symptoms of effusion within the chest. In other cases there may

be headache, restlessness, and delirium, with a slow and full pulse, indicating serous effusion and pressure on the brain. When the œdema is general, and becomes rapidly developed, the condition of the blood is changed from its anæmic character. It becomes dark and ropy, resembling in some degree the appearance of the blood taken from a patient affected with cholera (WRIGHT).

2. In the *second, the asthenic*, or *chronic* form of beri-beri, the patient is very often more or less worn out by some previous disease; or he may have had a previous acute attack of beri-beri, of which there may be a relapse; and it appears that men in whom the disease has once manifested itself are the more subject to future attacks (CHRISTIE), for it is found that one attack predisposes to another (WRIGHT); and then the dropsical symptoms more generally resemble those observable after protracted fevers or other debilitating causes. Abdominal dropsy is most prevalent, accompanied with symptoms of general relaxation—a small and quick pulse, constipated bowels, scanty urine, loss of appetite, universal œdema, much pitting on pressure, and paralysis of the extremities.

The heart partakes of the general debility. It is flabby, and the venous circulation becomes retarded. Soon, perhaps, it dilates, when a temporary bellows sound may be heard. After several such attacks and recoveries the heart becomes thickened, and hence we have the *post-mortem* appearance of either a large and flabby heart, or of one eccentrically hypertrophied (EVEZARD).

3. In the *third* and *mildest form* of the disease the patients are first attacked with some stiffness or rigidity of the legs and thighs, succeeded by numbness, slight œdema, and sometimes paralysis of the lower limbs. The œdema is in general limited, with slight pitting on pressure. There is no unnatural heat of skin; the pulse is seldom above the natural standard; the urine is scanty; and the appetite unimpaired. There may be occasional palpitations of the heart, with costiveness, blanched conjunctivæ, flabbiness and paleness of the tongue, and whiteness underneath the nails. Although such patients generally say that they are well, they will sometimes acknowledge a slight feeling of numbness and coldness of the extremities—symptoms which would readily disappear under appropriate treatment; but after a close night, with either a fog or a shower of rain, such a patient would apply for medical aid in the morning, with a scarred aspect, sighing breathing, violent palpitation of the heart, sometimes with a diffused impulse, pain in the præcordial region, and a variable fluttering pulse. In such cases there are also dyspeptic symptoms, with acid eructations and puffiness of the stomach. The scanty and high-coloured urine has an acid re-action when voided, shows a specific gravity of from 1025 to 1040, and contains an excess of urea. It is such cases which are apt *suddenly* to become aggravated, and to pass into the acute form of the disease. The œdema then progresses from the lower limbs to the hands; the throat becomes swollen, and the face bloated. A sense of numbness is experienced round the mouth, and a general sense of numbness is experienced all over the body, particularly over the extremities, which appear to be unusually weighty and rigid (hence, when walking, the gait is unsteady). The

urine, secreted in diminished quantity, is extremely hot when passing through the urethra; and as the disease progresses, a total suppression of the urine generally takes place. A sense of pain and tightness is felt immediately beneath the inferior edge of the sternum, which sometimes becomes so distressing as to induce the patient to solicit that the part may be cut open, hoping to relieve the tightness by that means. Dyspnœa becomes so urgent as to prevent the patient from lying down; heavy sighing (*suspirous breathing*) occurs, with great anxiety and restlessness. The lips and tongue may now be seen to be livid, and the extremities become cold (RIDLEY).

Universal debility, extreme prostration, anxiety, dyspnœa, numbness, œdema, anasarca, and paralysis, are the most characteristic phenomena of the disease. And in some instances, when those phenomena are fully expressed, the advent of the fatal event is so rapid that the patient may die within six, twelve, twenty-four, or thirty-six hours; and in other instances the disease is more frequently prolonged over several weeks. Death is in some cases extremely sudden; and from the anxietas, and the fact that obstructions sometimes occur in the veins (EVEZARD), it is not improbable that death in such instances may be due to *embolism.*

Mr. Ridley especially notices the suddenness of dissolution in most of the cases which he observed among the troops in garrison at Ceylon in 1814. "Very frequently, when speaking to one man," he says, "I have been called to another, whom I had just before left under promising circumstances, and have found him gasping, his eyes protruding, his hands clenched; and in a few minutes he was dead; and it has sometimes happened that the man I was addressing has been taken off in the same manner" (*Dublin Hosp. Reports,* vol. ii., p. 234).

It is clear, therefore, that the disease cannot be referred to granular disease of the kidney with *albuminuria,* although albumen is sometimes present in the urine in a high degree, as Dr. Ranking attempts to show; nor yet with scurvy, as Dr. Morehead teaches. It seems rather to be a constitutional disease *sui generis.*

Causes.—The etiology of beri-beri is but little known; but there are two points that seem well established in the history of the disease, and which must have a prominent recognition in all investigations relative to the causes which develop the disease. These points are,—*First,* The limited geographical range of *beri-beri; Second,* The fact that the morbid train of phenomena is never developed till the patient has resided upwards of eight or twelve months in the settlement where the disease is endemic.

Malaria, alternations of climate and temperature, noxious material in the waters of districts, have all been indicated as operative agents in bringing about the disease. But, looking to the fact that all the phenomena of the disease point to anæmia, it may be generally stated that whatever tends to induce this state will favour the development of beri-beri. Accordingly, the disease will owe its origin in one place and in one person to the operation of a series of conditions which may not obtain in another place and with another person.

Mr. Christie found in Ceylon that the aged and debauched were those most liable to the disease. A great proportion of his patients were men accustomed to lead a sedentary and debauched life, such as soldiers' tailors, shoemakers, and the like; who, working at their trade, were often excused military duty, and who, by double earnings, were able to procure a larger quantity of spirits than other men.

With regard to œdema there is a great tendency, in almost every disease in Europeans, to result in serous effusions, after long residence in India. Indeed, Sir Ranald Martin observes, in his admirable introductory chapter to the second part of his great work on the *Diseases of Tropical Climates*, that of all conditions incident to tropical invalids, anæmia is the most common. With regard to some places—such, for example, as Masulipatam (where *beri-beri* abounds, in the jails especially)—it has been observed by Mr. Evezard that *œdema* is frequently one of the latest symptoms of *anæmia* outside the jail. He also observes that anæmia appears to be almost the normal state of about the third of the population, particularly the higher castes—a circumstance which he states is partly due to the constitution of the Telugoo, and partly to the eliminating power of the drinking-water, which contains so much of *chlorides* and no *iron.* The natives are well aware of the effects of the brackish water which the majority of them are obliged to drink at the end of the rainy season; and when they find themselves suffering from the anæmia, they procure water from Goodoor, a village about six miles due west of Masulipatam. The water at Masulipatam seems to abound in saline material, such as chlorides, lime, magnesia, and a trace of alumina and iron, in the form of a carbonate (EVEZARD).

The sanitary condition of prisoners in jails is, it is presumed, much worse than the free population outside. The criminals in the jails of India have just sufficient food to keep them alive, and scarcely variety enough. They have not sufficient food to store up for fat or warmth, and none of those luxuries which are supposed to check waste of tissue, such as tea, coffee, tobacco, betel, and the like; and they have not sufficient clothing, many of the prisoners having only one cloth. Mr. Evezard, however, considers that the persistence of the anæmic state is more due to the damp and foul atmosphere of unventilated cells than to food. The soil is for the most part salt. The water of the tank in the jail compound is salt, and its recession leaves a wide margin of saline substance in the soil, which re-attracts moisture like a cloth that has been wetted in sea-water, and which afterwards retains and absorbs the moisture. The *chunam* and mud of the jail walls, and the walls of the cells too, are salt; and in the morning they may be seen dripping with moisture, even in the dry season.

Since improvements have been made in drainage, and since the introduction of a mass of iron, which is allowed to rust and corrode, into each *chatty* of water used by the prisoners for drinking and cooking, not a single case of beri-beri has occurred during the wet season. It is lying on the wet ground in wet weather which, Mr. Evezard believes, brings on beri-beri among Sepoys who have previously rendered themselves predisposed by voluntary starvation.

Under conditions such as these, it is not difficult to understand how a state of ill-health is insidiously established, during which period some *inbred* morbid material is developed in the constitution, which at last produces the phenomena of beri-beri when certain endemic circumstances favour the full expression of the disease.

Diagnosis.—The most uniform phenomena in the fully expressed disease is the characteristic numbness of the surface generally, and the paralytic affections of the lower extremities which accompany all the forms of the disease. The disease has been confounded with the *barbiers*—a chronic disease in which paralysis, tremors, spasms, contractions of the limbs, and emaciation are the most remarkable symptoms. Some, however, are inclined to regard the acute or first form described as the true *beri-beri*, while others consider the second form described as the disease known by the name of *barbiers.* It is admitted, however, that the one is often associated with the other, either of them being the primary affection, for cases commencing in the form of *barbiers* often suddenly take on the more fatal and acute form of *beri-beri*, while the latter frequently presents the symptoms characteristic of the former (MALCOMSON). The two classes of cases prevail in the same places, seasons, and circumstances, and require the same treatment. It is, therefore, more consistent to consider the disease under the three forms described in the text.

The Prognosis is generally unfavourable, the disease approaching insidiously, the anæmia and constitutional state having often advanced so far as to be beyond the reach of repair. A temporary recovery may be established, but relapses are frequent and convalescence is lingering. A first attack generally leaves unpleasant phenomena behind. In the case of Mr. Ridley, a surgeon of the Royal Artillery, who wrote an excellent account of the disease as he saw it in Ceylon, and who suffered from it himself, the memory became considerably impaired, and an extraordinary fluttering of the heart, almost producing faintness, occurred at intervals after convalescence was established. Violent palpitation succeeded, which, on subsiding, left extreme lassitude and faintness; and when these phenomena first occurred, a violent beating of the carotids came on; and when this rapid pulsation ceased, he was left so extremely weak and languid as to excite considerable apprehension of a fatal result.

In all cases of *beri-beri* a *suddenly* fatal result may be apprehended at any period after the full expression of the phenomena, probably from *embolism* or *cardiac affection.* The symptoms which denote danger are general œdema, much *anxietas*, and palpitation of the heart, the respiration becoming more quick as the fatal termination approaches. It is laboured, quick, and interrupted with sighs and groans, the pulse becoming weak and irregular at the extremities, with palpitations of the heart. If coma or delirium supervene, a fatal event may be expected. Much nausea, obstinate costiveness, a dry and black tongue, are all symptoms of danger. The prognosis is more favourable if the œdema sets in very gradually, and is confined to the lower limbs, when the skin is moist and moderately warm, when the patient enjoys sleep, and when the principal natural functions are unaffected.

Treatment.—*Bleeding* has been considered applicable to those cases where there is extreme difficulty of breathing and delirium, when the patient is robust, and when the œdema does not pit much on pressure, where there is rapid and full pulsation of the large arteries, and if the urine shows the existence of albumen (Wright); but, judging from the pathology of this disease, the constitutional influence of stimulants, of generous diet, and of tonics, ought undoubtedly to be the basis of treatment. Should there be irritability of the stomach, the effervescing draught with doses of laudanum and camphor mixture are useful. Saline drinks should be administered, and the extremities should be rubbed with stimulating liniments and rolled in flannel bandages. In the *asthenic* or *chronic* form of the disease, the strength must be supported by the most nourishing diet that can be given in small bulk, aided by tonics, and wine if necessary; while doses of equal quantities of *squill* and *digitalis* (ten to fifteen drops of each) may be given twice or thrice daily. In the *third* and mildest form, a native remedy called *Treeak Farook* is very useful. The ingredients of this medicine are unknown; but it professes to be the "*Theriaca Andromachi*" of old writers. It is prepared in Venice, and transmitted to India through Arabia, and was first recommended by Dr. Herklotts, of the Madras Presidency, as a remedy in beri-beri. It is a thick extract (in which some terebinthine material largely enters), which is only to be procured from the Moghuls, and in those towns which still keep up some communication with the Arabian Sea. Many observers bear testimony to its good effects in removing the œdema and subduing the pulse (Wright, Traill, Geddes, Malcolmson). In some recorded cases the pulse has fallen in four days from 108 to 84 beats per minute under its use.

The prescription most approved of consists of pills of the following ingredients:—

℞. *Treeak Farook*, ℥ss.; *Pulv. Rhei.*, ʒiiss.; *Confectio. Aromat.*, ʒss.; *Mellis* 9, S., misce, et divide in pill xlviii. (Malcolmson).

The electuary form is also much used in India. The remedy does not seem to be an active medicine, except in combination. Four or five stools are obtained daily under its use, and its action is not accompanied by any violent purging, increase of pulse, or determination to the surface; and after it has been used from one to two weeks, the œdema generally disappears, when the numbness and paralysis subside. If it purges, the quantity of rhubarb must be diminished.

The patient should feed on animal food, wheaten cakes, and milk.

In instances where the native remedy has failed to produce a beneficial effect, *nux vomica* has been more successful, commencing with doses of two grains daily, and increasing the dose gradually according to the physiological result. The extract of *nux vomica*, in doses of half or a quarter of a grain, combined with iron or gentian in a pill, is the most convenient form.

Local abstractions of blood from the spine have also proved useful; and a blister applied over the loins has given relief in many obstinate cases.

No single rule of treatment will apply in all cases. The anæmic condition must be counteracted, on the principles of treatment explained under that disease; and any specially abnormal state, such as diseased heart, must be treated accordingly.

When the disease prevailed very generally in the Carnatic, in 1782 and 1783, the cases were most successfully treated by a pill containing a quarter of a grain of *extract of elaterium*, combined with *extract of gentian*, given every hour until copious watery evacuations were procured; and this plan was repeated every third or fourth day till a cure was accomplished. At another time this plan of treatment was not so successful; the cases recovering best under large doses of *spirits of nitre, antimonial wine, frictions with warm camphorated oil, aperient medicines, wine, and a nourishing diet.*

Mr. Evezard's method of medicinal treatment consists mainly in the administration of *acetate of potash* in gin, in the following formulæ:—

℞. Gin, ℥ss.; Potass. Acet., gr. v.; Aquæ, ℥iii.; *misce.* To be given three times a day. If vomiting persists, Hydrocyanic Acid may be useful in relieving it; to the extent of one drop three times a day, given in milk.

CHAPTER XII.

INFLUENCE OF FOOD ON SOME CONSTITUTIONAL DISEASES DESCRIBED IN THE PREVIOUS CHAPTER.

Rightly to understand the nature of many of the diseases of a constitutional kind which have now been considered, it will be necessary here to review to some extent the influence and effects of certain kinds of food on the human frame. Combined researches in chemistry, physiology, and pathology, during recent years, have cleared up much that was doubtful, and established certain principles on which proper scales of diet may be founded, so as to maintain the health of the body under a great variety of conditions as to labour, confinement, freedom, and exercise. Statistics have shown more decidedly now than hitherto how intimately disease and mortality are associated with the supply of food to the people. The records of the Registrar-General of England, and those of Sir William Wilde in Ireland, the evidence of the Commissioners relative to the supplies in the Crimea during the war against Russia in 1854, 1855, and 1856, and many valuable reports relative to the health of the Navy before and after their diet was changed, fully substantiate these statements.

While physicians were well aware of the intimate connection that existed in a general way between food and disease, it is due to the labours of chemists and physiologists especially that this connection has been reduced to an intelligible form, and that the principles which they have established are now put to a practical use.

The great fact which recent chemical and physiological investigations have established may be expressed thus:—"*That the various alimentary* *substances made use of by man and animals contain at least four classes of constituents, each of which performs its own assigned function in the living animal economy. If the substance contains nitrogen, it seems most fitted for the nourishment of tissue, and has been called plastic or nitrogenous; if it is deficient in nitrogen, and has an excess of carbon or hydrogen, it appears to undergo combustion in the body, and is called a non-nitrogenous or a respiratory element of food (hydro-carbons); if it is fatty in its nature, it performs the double duty of maintaining animal warmth, and of assisting in the assimilation of nitrogenous compounds; and, lastly, if it is saline in its quality, it goes to build up the solid textures of the animal frame, and aids the important work of carrying new materials into the system, and old or effete matter out of it*" (LETHEBY). Man and animals cannot maintain health if their food does not contain all of these constituents; and common instinct, with experience, tells us that these classes must be associated in due proportions, under a variety of modifying circumstances. There are undoubted *habits* of feeding which, while they appear to be dictated by common instinct, are also sanctioned by science. For example, white meat being deficient in fat, bacon is eaten with veal and with fowl; melted butter is used with fish; eggs and butter are mixed with sago, tapioca, and rice; cheese is eaten with maccaroni; salads and vinegar are eaten with cold or salted meat; a vegetable is mixed with an animal diet; bread is eaten with butter, bacon with greens, pork with pease pudding, and so on. Old habits and instincts not only declare that these combinations are compatible, but Science informs us now why such combinations are demanded for the maintenance of health; and when they cannot be obtained, health is endangered, the constitution is gradually altered, temperament is modified, life is shortened, families extinguished, armies are swept from their encampments, and races of men from the face of the earth.

The experience of Dr. Christison (who has paid great attention to this subject for the last twenty-four years) has shown,—(1.) That the most successful dietaries for bodies of men, deduced from practical observation, contain carboniferous and nitrogenous food in proportion of about three of the former to one of the latter by weight. (2.) That while *nitrogenous* may replace *carboniferous* food for supporting respiration, *though at a great loss, carboniferous* food (without nitrogen) cannot replace *nitrogenous* food for repairing textural waste. (3.) The daily amount of nutritive principles of both sets must increase with exercise and exposure, otherwise the body quickly loses weight, and ere long becomes diseased. If the above proportion between the two sets be maintained, the weight of real nutriment per day varies, for adults at an active age, between seventeen and thirty-six ounces; the former being enough for prisoners confined for short terms, the latter being required for keeping up the athletic constitution, or that which is capable of great continuous muscular efforts. (4.) Dietaries ought never to be estimated by the rough weight of their constituents, without distinct reference to the real nutriment in these, as determined by physiological and chemical inquiry.

Dr. Clymer gives the following summary of results from Parkes, Panum, and Ranke:—

"A man of average size and activity will, under ordinary conditions of moderate work, take in twenty-four hours from $\frac{1}{26}$th to $\frac{1}{20}$th of his own weight in solid and liquid food; of so-called *solid food* (bread, meat, &c.), about 40 ounces (*i. e.*, with an average range of from 34 to 46 ounces), and of *water* from 50 to 80 ounces; making in all from 80 to 120 ounces by weight of ingesta. The rate of the solid to the liquid food greatly varies; in most cases it is 1 to 2, but in some only 1 to 1. Much bodily activity requires a large increase in the solid, but less in the liquid food. But as the so-called solid food contains a certain percentage of water, if we consider only the water-free food, the average amount in the twenty-four hours for healthy men will be, *water-free food* 22 to 23 ounces, *water* 60 to 90 ounces. The relative amount of the water-free food to water is usually as 1 to 4 or 1 to 5. Assuming the average to be 23 water-free ounces daily, and the mean weight to be 150 lbs., the body receives $\frac{1}{105}$th of its own weight in water-free solids. The range in different persons is $\frac{1}{80}$th to $\frac{1}{120}$th of the body weight. Each pound weight of the body receives about 0·15 ounces (range from 0·1 to 0·2 ounces) of water-free food, and 0·5 ounces by weight of water in twenty-four hours, the amount differing in rest and activity. The following table gives the average amount for men of mean height (5 feet 6 to 5 feet 10) and weight (140 to 160 lbs.) under different conditions of activity."

"A man will take on an average in twenty-four hours,—

	Water-free food in ounces avoir.	Water in ounces.
When nearly at rest, ...	18·5	70 to 90
When in moderate and usual exercise, .	23	70 to 90
Under great exertion, ...	26 to 30	80 to 100 or more.
Undergoing enormous exertion, . . .	30 to 36 or even 40	Uncertain.

"Much depends on the kind and digestibility of the food; a larger quantity of indigestible food being taken, much passing undigested by the bowels. Of the water about $\frac{3}{5}$ths or $\frac{4}{5}$ths is taken as water; the rest is water in the so-called solid food. A man's food must be increased in proportion to the work; if not, one of two results follows: his usefulness as an agent of force lessens, being unable to do all his work; or he continues his work at the expense of his tissues, his weight decreases, and morbific causes act upon him more easily."

The phenomena of nutrition are owing to the various chemical interchanges of nitrogen and carbon, with the concurrent influences of oxygen and hydrogen (chiefly though not entirely in the form of water) and of various salts. A man of mean weight, height, and activity, requires in twenty-four hours,—

Nitrogen, about	250 to 350 grains.
Carbon, ,, ...	3500 to 5000 grains.
Salts, not fixed, ...	400

"According to Ranke's experiments, the relative amount of nitrogen and carbon during rest and during activity was 1 N to 11 C, and 1 N to 15 C. On an average about $\frac{5}{6}$ths of the carbon are given in the starches and fats, and $\frac{1}{6}$th in the albuminates. When we speak of nitrogenous and carboniferous food, the proportions come out differently. One part of nitrogenous food to from 3 to 6 parts of carboniferous (1 to 4 being the mean) forms the usual proportion in apparently all nations (PARKES).

"The histogenetic nature of food must be determined by direct physiological investigation, which should show comparatively the different influence of aliments upon the metamorphosis of matter in the essential animal tissues.

"Professor Panum, of the University of Copenhagen, recently instituted a series of experiments to ascertain *the degree of accuracy with which it is possible,* by quantitative determinations of urea, *to discover how much albumen an individual can actually appropriate, digest, and decompose from an indefinite quantity of food, consisting solely of albuminous matter and water.* His experiments were made on a dog, and he chose the purest albuminate which can be produced in the pure state—the gluten of wheaten meal. The proportion between the dry albuminate taken and the urea produced was strikingly constant, being, in full feeding, 1 : 4·35; in medium feeding, 1 : 3·58 ; and in slight feeding, 1 : 2·81.

"Professor Panum also conducted a series of experiments with a view to the solution of the practical question, *whether the production of urea can serve also as a measure of the histogenetic nutritive value of such foods as along with albuminates contain carbo-hydrates or fat in any considerable quantity?* It would appear upon the whole that 1,550 grains of starch with 500 grains of butter diminished the production of urea tolerably equally by about 31 grains, corresponding to about 172 grains of dry albuminous matter."

Approximately, it may be concluded that a full-grown man of average weight (140 to 150 lbs.) and height 5 feet 7 inches) requires *one-twentieth* part of his weight in food during the twenty-four hours; that is, *seven* or *seven and a half* pounds of food, including solids and liquids; *one to one and a half* pounds (16 to 24 ounces) being solids, the rest water (PARKES).

On an average, it is found that a man requires *four* or *five* ounces of chemically dry NITROGENOUS food daily;—that in a state of rest he will require *three and a half* ounces; under a state of considerable exertion *five and a half* ounces; and under extraordinary exertion he may require as much as *six and a half*, or even *seven* ounces of dry *nitrogenous* aliment daily. The quantity of HYDRO-CARBONIFEROUS aliment required to keep a man in health cannot be less than from *fourteen and a half* to *fifteen* ounces in twenty-four hours; and even *nineteen* to *twenty-two* ounces under great exertions. The amount of FATTY MATTERS ought to equal about half the quantity of the *nitrogenous* aliment. But in a state of rest about *one* ounce in the twenty-four hours will be sufficient; while under great exertion *two and a half* ounces may be required daily. The amount of WATER required varies from *seventy* to *one hundred and thirty ounces;* and the SALTS supplied in the food should amount to from *half an ounce* to *an ounce* daily, consisting of *chloride of sodium,* chloride of potassium, salts of *lime* and magnesia, carbonates, citrates, lactates, and acetates (PARKES).

To determine by calculation the amount of these different aliments, and therefore the nutritive value of a given diet, the following scale is given, (Table I.) It shows the mean amount of water, nitrogenous substances, fat, and carbo-hydrates—starch and sugar—which ought to be contained in 100 parts of each of the substances in common use mentioned in Table I.*

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* This table is used by Dr. Parkes in the Laboratory of the Army Medical School, and he kindly permits me to give it here.

TABLE I.—NUTRITIVE VALUE OF FOODS IN 100 PARTS (Parkes).

	Water.	Nitrogenous Substances.	Fat.	Carbo-hydrates, Starch, and Sugar.
Meat without Bone,* . . .	74	16	9	...
Fat of Meat,†	63	14	14	...
Bread of average quality (White Wheaten), . . .	40	8	1·4	51
Starch, ...	...	...	...	100
Fat, ...	...	...	100	240 of Starch.
Pease, ...	15	24·4	2·6	49
Potatoes, ...	74	1·5	0·6	29
Rice, ...	10	3·8	0·8	85·2
Milk, ...	87	4·3	3·7	5·2
Maize (after Pozziate), . .	13·5	9·9	6·7	64·5
,, (Von Bilra), . .	10·6	15·09	3·8	67·46

* The mean of usual statements.
† Calculated by Dr. Parkes from the statements of Lawes and Gilbert.

TABLE II.—DIETARIES AND THEIR NUTRITIVE VALUES (Letheby).

DIETS.	WEEKLY CONSUMPTION IN OUNCES.							DAILY DITTO.		
	Bread or Biscuit.	Meat.	Potato.	Meal, &c.	Milk.	Cheese.	Butter.	Carboniferous.	Nitrogenous.	Total solid Nutriment.
Physiological, . . .	140	84	..	..	..	..	3·5	12·7	4·0	16·7
Prison Punishment, .	112	..	..	..	..	..	..	8·2	1·4	9·6
E.County&BoroughJails—										
Under 7 days, . .	121	..	..	23	39·5	..	..	12·4	2·2	14·6
Not hard labour, .	172	7·8	3·2	22·8	15·4	3·5	..	15·7	3·1	18·8
Hard labour, . . .	163	14·6	63·4	27·2	41·6	1·5	..	18·2	3·5	21·7
Scotch Prisons—										
Under 3 days, . .	112	..	..	28	..	..	..	11·2	1·9	13·1
Not hard labour, .	30	7·5	152	73	175	..	..	19·0	3·4	22·4
Hard labour, . .	76	10	176	100	175	..	..	27·0	4·5	31·5
Irish Prisons—										
Under 1 month, . .	56	..	192	70	70	..	..	19·5	2·9	22·4
Not hard labour, .	56	..	192	60	170	..	..	20·5	3·4	23·9
Hard labour, . . .	64	..	219	70·5	170	..	..	22·0	3·6	25·6
Military Prisons—										
Under 84 days, . .	56	..	..	119	210	..	..	22·2	3·8	26·0
Over 84 days, . . .	56	..	..	168	210	..	..	27·8	4·7	32·5
Destitute Debtors, .	156	16	52	22	21	..	..	16·3	3·1	19·4
Convict Prisons, . .	161	36	112	12	12·8	..	..	18·4	3·6	22·0
Unions (Adults), . .	112	15·5	51	17	34	5·2	1·1	14·2	2·8	17·0
Unions (Children), .	90	14	32	..	105	..	3·5	11·1	2·3	13·4
Lunatic Asylums, . .	114	23	68	16	14	7	1·3	13·2	4·0	17·2
Public Hospitals, . .	93	52	56	14	7	..	3·2	12·1	3·5	15·6
Army—										
Crimea, ...	112†	112	..	..	..	..	..	14·5	4·8	19·3
Home, ...	168	84	112	..	..	..	..	19.4	4·8	24·2
Madras, ...	112	112	56	4*	..	..	..	16·5	4·9	21·4
Bombay, ...	140	112	56	56*	..	..	..	22·2	5·6	27·8
Field (India), . .	168	..	..	168*	..	..	..	30·7	3·8	34·5
Navy, ...	112†	112	56	..	..	..	..	17·7	5·0	22·7
‡ Navigator (Crimea),	140	140	..	28	..	..	..	17·8	6·2	24·0
‡ Navigator (Home), .	320	96	64	..	..	12	4	18·6	7·7	26·3
Berwickshire Labourer,	122	..	..	224	224	..	..	37·1	7·0	44·1
Yorkshire Labourer, .	280	126	28	..	210	..	49	42·2	8·8	51·0

In this table only the most important articles of diet are mentioned, although the others, excepting beer, spirits, tea, and coffee, are calculated in the daily consumption. (*) are rations of rice, and (†) are of biscuit. Gruel is calculated at rate of two ounces meal per pint.

‡ A name given to those labourers who are employed in excavating, and such-like laborious work, chiefly connected with the construction of railways. They are also sometimes called "navvies."

TABLE III.—NUTRITIVE VALUE OF FOODS (Letheby).

Substances, 100 Parts.	Water.	Fibrine Albumen, &c.	Starch, Sugar, &c.	Fat.	Salts.	Carboniferous.	Nitrogenous.	Total Nutriment.
Human Milk, ...	89	3·5	4·2	3·0	0·2	11·4	3·5	14·9
Cows' Milk,.	86	4·5	5·0	4·1	0·7	14·8	4·5	19·3
Skimmed Milk, ...	87	4·5	5·0	2·7	0·7	11 5	4·5	16·0
Butter Milk, ...	87	4·5	5·0	0·5	0·7	6·0	4·5	10·5
Beef and Mutton, . . .	73	19·0	..	5·0	2·0	12·0	19·0	31·0
Veal, ...	77	19·0	..	1·0	0·6	2·4	19·0	21·4
Poultry, ...	74	21·0	..	3·0	1·2	7·2	21·0	28·2
Bacon, ...	20	0·8	..	70·0	1·3	168·0	0·8	168·8
Cheese (Chedder), . . .	36	29·0	..	30·0	4·5	72·0	29·0	101·0
,, (Skimmed), . .	44	45·0	..	6·0	5·0	14·4	45·0	69·4
Butter, ...	15	..	..	83·0	2 0	199·0	..	199·0
Eggs, ...	74	14·0	..	10·5	1·5	25·0	14·0	39·0
White of Egg, ...	78	20·0	..	..	1·6	..	20·0	20·0
Yolk of Egg, ...	52	16·0	..	30·0	1·3	72·0	16·0	88·0
White Fish, ...	79	19·0	..	1·0	1·2	2·4	19·0	21·4
Salmon, ...	78	17·0	..	4·0	1·4	9·6	17 0	26·6
Eel,.	80	10·0	..	8·0	1·3	19·2	10·0	29·2
Wheat Flour, ...	15	11·0	70·0	2·0	1·7	74·8	11·0	85·8
Barley-meal, ...	15	10·0	70·0	2·4	2·0	75·8	10·0	85·8
Oat-meal.	15	12·0	62·0	6·0	3·0	76·4	12·0	88·4
Rye-meal, ...	15	9·0	66·0	2·0	1·8	70·8	9·0	79·8
Indian-meal, ...	14	9·0	65·0	8·0	1·7	84·2	9·0	93·2
Rice, ...	14	7·0	76·0	0·3	0·3	76·7	7·0	83·7
Haricot, ...	19	23·0	45·0	3·0	3·6	52·2	23·0	75·2
Pease,.	13	22·0	58·0	2·0	3·0	62·8	22·0	84·8
Beans, ...	14	24·0	44·0	1·4	3·6	47·4	24·0	71·4
Lentils, ...	14	29·0	44·0	1·5	2·3	47·6	29·0	76·6
Wheat Bread,.	44	9·0	49·0	1·0	2·3	51·4	9·0	60·4
Rye Bread, ...	48	5·0	46·0	1·0	1·4	48·4	5·3	53·7
Potatoes, ...	74	2·0	23·0	0·2	0·7	23·5	2·0	25·5
Green Vegetables, . . .	86	2·0	4·0	0·5	0·7	5·0	2·0	7·0
Arrow-roots, ...	18	..	82·0	..	..	82·0	..	82·0

In this table the carboniferous matter is calculated as starch; 10 of fat being equal to 24 of starch.

It is important to bear in mind that the amount of *nitrogen, carbon, hydrogen,* and *salts,* should not alone be considered in calculating the value of diet. The form in which these elements exist is equally important. The *fats* and *starches* are not interchangeable, and should not be confounded under the common head of *carboniferous.* The mean amount of the four classes of solid aliments has been calculated by Dr. Lyon Playfair,—*On the Food of Man in Relation to his Useful Work,* Edinburgh, 1865—from many diets, and is given in the following

TABLE IV.—AMOUNT IN OUNCES (AVOIR.) AND TENTHS OF OUNCES FOR MALE ADULTS (Playfair).

	Subsistence Diet—*i. e.,* sufficient for the mechanical force necessary to carry on the internal work of the body.	Diet in quietude.	Adults in full health, but with easy work.	Adults in active work.	Adults in laborious work.
Nitrogenous Substances, . . .	2·	2·5	4·2	5·5	6·5
Fat, ...	0·5	1·	1·4	2·5	2·5
Starch, ...	12·	12·	18·7	20·	20·
Mineral Matters, ...	...	...	·71	·9	...
Carbon (total), ...	6·7	7·4	11·16	13·7	14·3

The preceding very interesting tables (II., III., and IV.,) of dietaries and their nutritive values, and of the nutritive values of foods, by Dr. Letheby and Dr. Lyon Playfair, are given here to show the actual proportions in which various substances used as food are associated in the several public dietaries of the country, and as a guide to the student of medicine when, as a practitioner, it may often be his lot to devise and construct scales of diet suited to various conditions of existence.

The calamities which happened to our soldiers in the Crimea (in 1854) show that the dietaries of working men cannot be safely reduced below the physiological standard; and, in the words of Dr. Christison, "any person conversant with the science of the present subject could have foretold, as a certain consequence, sooner or later, of their dietary, that the British troops would fall into the calamitous state of health which befell them in the Crimea."

In cases where preserved food must be used for want of fresh food, there are difficulties to be overcome which experience and science have made to disappear in a great measure. It is known that salt meat has a tendency to engender disease, and to favour the development of disease under certain circumstances; but it is not known, either physiologically or chemically, what is the exact nutritive value of salt meat in a scientific point of view. There are three circumstances connected with its use which tend to diminish its value as an article of diet, namely,—(1.) The investigations of Liebig demonstrate that the process of salting meat is very improper, for the brine extracts the juices of the flesh, and so removes most of the important constituents. (2.) The salt meat for the navy and for soldiers in the field is always highly salted, in order to keep for two years or more in every climate; and such highly salted meat must be so thoroughly steeped in cold water, to remove the salt, before it is eatable in large quantity, that much of its most nutritive constituents must be washed out—namely, its albumen and sapid extract, called osmazôme (CHRISTISON). (3.) The sarcolemma of salted meat is always hardened; and hence those foods require a long time for digestion, and frequently disagree with the stomach (LETHEBY). Few can eat a pound of salted meat daily for any length of time, even when fed on rations by no means liberal. Nitrogenous elements must therefore be added to a diet where salted meat predominates, rather than increase the amount of that substance; and this is best done by adding pease, flour, currants, rasins and oil, butter, or lard, to the ration.

"Experience has shown," says Dr. Letheby, "that there are certain articles of food which are not particularly nourishing in themselves, but which serve some very important purposes in the animal economy. This is the case with tea and coffee: in fact, the use of a vegetable infusion, containing astringent matter and an active principle rich in nitrogen, has been almost universal among mankind from the earliest times."

"The physiological action of these beverages appears to be of a somewhat singular kind; for while they excite the brain, they calm the nervous system generally, and though they produce a state of wakefulness and activity, yet they also induce a species of languor and repose.

Lehmann has ascertained by experiment that coffee greatly diminishes the wear and tear of the system: it oils the machinery, as it were, and checks the waste of friction; for those who use it find that during active exercise the destruction of tissue is prevented, and that there is less demand for food; in fact, with a maximum of work to perform, and a minimum of food to accomplish it, he will best sustain his vital power who resorts now and then to a cup of tea or coffee. Hence its value as a means of economizing food, and hence its importance to the poor labouring man." In many of our large merchant-ships the crews are engaged on the condition that coffee shall take the place of grog; and those captains who are careful of the health of the men give them warm coffee before or after they have been aloft in cold and stormy weather—a practice which cannot be too much overvalued.

Effects of Overfeeding.—Too much respiratory food favours the development of fat, and checks the proper nutrition of the muscular tissues; hence it is that rice feeders and potato eaters, and those who indulge in fermented liquors, are often bloated in their appearance, become extremely fat, and are not capable of prolonged exertion. The brewer's drayman is a bad subject for the wards of an hospital; for though he usually has all the appearance of a man possessed of great muscular strength and vital endurance, yet he is not so in reality, for the muscular tissues have been encroached upon by fat, and the general power has been weakened by an undue influence of the respiratory element. Most of the animals in our menageries, from a too liberal allowance of respiratory food, die from fatty degeneration. Accumulation of the nitrogenous elements in the blood is often also a prolific source of disease, and their non-elimination (as shown in previous pages) is conducive to the propagation and development of many general diseases. Attention has now been drawn to the influence of such a condition in establishing the characteristic diseases of overfed convicts (LETHEBY, THOMPSON).

Corpulence.—One of the most distressing results of overfeeding from a wrongly adjusted dietary is *corpulence.* It is to be considered a disease when "it renders persons, from a difficult respiration, uneasy in themselves; and, from inability to exercise, unfit for discharging the duties of life" (CULLEN).

Dr. T. King Chambers, from observations of thirty-eight persons, whose weight varied from 224 to 504 pounds, comes to the following conclusion:—

In the corpulent (1.) The bony framework of the body is less massive than in the spare, as indicated especially by the smallness of the hands and feet.

(2.) The skin is usually fresh coloured and thin.

(3.) The respiratory function presents the following well marked peculiarity—namely, the volume of air the lungs are capable of containing in the chest and expiring from it, is considerably less than the average quantity contained and expired by healthy persons of the same height. The vital capacity is diminished and less carbon expired.

Hence, the wind becomes short and the mental powers inactive from *corpulence* ("Gulstonian Lectures," 1850).

Much has been written on the subject of *corpulence;* and the attention of the public has been recently successfully arrested by one who appreciated the relief he obtained by following the directions of a physician who happened to take a scientifically correct view of his case. Mr. Banting (whose pamphlet on the subject of "corpulence" is now well known) managed to reduce his physical proportions under the following dietary:—

Breakfast—Four or five ounces of beef, mutton, kidneys, boiled fish, bacon, or cold meat of any kind (except pork), a large cup of tea (without milk or sugar), and one ounce of dry toast. *Dinner*—Five or six ounces of any fish (except salmon or eels), any meat (except pork), any vegetables (except potatoes or rice), one ounce of dry toast, fruit out of any pudding, any kind of poultry or game, and two or three glasses of good claret, sherry, or Madeira (champagne, port, or beer forbidden). *Tea*—Two or three ounces of fresh fruit, or a rusk or two, and a cup of tea without milk or sugar. The tea may be very much enjoyed when taken in the Russian fashion—*i. e.*, with a thick slice of lemon floating on the top, instead of milk. *Supper*—Three or four ounces of meat or fish, similar to dinner, with a glass or two of claret. *Nightcap,* if required, a tumbler of grog (gin, whisky, or brandy, without sugar), or a glass or two of claret or sherry.

The quantities of the different articles specified in this liberal diet-roll, Mr. Banting states, must be left to the natural appetite; but for himself he took at *breakfast* six ounces of solid and eight of liquid food; at *dinner*, eight ounces of solid and eight of liquid; at *tea*, three ounces of solid and eight of liquid; at *supper*, four ounces of solid and six of liquid; and the *nightcap* he introduces to show that it is not injurious; whilst, for the encouragement of smokers, it may be mentioned that tobacco is allowable.

When Mr. Banting began this treatment in August, 1862, he weighed 202 lbs.; and after a year's perseverance in it, in September, 1863, he had lost 46 lbs.; and had reduced his girth 12¼ inches. The sound dietetic principles on which corpulence ought to be treated had been previously enunciated by Dr. T. K. Chambers, in his excellent monograph on that subject; and Mr. Banting's dietary is in exact accordance with what is there stated, and with what has been stated in the text. A still more varied dietary might be prescribed on similar principles by reference to the tables at pages 104 and 105.

Effects of Deficient Food.—"A deficiency of food, especially of the nitrogenous part, quickly leads to the breaking up of the animal frame. Plague, pestilence, and famine are associated with each other in the public mind, and the records of every country show how closely they are related. The medical history of Ireland is remarkable for the illustrations of how much mischief may be occasioned by a general deficiency of food. Always the habitat of fever, it every now and then becomes the very hotbed of its propagation and development. Let there be but a small failure in the usual imperfect supply of food, and the lurking seeds of pestilence are ready to burst into frightful activity. The famine of the present century is but a too forcible illustration of this. It fostered epidemics which had not been witnessed in this generation, and gave rise to scenes of devastation and misery which are not surpassed by the most appalling epidemics of the Middle Ages. The principal form of the scourge was known as the contagious famine fever (typhus), and it spread, not merely from end to end of the country in which it had originated, but, breaking through all boundaries, it crossed the broad ocean, and made itself painfully manifest in localities where it was previously unknown. Thousands fell under the virulence of its action, for wheresoever it came it struck down a seventh of the people, and of those whom it attacked one out of nine perished. Even those who escaped the fatal influence of it were left the miserable victims of scurvy and low fever. Another example, not less striking, of the terrible consequences of what may be truly called famine, was the condition of our troops during the early part of their sojourn in the Crimea, in 1854. With only just enough of food to maintain the integrity of the system at a time of repose, and at ordinary temperatures, they were called upon to make large muscular exertions, and to sustain the warmth of the system, in the midst of severe cold (LETHEBY).

Death from Starvation is unfortunately not uncommon. In February of 1862 a man, thirty-six years of age, was discovered lying in a stack of corn or hay, near Morpeth, dying from this cause. All attempts to rally him failed, and he ultimately died. He was an intelligent man, and had been editor and proprietor of a penny journal, called the *Falkirk Liberal.* A diary was found in his possession, containing entries of his condition from the 8th to the 25th of February; from which it appeared that during *seventeen* days he had twice tasted a piece of bread; but for the last *thirteen* days he had been entirely without food. During the first *ten* days of the *thirteen* he was able to obtain water, but on the *eleventh* day he found his legs were useless, and he lost all motor power in the lower extremities, so that "one-half of his body appeared to be dead." The case is of interest to the pathologist, as showing the length of time during which existence can be maintained if water alone be taken.

In cases of very gradual starvation an urgent feeling of hunger is not a prominent symptom; and even when it exists at first it usually soon diminishes, and is succeeded by a feeling of exhaustion and faintness, and even loathing of food, if abstinence has been long protracted (R. B. HOLLAND). The mental condition connected with poverty may in part account for this deficiency of appetite. A depression produced on the nervous system is very early manifested in the impaired energies of all the vital functions, the weakened conditions of the intellectual faculties and moral feelings, and diminution of the general sensibility. Disturbance of the cerebral functions is at first shown by an unnatural languor, despondency, and listlessness, slowness and hebetude of intellect, with an inability to employ the thoughts steadily and profitably on any subject. Notwithstanding all this general languor, however, the patient sometimes manifests a highly nervous state; he is startled by any sudden noise, and hurried by the most trifling occurrences. He is liable to attacks of giddiness, "swimming in the head," staggering, dimness of sight, with temporary delirium, and either falls as in an apoplectic fit, or lapses gradually from a lethargic state into one of stupor, or even of complete coma. In many respects the symptoms in these cases have a considerable resemblance to the effects of exposure to cold. In consequence of the torpor of the brain and intellectual faculties, it is often extremely difficult to obtain the requisite information from patients. Instead of showing any anxiety to communicate the symptoms and cause of their illness, or to relate the privations they have undergone, they generally have an unwillingness to be questioned, lie in a listless or lethargic state, without taking any notice of what is going on, and seem desirous only not to be disturbed. Such listlessness and torpor of the mental faculties, the tendency to fainting, or to perfect syncope, and, finally, a state of cerebral oppression, amounting in some cases to coma, are among the most characteristic symptoms of defective nutrition, and the surest indications of its existence to a serious extent.

When privations of clothing and lodging are added to insufficient diet, long exertion, insufficient repose, intemperance, and the miseries of poverty, the symptoms already detailed are of the most aggravated kind. "Long before insufficiency of diet is a matter of hygienic concern—long before the physiologist would think of counting the grains of nitrogen and carbon which intervene between life and starvation—the household will have been utterly destitute of material comfort;—clothing and fuel will have been even scantier than food; against inclemencies of weather there will have been no adequate protection; dwelling-space will have been stinted to the degree in which over-crowding produces or increases disease; of household utensils and furniture there will have been scarcely any,—even cleanliness will have been costly or difficult; and, if there still be self-respectful endeavours to maintain it, every such endeavour will represent additional pangs of hunger. The home, too, will be where shelter can be cheapest bought,—in quarters where commonly there is least fruit of sanitary supervision, least drainage, least scavenging, least suppression of public nuisances, least, or worst, water supply, and, if in town, least light and air. Such are the sanitary dangers to which poverty is almost certainly exposed, when it is poverty enough to imply scantiness of food" (Simon, *Sixth Report on Public Health*, 1864, p. 14). But a multitude of cases of minor degrees of suffering occur in which the symptoms are less marked than those described. Such cases are indicated by a sallow and dingy appearance of the skin, a soft and flabby feeling of the flesh, more or less emaciation, general debility, feebleness of the circulation, and frequently swelling of the ankles. The stomach becomes disordered, the appetite defective, and digestion impaired. The individual feels languid and desponding, is soon fatigued, incapable of exertion, and has an irresistible desire to fall asleep, from which he is apt to awake suddenly and in a fright. The body is easily chilled, breathlessness and palpitation are experienced after slight exertion, attacks of *vertigo, tinnitus aurium*, and transient blindness, are common, and there is a peculiar forlorn and dejected aspect of countenance which is very characteristic. This state of things is commonly soon succeeded by some specific disease; though it may continue, with only slight variation, for a very protracted period, until the patient falls by slow degrees into a state of mental as well as physical incapacity; and, being no longer able to procure any employment, is completely invalided, and applies for medical relief. It may perhaps be thought that these remarks apply to cases of deficient nourishment which are less frequent; but the experience of those who have practised extensively among the wretched purlieus and miserable abodes which exist in every large metropolitan town can testify to the contrary. I well remember listening to the interesting clinical lectures of Dr. Christison, of Edinburgh, on the cases of *scurvy* which prevailed in that town and its vicinity in 1847, and hearing the melancholy recitals of misery and starvation under which the poor suffered at that time; some under the hard taskmasters of the illegal "truck system," * and others from absolute want at home. Among many, of whose cases I have preserved notes, a shoemaker had to support his wife and five children on *eight shillings* a week; and, in order to feed his children better than himself, he subjected himself to privations which in time developed *scurvy.* His daily diet consisted of *one pennyworth of bread, with tea, but no milk, in the morning—no dinner—and one pennyworth of bread, with tea, and no milk, in the evening.* After existing *three months* on this diet the disease broke out. But, apart from these extreme cases, the instances are innumerable in which deficiency of food acts as a pre-disposing cause of many diseases. It is now generally known that plethora and symptoms of an opposite state very nearly resemble each other, and a discrimination of these differences is of the greatest importance. While coma is often an attendant on plethora, it is not to be forgotten that it is one of the most severe and fatal signs of exhaustion from defective nutrition; and when it supervenes towards the termination of diseases of exhaustion, and the pulse becomes slower, it often acquires a degree of fulness, and gives an idea of strength, quite at variance with its previous character, and little to have been anticipated from the debilitated state of the system.

But in degrees far short of what is popularly known as starvation or famine, insufficiency of nourishment may bring very hurtful consequences to health. Local defects or local peculiarities of diet may exercise an important influence in determining or colouring particular localizations of disease; and generally it may be said, that in order justly to estimate the sanitary circumstances of a people, sufficient regard must be had to the quantity and quality of the people's meat and drink (Simon, *Report on Public Health,* p. 11, 1864).

The injurious effects produced by improper nutrition require to be studied both in relation to food and drink; and the diseases now considered, which, from section B of General Diseases, are greatly influenced by the nature of the food on which the people live.

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* The "*truck system*" became developed chiefly during the formation of our great lines of railway throughout the country. The labourers (navvies) were poor, and came to work without money to buy provisions, and their field of labour was often far removed from any place where food could be bought in quantity. The contractors, their employers, then established provision stores, and in place of paying the men in money, they compelled them to take remuneration for their labour by value received in food. By this method, now declared illegal under all circumstances, the labourers often suffered from a deficient and bad supply of provisions.

It has been found necessary this year (1871) to inquire by Parliamentary Commission into the extent of this system, still carried on in some places.

CHAPTER XIII.

GENERAL MANAGEMENT OF THE SYSTEM LIABLE TO CONSTITUTIONAL DISEASES.

IMPORTANT general indications for the treatment of the CONSTITUTIONAL diseases are suggested by their pathology as expounded in these pages. This general treatment ought to be directed towards the management of the system for the *prevention*, *control*, or *arrest* of the development of such affections, or to maintain them within such limits as are consistent, at least, with the well-being of the individual.

The topics to be noticed under this head are of so comprehensive a scope that they can only be indicated as shortly as possible, with the object of directing the mind of the student to their more extended study. They embrace, in fact, the whole subject of general and individual hygienic management. The student is therefore referred at once to two standard authorities, with whose maxims and practice his mind ought to become most thoroughly imbued, in order to direct successfully the measures for the prevention and arrest of the CONSTITUTIONAL DISEASES. The works here referred to are—(1.) *The Management of Infancy*, by Dr. Andrew Combe, ninth edition, 1860, edited by the late Sir James Clark; (2.) *Practical Hygiene*, 1864, the work of my colleague, Dr. E. A. Parkes, which has passed through three editions. From the first of these books the student will learn how he must begin at the very beginning. He will learn to appreciate the influence of the constitution of parents on the health of their children, and will learn from it how to direct the management of infant life. From the second work he will learn how he may carry out the details of practical hygiene. This work of Dr. Parkes brings together an amount of material which shows the extent to which exactness and certainty have been imparted to the Science of Medicine. It is a work which marks a new era in Practical Medicine.

From the nature of the CONSTITUTIONAL DISEASES (fully expressed at pages 391 to 394, vol. i., and described in the preceding pages), it is to be noticed that there are three periods in the history of these diseases necessary to be recognized in the pathology and treatment of them.

The first period may be described as *the period of their constitutional development.* During this period the Physician is rarely if ever consulted, unless a constitutional tendency to the disease is suspected, obvious, or perhaps hereditary.

The second period may be described as the period during which the *constitutional disease is fully expressed* by the phenomena and symptoms already described as peculiar to each of them.

The third period in the history of these diseases may be described as a *series of intervals*, during which the health seems to be improved between the febrile paroxysms, or fully expressed conditions of ill-health.

It is for the cure of the fully expressed disease that the Physician is generally consulted—a period when he knows he can do the least good.

But when the public are aware of the extent to which health may be preserved, and constitutional diseases averted and mitigated by judicious management and treatment during the first and last periods here noticed, the Physician will be more frequently consulted as to how the health is to be so preserved and improved as to ward off CONSTITUTIONAL DISEASES.

Whenever the Physician has to treat any of these constitutional affections in their fully expressed condition, if he is successful in subduing the symptoms for the time, by means of the treatment already mentioned under each of them, *the interval of comparative freedom from the paroxysmal expressions of disease is a time most precious, which ought not to be wasted, but which ought to be taken advantage of in preserving and improving the general health.* To effect this end there are three things to be considered in the management of the system during the intervals of comparative freedom from constitutional affections. These are—(1.) *Diet;* (2.) *The use of Water;* (3.) *The use of Wines and other alcoholic beverages.*

1. *With regard to diet,* one of the highest problems in physiology, as Dr. Parkes clearly enunciates, is so to regulate the supply of the *nitrogenous substances*—the *albuminates*—that the *digestive power* of the stomach and intestines may be increased, together with the *formative power* in the nitrogenous tissues, and the *eliminating powers* in the after-stages of assimilation (l. c., p. 132). All these three parts of the process must be duly balanced, otherwise health is destroyed. Half-digested food in the stomach or intestines produces irritation. It undergoes chemical changes in the alimentary canal, and quantities of gas are given off. Dyspepsia or constipation may be produced, or an irritation causing a diarrhœa which fails to empty the bowels. The fæces then contain a large amount of nitrogen, especially after eating vegetable albuminates. The tissues are unable to appropriate the excess which remains in the blood. Urea and carbonic acid, which ought to be eliminated in normal abundance, fail to be provided for from the imperfectly oxidized products of disintegration, and irritation of eliminating organs is set up. It is highly probable that gouty affections arise in this way, and partly from the use of liquids which delay metamorphosis. A great excess of albuminates without other food produces marked febrile symptoms, malaise, and diarrhœa; and ultimately albumen may appear in the urine, or extensive irritation of the skin may supervene. To increase the adaptation of the albuminates, fats and salts must be added to the diet; and the supply of oxygen must be increased by exercise, or the supply of starchy foods which appropriate oxygen must be diminished. Lessening the supply of the albuminates effects a decline or loss—first, of the muscular system; and, at a later period, of the nervous system and mental powers. Such decline or loss may be delayed by increasing the supply of fats and starches, which, by absorbing oxygen, limit disintegration; and by perfect rest the loss of flesh may be still further delayed.

In the management of the system, therefore, the albuminous tissues can be, to a certain extent, brought under the control of the physician by a judicious adaptation of diet and exercise alone, without the employment of drugs. Drugs, however, when judiciously employed, are important aids at two ends of the intestinal canal—namely, to aid primary digestion and to assist elimination.

Fatty aliments are essential to the formation of muscular, and especially also of nervous tissues. With the nitrogenous substances or *albuminates*, they are essential to the production of mechanical force. Animal fats appear easier of absorption than the vegetable fats; and the relative proportion of *fat* to *albuminates* ought to be as 1 or 1¼ to 2.

Starchy and *sugary food* saves the *albuminates* and *fats* from too rapid disintegration, so that, by a judicious employment of them in a dietary, the elimination, and perhaps the formation of the albuminates and fatty tissues can be so modified that the administration or withholding of starches and sugars as articles of food must enter largely into the management of the system, according to the circumstances of the case and the nature of the constitutional disease.

The production of *lactic acid* in the system seems probably connected with the metamorphosis of starch. To the constant introduction of an excess of alkali in the food, and the no less constant production of acid during the digestion of foods (especially starches, sugars, and fats), associated with the effects of respiration, are to be ascribed the singular alternation of acid and alkaline fluids in the body. The relative amount of *starch foods* to the *nitrogenous* or *albuminate* substances ought to be as 2½ or 3½ to 1. Excess of starches, sugars, and fat produce excess of fat; sometimes also acidity and flatulence; and the urine may become saccharine under excess of starch as an article of diet. *Salts* and *water* are essential articles of diet. *Lime*, in the form of *phosphate*, is absent from no tissue; and when tissues degenerate and morbid growths are overgrown, or begin to decay, or when cells grow rapidly, as in *enchondroma*, lime is present in large amount. Both *lime* and *magnesia* are essential to the growth and repair of bone; and the judicious withholding or administration of *lime* and *magnesia* in food is an important element in the constitutional management of diseases where the bones are affected, and in the repair of fractures. *Potash* and *soda*, in the form of *chlorides* and *phosphates*, are not less important constituents of diet. They form part of almost all the tissues; and, being especially concerned in the molecular currents amongst the elements of texture, seem to be less fixed than the magnesian and lime salts. The potash seems especially associated with the formed elements of texture, such as the blood-cells and the muscular fibres; while the soda salts are more largely concerned with the fluids which encompass these elements. These two alkalies (*potash* and *soda*) have been seen to be most important remedial agents in many diseases. *Chlorine, chloride of sodium, phosphoric acid, sulphur*, and *iron* must be furnished in all food, either separately or combined with the foods already noticed.

The salts which form carbonates in the system, such as *lactates, tartrates, citrates*, and *acetates*, exist chiefly in fresh vegetables; and although their nutritive power otherwise is small, it is absolutely necessary that they be supplied in the food. Scorbutic states inevitably ensue if they are withheld.

These are the main points to be inquired into and provided for in adjusting the diet best adapted for the managemant of constitutional diseases. Every intelligent patient may aid his physician, however, thus far—namely, that if he consider the subject *bonâ fide* for himself, he will become the best judge of the exact diet which suits him. Dr. Parkes observes that probably 30 *per cent.* of the persons who consult physicians owe their diseases in some way to food, and in many cases they are perfectly aware themselves of their error or bad habit; yet, with the singular inconsistence of human nature, either conceal it from the man to whom they are professing perfect openness, or manage to blind themselves to its existence (l. c., p. 461). Individual hygienic management must be inculcated upon the patient; while, by regulating diet on the principles laid down by Dr. Parkes in Chapter V. of his work *On Practical Hygiene,* the physician will find he can exercise a great power in the control and limitation of constitutional disease. Most valuable information respecting the adjustment of the dietary will be found in Dr. Edward Smith's *Practical Dietary;* and in *A Manual of Diet and Regimen for Physician and Patient,* by Dr. Horace Dobell.

2. *With regard to the use of Water,* its influence as an agent in the management of certain conditions of constitutional ill-health is apt to be too much neglected, or not sufficiently appreciated. Ever since the powerful agency of water, under the popular name of the "*water cure,*" has been so much bequacked, the medical profession have almost allowed themselves to be seized with a kind of *hydrophobia.* Nevertheless, there can be no doubt that water has a most powerful influence for good or evil in the treatment of constitutional diseases. Under its influence as a fluid of daily consumption considerable chemical changes are promoted in the body. In the intervals between the extreme expressions of constitutional diseases, such as *gout, rheumatism, asthma,* and the like, it is a useful and most efficient remedial agent, not only as a diuretic, but also as a local application, especially in obviating the congestions of the viscera associated with these constitutional affections (SIMON). "To accelerate the defective textural metamorphosis or waste is very often the practical result to be aimed at, and such means exist in the simplest and most manageable form in many of the appliances of the so-called '*water cure.*' With increased water-drinking there is increased discharge of solids by the urine. External appliances, such as the cold *sitz bath,* lasting a quarter of an hour, increase for a time the elimination of urea and uric acids. The protracted sudorific packings produce a still more considerable waste of tissue. Generally, therefore, in cases where the use of water is suitable, and conducted under competent medical advice and direction, the healthy nutrition of the body is promoted, in proportion as refuse materials are actively disengaged" (SIMON, in Holme's *Surgery,* vol. i., p. 118).

3. *The properties of Wines, and of alcoholic beverages* generally, offer a wide and important field for the careful study of the physician. Although so extensively used as beverages, with our daily food, and so generally recognized as valuable agents in the treatment of disease, there is not only great diversity of opinion as to their precise effects, but as yet very little is certainly known of the action of alcohol when adminis-

tered in the forms of *wine, beer,* or *spirits.* None of the general statements so frequently met with as to the composition or effects of any particular class of beverages can be relied on as a guide to the physician in prescribing; and much error seems to prevail on the subject, not only in the popular mind, but amongst medical men.

Alcohol is the most potent agent for good or evil in all of these beverages; and, therefore, its amount and its effects challenge attention in the first instance. A pint of beer (twenty ounces) may contain *one* or *two or more ounces of absolute alcohol,* or *less than a quarter of an ounce.* This alcohol may be associated in the beer with an amount of *free acid* varying from *fifteen* to *fifty grains,* and with an amount of *sugar* varying from *half an ounce* to *three* or *four times* that quantity. A glass of sherry (two ounces) may contain from a *quarter* to *half an ounce or more of absolute alcohol,* with *sugar* varying in quantity from *a mere trace to twenty* or *thirty grains,* associated with *a verg variable amount of free acid,* and with other ingredients. Even in brandy or whisky the amount of alcohol is widely different in different specimens.

It is impossible, therefore, for a physician to know what his patient is drinking, unless he is acquainted with the chief constituents and their amounts contained in the identical liquor which he may prescribe; and of course, before sound conclusions can be arrived at, the conditions under which these beverages are administered or taken must also be very precisely observed.

The physiological effects of alcohol have been investigated and inquired into with considerable care by Rudolf Masing, Böcker, and Mulder; by MM. Lallemand, Perrin, Duroy; by Percy, Ogston, Bence Jones, Carpenter, Spencer Thomson, Hammond, T. K. Chambers, Edward Smith, Anstie, and Parkes.

All the observations and inquiries on this subject tend to the conclusion that *alcohol* passes through the body unaltered in chemical constitution, and does not, so far as we know, leave any of its substance behind. At the same time, there is ample evidence to show that a very large amount may be retained in the fluids of the brain for an indefinite length of time (PERCY, AITKEN, and others, see *Alcoholism*). During that period of retention it exerts an influence for good or for evil; and although it may not be regarded as an "aliment" in the strict sense of that term, it undoubtedly aids the appropriation of aliment under some circumstances; and so far may be regarded as an "accessory to food" in comparative health, or as a "*medicine*" in disease. Under this aspect of the subject, Dr. T. K. Chambers, in his interesting clinical lectures (p. 570), lays down the following rules for the administration of alcohol:—

1. Alcohol may be given with advantage when the nervous system is exhausting itself, and when the tissues of the body generally are being exhausted by an activity in excess of the other bodily functions. It lessens the destructive metamorphosis which goes on; and chemical changes in the blood are partially arrested (HARLEY, quoted by PARKES).

2. It must be given, increased in amount, or left off, under the guidance of the appetite for food. As long as a person in ill-health takes and digests food better with alcohol than without, so long will

alcohol be of service to him. Beyond this general statement there is no evidence. In *very small quantities* it appears to aid digestion in the stomach; in larger amounts it checks it. A moderate use of beer or of the weaker wines (*i.e.,* of pure unbrandied well-fermented wines) may increase appetite and improve nutrition. On the other hand, the use of malt liquors (even when pure and good) is injurious to persons of sedentary habits, or unless much exercise be taken in the open air; but sound, well-fermented beer is the best of all dinner drinks *for persons of good digestion, who work hard in the open air* (DRUIT).

3. When the marked features of disease consist in the retention of effete matters which ought to be discharged, the use of alcohol must be totally abstained from, as for example in Bright's disease.

4. The daily allowance of alcohol ought to be divided into two or three doses only.

Under all circumstances its effects must be watched; for while it may sometimes be desirable to diminish the metamorphoses of tissue by its use, it must not be forgotten that large quantities of alcohol tend to cause an accumulation in the system of imperfectly oxidized bodies, such as *uric* and *oxalic acid.*

The general evidence also tends to show (but does not absolutely prove) that pure alcohol has its pernicious effects greatly lessened, and its good effects more powerfully developed, when highly diluted, and still more so by admixture with other substances, as the carbo-hydrates and salts contained in *beer* and *wines.* The use of strong wines (15 *to* 23 *per cent.*) undiluted should therefore be discouraged as much as possible; and if such an amount of alcohol is found necessary for the due preservation of the wine, and naturally results from the fermentation of the grape, the wine ought to be diluted with water when used as a beverage. On the other hand, light wines cannot be long exposed to the atmosphere without acetous fermentation commencing in them. Light wines with a small amount of alcohol for the use of invalids must therefore be preserved in bottles of such a size that the whole may be consumed in a day; for much mischief may arise if wines are used by invalids after acetous fermentation has commenced. Besides alcohol and ethers, wine contains several substances of great value as articles of diet,—namely, some albuminous substances, such as *sugar,* as well as other carbo-hydrates, and abundant salts. The vegetable salts are most valuable, and in this respect are highly anti-scorbutic (PARKES, l. c., p. 227).

For the purposes of the Physician, the prominence of certain substances in wines renders some more desirable than others, or more useful as "*medicines.*" These substances are—(1.) *alcohol,* (2.) *acids,* (3.) *sugar,* (4.) *solids* or *extracts.* Of these in their order:—

Alcohol, to a variable amount, ought to exist in wines *as a natural product of the fermentation of the grape;* and the conditions of its existence in the wine are then very different from those which obtain when alcohol (distilled even from wine) has been added to wine. It is highly desirable, therefore, to avoid adventitious alcohol; but it is impossible to prove that spirit has been added to a wine unless the amount is absurdly excessive. One vintage sometimes produces a wine with a considerably greater amount of *alcohol* than another of the same vineyard. For example, the samples tested in London (for duty) of *Chateau Lafite*, of vintage 1858, showed 16·5 *per cent.* of proof spirit; the same vineyard in 1859 showed 17·7; and in 1860 it gave 14·8;—the three years varying about 3 *per cent.* The samples of common *St. Emilion* in 1858 showed 16·5 *per cent.* of proof spirit; in 1859 the same vineyard showed 15·4 *per cent.;* and in 1860 it gave 16 *per cent.;*—thus not differing more in the three years than about 1 *per cent.*

The amount of the alcohol by measure may be most readily estimated by the vaporimeter of M. Geisler, of Bonn, which indicates the amount of the alcohol by the tension of vapour at a certain temperature, from the fluid containing the alcohol forcing up a column of mercury. Dr. Parkes gives also a very ready process by evaporation, and the use of a urinometer (*Practical Hygiene*, p. 220).

Having thus ascertained the *per centage* of alcohol in *wine, beer*, or *spirits*, it is easy to calculate by simple proportion the dose of alcohol administered. For example, a pint bottle of Claret (*thirteen* ounces) of the strength of *eleven per cent.* of alcohol, will be found to contain 1·43 ounces of absolute alcohol, thus:—

$$100 : 11 :: 13 = 1{\cdot}43.$$

It is the basis of the rule laid down by Dr. Parkes (l. c., p. 224), which says,—"*To tell how much pure alcohol is taken in any definite quantity of wine, measure the wine in ounces, and multiply it by the per centage of alcohol with a decimal point before it.*"

Acidity.—Free acid in wine is a necessary result of its fermentation. Its presence is likewise necessary for the evolution of the *bouquet*, for the agreeableness of the wine, and for its wholesomeness. It is, therefore, a popular error to denounce absolutely the existence of acidity in wine. The *relative amount of free acid* present in any particular wine is a very important point to determine. Much error prevails respecting the relative amount of acidity in different wines, and an excessive amount of free acid is very easily disguised by the relative sweetness of the wine. The estimation of the free acid may be measured by a solution of carbonate of soda, containing 530 grains in the 10,000 grain measure = 53 grammes in the litre; and the amount of acidity is represented by determining how many grains of crystallized oxalic or acetic acid a certain quantity of the soda solution will neutralize. The details of the process are as follow:—

(1.) Take 50 or 100 c. c. of recently opened wine; (2.) Add from a burette a standard solution of soda, in small portions at a time (say 5 c. c., or drop by drop); (3.) After every addition, test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper;—when the streaks cease to become red, the analysis is complete; (4.) Estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallized oxalic or acetic acid. Good wine contains a quantity of acid that is equivalent to from 300 to 450 grains of crystallized tartaric acid in a gallon. Wines with less than 300 grains of acid in a gallon are too flat to be drinkable with pleasure. Wines with more than 500 grains in a gallon are too acid to be pleasantly drinkable; and wines with more than 700 grains in a gallon are undrinkably sour (Druitt's *Report on Cheap Wines*, p. 178).

It is also very important to get at the *quality* of the acid,—to the extent, at least, of dividing the *volatile* from the *non-volatile* (and less digestible acids). A large amount of acid and acid salts may not be readily digested; and in many diseases the supply of acid to the system is a thing to be desired or prevented (see also PARKES, l. c., p. 219).

The necessary standard solutions and burette apparatus are supplied by Mr. Griffin, 119 Bunhill Row, who has prepared a set of apparatus which shows all that chemistry can teach of the quality of wine; by Messrs. Bulloch & Reynolds, Hanover Street, London; also, Harper & Sutton, Operative Chemists, Norwich.

Sugar is characteristic of all the *sweet* wines, and of many wines in their immature condition. Its amount tends to diminish with age, so that old wines of the *sweet* sort may be less pernicious, as regards sugar, than the new. *Sweet* wines, or wines which contain a large *per centage* of sugar, are to be avoided by those who are disposed to corpulence; and they are extremely injurious to those who are disposed to the formation of *oxalic acid*, or to the discharge of sugar in the urine. Its amount in any particular sample of wine is best determined by Soleil's *saccharimeter*. The principle on which the process depends is that the varieties of sugar possess the power of twisting the plane of polarization of a ray of polarized light which is transmitted through solutions containing these varieties of sugar. *Cane sugar* and *glucose* twist it to the right hand; *fruit sugar* twists it to the left hand. All gradation lists given in books as to the amount of sugar in wines are purely empirical, and apply only to the particular sample of wine examined. In stating the sweetness of Champagne, for example, nobody seems to notice that it is a manufactured article; and that the quantity of *syrup added to it* actually varies from *four* to *twenty per cent.* The Champagne known as "*still*" has no sugar. Some sorts of Madeira are also nearly free from sugar; and some sorts of Sherry are much sweeter than well-fermented Ports.

The co-relation of sweetness with acidity and with alcohol are most important points to be determined with reference to the easy digestion of wines; and hence they are all important for the physician to know.

The Amount of Solids may be learned (approximately) by determining the specific gravity after the alcohol is driven off. A low specific gravity shows that alcohol has been added, or that the solids are in small amount (PARKES, l. c., p. 226).

The following table gives a standard for the determination of the solids (PARKES, l. c., p. 218):—

Specific Gravity after loss of Alcohol.	Per Cent. of Extract.
1004,	1
1008,	2
1012,	3
1016,	4
1020,	5
1024,	6
1028·1,	7
1032·2,	8
1036·3,	9
1040·4,	10

It is much to be regretted, especially for the sake of invalids, that so few wine merchants are acquainted, or care to be acquainted, with the chemical constituents of their different wines. Medical men should, therefore, deal only with those wine merchants who will tell them the amount at least of the several constituents in their wines that have been referred to—namely, of alcohol, acids, sugar, solids—and who will guarantee the wine they supply as being in accordance with the sample of which they give the analysis. The sale room of every wine merchant ought to be provided with the means and appliances here indicated for ascertaining the exact amount of the substances contained in the samples of wine they offer for sale. The *amount of alcohol, of free acid, of sugar, and of solids* should be recorded also of every wine on importation, so that the wine merchant may be able to watch the metamorphoses, in respect of these constituents, effected on them by lapse of time. Wine merchants could thus materially assist the efforts of medical men in the cure of disease, and physicians could then prescribe with some knowledge of what they were prescribing; and to that extent at least the welfare of invalids and the sick would be better cared for.

But although by ascertaining these facts a physician may prescribe with better knowledge of what he is prescribing, and to that extent at least be better able to care for his patients, it must not be concluded that chemistry can detect every form of adulteration in alcoholic beverages. As Dr. Druitt justly observes,—"The only real test for wine is the empirical one. It is impossible to say that such a wine must be good in such and such cases, because it contains certain ingredients. . . . The stomach is the real test-tube for wine; and if that quarrels with it, no certificate of Liebig and no analysis are worth a rush" (*Report on Cheap Wines*, p. 6). With all possible aids, the one safeguard against fraud is to deal only with wine merchants and brewers of established character and reputation, who will be above attempting to dispose of unsound or adulterated liquors; and to avoid being tempted by the low prices of amateur dealers in wine, or of advertising companies and others, obviously more anxious to sell than to sell only the purest and the best.

The principal analyst of the Inland Revenue Department reports that he found illicit ingredients in no less than twenty out of twenty-six samples of beer tested in 1865; and he believes that, owing to the difficulty of detection, the practice of adulterating beer with poisonous ingredients is much more prevalent than might be inferred from the small number of discoveries made. These samples were all from the stores of licensed brewers; and although, doubtless, further adulterations are practised by the retailers, they are probably for the most part with less noxious materials, and chiefly with the object of increasing bulk—known as "*stretching.*" As to wines, the following advertisment, frequently appearing in the *Times*, shows that there is a trade openly carried on in this country in the treatment or *cure* of bad or spurious wines:—

"Wine merchants having wines turned acid may have them returned to their original wholesome perfection in two or three days, by applying to ——," &c., &c.

It is well known that the same practices prevail abroad, so that "pure as imported" is now no guarantee for purity of production. Hambro' wine has been shipped from London to Cadiz and back—an operation which raises the price of the stuff 100 *per cent.* The custom-house officers in 1865 stopped a large quantity of stuff imported as "Sherry," which had not a drop of grape-juice in its composition; and it cannot be doubted that the increased consumption and increased competition for low prices have greatly added to the temptations to adulterate. Such liquors, for any medicinal purposes, must be worse than useless; and whenever Wine, Brandy, or Beer is prescribed medicinally, they ought to be obtained from wine merchants or brewers of the highest respectability. Wine merchants are generally quite willing to sell in small quantities either wine or spirits—a recent change in licensing laws enabling them to do so, and so making it no longer necessary to send to the public-house when only a single bottle is wanted.

The blindly empirical and routine mode in which alcoholic beverages are generally prescribed, in absolute ignorance of their constitution and genuineness, and the importance of them in the treatment of disease or of ill-health, renders it advisable in a text-book to insist fully on these topics, believing that the physician cannot cope successfully with diseases, and especially with CONSTITUTIONAL DISEASES and the ill-health with which they are associated, unless he learns judiciously to use the immense power at his disposal in the influence of *diet, water, and alcoholic beverages,* as agents in the management of the system during the intervals between the paroxysms of these diseases. For much valuable information on wines, given in a very pleasing form, the reader is referred to *Report on the Cheap Wines from France, Italy, Austria, Greece, and Hungary; their Quality, Wholesomeness, Price, and their Use in Diet and Medicine,* by Robert Druitt, Esq., F.R.C.P., 1865.

CHAPTER XIV.

GENERAL NATURE OF LOCAL DISEASES.

THIS class of diseases is intended to comprehend all those which affect the structure of special organs or particular parts of the body, and which lead to marked disturbance of their functions.

Local affections are often accompanied by constitutional symptoms. Such symptoms, when they succeed to and depend upon the existence of the local lesion, are to be regarded as secondary to that lesion.

Many diseases, however, where the lesions are strictly localized and defined are really the results of *constitutional* or *specific* disease; and many lesions have been already described under the several GENERAL diseases that have been considered in the previous pages of this work. The *local affection* in such cases is often so striking and important that it has especially challenged attention, while the *constitutional state* from which it may have sprung is as yet concealed and unknown. The lesion here is secondary to the general affection. Not a few cutaneous eruptions are of this nature; so also are some forms of dropsy.

The diseases about to be considered are essentially characterized by more or less defined local lesions.

These lesions, with reference to each organ, may be most fitly considered in the following order or arrangement of their anatomical forms:—

(a.) *Catarrh,* or increased flow of secreted fluid.

(b.) *Inflammation of the suppurative, ulcerative, plastic, pyæmic, rheumatic, gouty, syphilitic, scrofulous,* or *gonorrhœal.*

(c.) *Gangrene.*

(d.) *Passive congestion.*

(e.) *Extravasation of blood* or hæmorrhage.

(f.) *Dropsy.*

(g.) *Fibrous deposits.*

(h.) *Altered dimensions, as dilatations, contractions, hypertrophy, atrophy.*

(i.) *Degenerations, amyloid or lardaceous, fatty, atheromatous, pigmental, calcareous, fibroid.*

(k.) *Syphilitic.*

Cancer.

Non-malignant tumors.

Cyst.

Scrofula, with or without tubercle.

Parasitic lesions.

Calculus and concretions.

Malformations.

Injuries.

Functional diseases.

The elementary nature of these lesions has been already considered in Part I., under topics relative to Pathology, pp. 23 to 233.

CHAPTER XV.

DISEASES OF THE NERVOUS SYSTEM.

SECTION I.—INTRODUCTION TO PATHOLOGY OF DISEASES OF THE NERVOUS SYSTEM.

It is with nerve-texture, as composing the essential parts of the nervous system contained within the cranium, and distributed as nerves throughout the body, that we have to deal in describing the local diseases of this section.

The interest which attaches itself to the study of the nervous system cannot be surpassed either in a physiological or pathological point of view. The truth and force of this statement will be more especially apparent when it is remembered that the nerves are the channels of Sensation; that nervous textures compose the organs which conduct the influence of the Will to the muscles, before motion can take place; and that while the textures which constitute the *nervous centres* associate Sensation on the one hand, they at the same time balance and co-ordinate the motions of the body on the other. And that, lastly, being the seat of the various mental processes through which *Sensation, Volition, Memory, Judgment,* and *all mental acts* are expressed, the pathological relations of the nervous texture contained within the cranium, or distributed throughout the body, are perhaps the highest and most important, in a scientific, philanthropic, and sanitary point of view, to which the mind of the physician can be directed.

The student is therefore called upon to give the DISEASES OF THE NERVOUS SYSTEM generally a large share of his study, because they are confessedly difficult.

The varied phenomena connected with the morbid conditions of the nervous system must be examined from the following points of view, namely—(1.) The purely anatomical structure of the brain and nerves; (2.) The chemical composition and properties of the nervous substance; (3.) The physiological relations of the several parts; (4.) The morbid and pathological relations. Each of these methods of study and investigation will mutually illustrate one another; and it is only from a consideration of all of them conjointly that we can arrive at the natural history of a case, and so act for the best in diagnosis, prognosis, and treatment. Every student knows how very many physiological doctrines regarding the brain and nerves receive elucidation from accurately determined anatomical information; and so such knowledge tends to explain various points in the pathology of cerebral diseases. He need only be here reminded of the phenomena explained by the decussation of the *pyramids* in the *medulla oblongata;* how the continuity of the fibres of the spinal cord upwards to the *cephalic centres* explains various secondary lesions of the brain as a consequence of lesions in the spinal cord altered by paralysis (Drs. Brown-Séquard, Turck, and Waller): and, lastly, the interesting observations made by Drs. Walshe and H. Bence Jones regarding the excretion of sulphates and phosphates by the urine in *acute chorea, delirium tremens,* and *inflammation of the brain* itself. These observations show, to some extent, how accurate chemical and anatomical investigations may become valuable in the elucidation of morbid phenomena occurring in living bodies.

Anatomical Constituents of the Brain and Nerves.—The nervous texture may be simply considered as arranged into three great divisions:—

1. A large quantity of nervous matter collected into one mass, and contained in one cavity, the cerebro-spinal. This mass is called the *Brain and Spinal cord,* or *Encephalon,* or *Cerebro-spinal axis,* or *central part of the nervous system;* and is composed of the *cerebrum, cerebellum, sensori-motor ganglia,* and *spinal cord.*

2. A nervous texture, arranged in the form of long continuous cords or threads, mutually connected, and running in every direction throughout the body. These are simply called the *Nerves.*

3. An accumulation of peculiar nervous substance, in the form of small, round, and somewhat oval masses, called "*ganglia,*" variously connected with each other, and with the surrounding parts, and forming what is known as the *Sympathetic system.*

Chemical Composition of Brain and Nerve-Tissue.—The white or grey matter of the brain has been generally taken to represent pure nerve-substance. It consists of albumen, fatty matter, salts, and from $\frac{4}{5}$ths to $\frac{7}{8}$ths of water. The fatty constituents are remarkable, inasmuch as two of them, being acid compounds, contain a large amount of phosphorus, from 8 to 10 parts in 1,000 of the mass, or $\frac{1}{20}$th to $\frac{1}{30}$th of the whole solid matter. This is continually being metamorphosed during functional and morbid changes of the nerve-substance; and the amount of alkaline phosphates in the urine may be taken in some measure as an estimate of the amount of nerve-tissue disintegrated, the earthy phosphates being disregarded, inasmuch as they have been shown mainly to depend on the quantity taken in the food. The phosphorus set free by disintegration of the nervous tissue unites, in the form of an acid, with the alkaline basis in the blood, and is thence separated by the kidneys and discharged with the urine.

Weight of the Brain and its Parts.—In appreciating morbid states of the brain and nervous textures after death, it is useful to remember that the absolute and specific weights of the brain range within certain limits, consistent with the healthy exercise of function. The following statements are made of standard numbers for reference and comparison illustrative of this point:—

Absolute Weight.—The absolute weight of the brain or encephalic mass varies in concurrence with variations of age, body-weight, and height of persons; and generally it may be stated as in the following table (page 125), compiled from the valuable records communicated to the Royal Society on the 28th February, 1861, by Dr. Robert Boyd, the Physician to the Somerset County Lunatic Asylum.

It is by accurate observations such as these that the gradual growth of every organ of the body is demonstrated to advance slowly and in concurrence with advancing age and increase of body-weight. It is now also a matter of experience, although too often overlooked, that the functions and organs of the body, and by no means rarely the brain and the Intellect, may be injured for life by pressing upon them too hardly and continuously in early years. Whatever theory we hold (as to the functions of the brain or Mind), "it is certain that the powers of the brain are only gradually developed; and that if forced into premature exercise, they are impaired by the effort. This is a maxim, indeed, of great import, applying to the condition and culture of every faculty or function of body and of Mind, and singularly so to the Memory, which forms in one sense the foundation of intellectual life. A regulated exercise *short of fatigue* is improving to it (and to all faculties or functions), but we are bound to refrain from goading it by constant and laborious efforts in early life, and before the instrument is strengthened to its work, or it decays under our hands" (Sir Henry Holland's *Mental Pathology*).

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT AND THE WEIGHT OF CEREBRAL ORGANS AS TO AGE AND HEIGHT.

Age.	Sex.	Body-Weight.		Height of Body.	Weight of Cerebrum.	Weight of Cerebellum.	Weight of Pons and Medulla.	Weight of Encephalon.
Years.		Lbs.	Oz.	Inches.	Ounces.	Ounces.	Ounces.	Ounces.
1 to 2	Male,	14	6	28·5	29·21	3·54	·5	33·25
	Female,	13	2	27·7	26·19	3·15	·46	29·8
2 to 4	Male,	20	0	31·6	34·03	4·02	·66	38·71
	Female,	18	7·5	31·6	30·77	3·7	·5	34·97
4 to 7	Male,	25	8	37·5	35·44	4·17	·62	40·23
	Female,	24	9	37·0	35·04	4·19	·68	40·11
7 to 14	Male,	42	6	47·0	40·36	4·84	·76	45·96
	Female,	38	6	45·0	35·86	4·27	·65	40·78
14 to 20	Male,	68	0	60·5	41·77	5·32	1·0	48·54
	Female,	63	14	57·7	38·88	4·65	·85	43·94
20 to 30	Male,	92	14·5	66·75	41·98	5·19	·93	47·9
	Female,	86	13	62·0	38·0	4·82	·88	43·7
30 to 40	Male,	98	3·5	66·5	42·06	5·15	·98	48·2
	Female,	87	0	62·0	37·92	4·74	·91	43·09
40 to 50	Male,	102	0	66·8	41·48	5·22	1·06	47·75
	Female,	84	9·5	62·0	37·12	4·69	·89	42·81
50 to 60	Male,	102	0·5	66·0	41·09	5·13	·98	47·44
	Female,	86	0	62·0	37·38	4·62	·86	43·12
60 to 70	Male,	103	13	65·7	40·21	4·98	·97	46·4
	Female,	86	14	61·5	37·13	4·68	·83	42·69
70 to 80	Male,	106	13	65·7	39·6	4·97	·94	45·5
	Female,	80	4	61·0	35·58	4·47	·88	41·27
80 to 90	Male,	99	0	66·7	39·62	4·79	·89	45·34
	Female,	79	0	60·0	34·47	4·47	·82	39·77

Bulk.—The bulk of the encephalon varies from 65 to 84 cubic inches.

Specific Gravity.—The result of recent observations in Germany, France, and Britain, shows that any considerable change in the specific gravity of the cerebral substance is incompatible with a healthy exercise of the nervous functions.

To Dr. John Charles Bucknill, Physician to the Devon County Lunatic Asylum, medical science is indebted for the first most extended account of the specific gravity of the cerebral substance, and its relation to disease, and more especially to *atrophy* and *paralysis*. The following are the general results of his observations as detailed in *The Lancet*, 25th December, 1852, and for the most part made upon patients labouring under different forms of mental disease:—

(1.) Average specific gravity of healthy brain, 1·036. (2.) In paralysis of a chronic character, complicated with insanity, the specific gravity ranged between 1·036 to 1·046. (3.) In some acute cases the specific gravity was as high as 1·052. (4.) In paralysis terminating by coma, 1·040. (5.) In paralysis terminating by syncope or asthenia, 1·036 to 1·039. (6.) In general terms, a higher specific gravity was found when life terminated by coma or asphyxia, than when it ended by syncope or asthenia.

In addition to these observations, an able and elaborate paper has since been published by Dr. Sankey, showing the relative specific gravity of the *grey* and *white matter* of the brain, and of so extensive a nature as to furnish very copious data for comparing morbid states with the standard of health. The following are the general results of his researches, as given in *The British and Foreign Medico-Chirurgical Review* for January, 1853, p. 257:—

(1.) Mean specific gravity of the grey substance of the brain in either sex, 1·034. (2.) In the earlier and later periods of life the specific gravity of the grey matter is below the mean. (3.) The cerebral substance acquires its greatest density in males between the ages of fifteen and thirty, and in females between the ages of twenty and thirty. (4.) The density diminishes with prolonged illness. (5.) It decreases with a lapse of time after death in the ratio of ·001 for every twenty-four hours. (6.) A density of ·006 above the average indicates the existence of the following conditions during life:—Acute cerebral symptoms, or chronic disease with no cerebral symptoms, or only slight delirium; also with conditions associated with hyperæmia. (7.) Mean specific gravity of white matter, 1.041.

Both sets of observations referred to above have been made upon the brain as a whole; and as the observations of Dr. Sankey show that no constant relation exists between the absolute weight of the brain and its specific gravity, it is necessary to examine the brain as we do its anatomy—namely, by comparative observations on its *central parts* or *gangli.a*

At the time Dr. Bucknill published his observations I was engaged in determining the specific gravity of the central parts of the brain, which are sometimes called the *central ganglia*, and which are now generally regarded as the parts more immediately related to the combined exercise of sensory and motor functions. These centres consist of the *corpora striata; thalami optici; tubercula quadrigemina; and the large* *mass of vesicular nervous matter associated with the convolutions of the hemispheres and the substance of the cerbellum.* While these parts are the immediate seats of the origins of the nerves, they may be looked upon as parts where some changes in connection with the functions of special nerves are constantly going on, of such a kind that a result is expressed through "Volition, Perception, or Emotion, or the balancing or coordinating of movements" (TODD).

These parts have a specific gravity as follows:—The central ganglia, 1·040 to 1·047; the cerebrum, 1.030 to 1·048; the cerebellum, 1·038 to 1·049.

The same kind of morbid states which modified the specific gravity of the brain-substance, as recorded by Drs. Bucknill and Sankey, also manifest their influence on the central parts. Thus death by *coma*, and especially in *typhus fever*, was indicated by an extremely high specific gravity; and while it was observed that a slight difference was common in most cases when similar parts on opposite sides were compared, it is presumed that further observation, extended in this direction, especially in cases of hemiplegia, may lead to important results. In one case of *choreic hemiplegia* which I had an opportunity of carefully investigating, the specific gravity of the *corpus striatum* and *optic thalamus* on the right side was found to be 1·025, while the specific gravity of the corresponding parts on the left side was observed to be 1·031 (*Glasgow Med. Journal*, No. I., 1853).

Pathological Relations of the Nervous Organs and Texture.—Our knowledge of these relations is necessarily imperfect, and for the following reasons:—(1.) The functions of the various parts which, connected together, constitute the encephalon, are not yet determined accurately. (2.) The inconstancy and irregularity of the functional disorders which accompany the morbid state of the nerve-substance render it difficult to interpret the value of the symptoms by which the *nervous diseases* are manifested. (3.) Some of the diseases of the brain and nervous system which are marked by the most violent symptoms during life, such as *epilepsy*, *chorea*, *tetanus*, and *hydrophobia*, leave after death no constant lesion capable of being detected with the unaided eye, or even by microscopic examination; while *tumors* and serious destruction of the nervous mass may exist during life without producing any severe or pathognomonic symptoms whatever. (4.) We have no means of applying physical diagnosis to the cranium, as we have to the chest, although it has been proposed by some (Drs. John Fisher and Whitney) to found diagnosis upon *Cerebral Auscultation.* For an abstract of the nature of the investigations "On the Auscultation of the Brain" the reader is referred to Wood's *Practice of Medicine*, vol. ii., p. 621, and also to a notice of Henning's "Inaugural Dissertation on the Sounds perceptible about the Head and at the upper portion of the Spinal Column in Children," in *Med.-Chir. Review* for 1857, p. 528.

The general principles on which the pathological relations of the *brain diseases* are determined, rest upon the anatomical, chemical, clinical, and physiological facts now accurately known. An accurate knowledge of anatomy is most essential in the study of disease of the nervous system.

In connection with the physiological view of the subject there are several cardinal facts which must be constantly kept in remembrance, and which may be shortly referred to here.

There are separate and distinct functions performed by the *grey* and *white* matter which enter into the structure of the nervous centres and organs. Reasoning from the general properties known to be possessed by cells in other structures, it is now a generally received doctrine, that the cells of the grey substance of the brain and nervous centres are the seat or source of that force which has received the name of the "*nervous force, nervous power*, or *nervous influence*," and which makes itself known by *sensation* and *motion*, as also by the various ways in which the *mental acts* are expressed. The *white nerve-fibres* are in connection with the *grey* or *cell-elements* of the nervous tissue, conducting from and to these centres the "influences" which are sent to, or which originate there and are thence sent forth.

The union of the nerve-tubes with the nerve-corpuscles is supposed to be connected with the transference of action from one nerve-fibre to another, as in *reflex action.*

It is likewise a remarkable fact that each nerve-fibre in a fasciculus acts quite independently from end to end,—quite isolated from the others in its vicinity; and thus at once we have the enunciation of three distinct sets of physiological phenomena associated with the diseases of the brain and nerves.

First,—Phenomena of Isolated Conduction.—Exalted or diminished action is presented by that nerve-fibre only which is affected by the irritating or depressing cause, and the adjoining fibre, though in ever such close approximation, is not implicated.

Second,—Phenomena of Sympathy or Irradiation of Sensations.—That irritation is propagated from a fibre originally excited to other centripetal nerves.

Third,—The Phenomena of Intelligence.—The brain furnishes the conditions necessary for the manifestation of the intellectual faculties, properly so called, such as the Emotions, Passions, Volition, and is at the same time essential to Sensation.

That the evolution of power or nerve-force immediately connected with Mind is dependent on or emanates from the hemispherical ganglia, is rendered probable by the following facts:—(1.) In the animal kingdom generally a correspondence is observed between the quantity of grey matter, the depth of the convolutions, and the sagacity of the animal. (2.) At birth the grey matter of the cerebrum is very defective, so much so that the convolutions are, as it were, in the first stage of formation, being only marked out by superficial fissures, confined to the surface of the brain; and as the grey substance increases, Intelligence becomes developed. (3.) The results of experiments have shown that, on slicing away the brain, the animal becomes more dull and stupid in proportion to the quantity of grey substance removed. (4.) Clinical observation points out that in those cases in which the disease has been found to commence at the circumference of the brain, and proceed towards the centre, the mental faculties are affected *first*—e.g., *meningitis* and the like; whereas in those diseases which commence at the central parts of the organ, and proceed towards the circumference, the mental faculties are affected last—e.g., *tumors* in the central white substance.

The white tubular matter in the form of the diverging fibres of the brain conduct influences, originating in the *hemispherical ganglia,* to the nerves of the head and trunk; while they also conduct, in an inverse manner, the impressions made on the peripheral parts up to the *cerebral convolutions.*

The spinal cord, by its connection with the brain, furnishes the conditions necessary for combined movements; and that its nervous force is also dependent upon its grey matter is rendered probable by the following facts:—

(1.) The universal connection of the grey matter with all motor nerves. (2.) Increased quantity of the grey matter in those portions of the spinal cord whence issue large nervous trunks. (3.) The collection of grey matter in comparatively large masses at the origin of such nerves in the lower animals as furnish peculiar organs requiring a large quantity of nerve-power, as in the *Torpedo, Gymnotus electricus,* and *Silurus.* (4.) Clinical observation shows that in cases where the *central portion* of the cord is affected previous to the external portion, the individual retains the sensibility and power of moving the limbs, but wants the power to stand or walk; whereas, when disease commences in the *meninges* of the cord, pain, twitching, convulsions, numbness or paralysis indicate lesion in the white conducting matter.

Independent endowment of nerves is shown by the fact that, whatever be the stimulus which calls their power into action, a uniform functional result is obtained; and hence it is inferred that the nerves are not altogether the mere conducting tubes of a stimulus from one place to another, but are in some respects the seats, or agents, or apparatus of power.

As far as we know, the brain alone furnishes conditions necessary for *Intelligence,* the spinal cord conditions essential to *Movement;* and together they furnish conditions connected with the *balancing and co-ordination of motor and sensific power.*

In dealing with the diseases of the NERVOUS SYSTEM, it is incumbent on the physician to ascertain, as correctly as possible, the *locality of the lesion, the nature of the affection,* and the *anatomical condition of the part affected.* Although it has been sometimes asserted that it is of little practical importance to discriminate accurately between diseases of one part of the brain or of its membranes and those of another, because the treatment may be the same for all, yet, for the sake of science—because "knowledge is power," and because the acquisition of such knowledge must eventually alleviate the sufferings and lessen the sorrows of humanity—the sooner such doctrines are ignored the better for the Science of Medicine; and, moreover, the majority of the medical profession are beginning to appreciate the principle that diagnosis should be carried as far as possible. To the advanced student, who would desire more minute information to guide him in the differential diagnosis of brain diseases than can be given in a text-book of medicine, he is recommended to study the work of Dr. J. Russell Reynolds, Professor of Clinical Medicine in University College, on the *Diagnosis of Diseases of the Brain, Spinal Cord, and Nerves*, as well as the writings of Dr. Sanders, of Edinburgh, Dr. H. Jackson, of the London Hospital, and Mr. Lockhart Clark, and the comprehensive manual of Tuke and Bucknill *On Insanity*. From these and other works the general remarks and descriptions of brain diseases have been mainly compiled.

SECTION II.—GUIDES TO THE DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.

I. **As to Locality or Site of Lesion.**—As yet we are able only in some cases of tumors of the encephalon to define their locality, as to whether they are in the cerebrum, cerebellum, or central ganglia.

With regard to the cerebrum, it may be determined in the majority of structural diseases which lateral half is affected, and in particular cases it may be predicated with strong probability that the lesion is situated in some one of the following sites:—(1.) The substance of the hemispheres (cortical or central); (2.) The ventricles,—hæmorrhage into these cavities, for example, may sometimes be distinguished from effusion into the substance of the hemisphere; (3.) The base of the brain; or (4.) Its superior surface. Inflammation of the brain-substance and of the meninges presents different symptoms when occurring in the two last-mentioned situations.

In the present state of science we are unable to localize exactly either the diseases of the *cerebellum* or of the *central ganglia* of the brain from symptoms during life.

Diseases of the *pia mater* and *arachnoid* may be discovered by the history and progress of a case from those of the *dura mater;* and sometimes it is possible to distinguish *meningitis* of the base from that of the convexity of the brain, chiefly from the influence which the diseased *meninges* exercise upon the functions of the parts beneath. Disease of the *dura mater*, for example, may often also be inferred from morbid conditions discoverable in the organs of *special sense*, or from disease in the *bones* of the *cranium*, or of the *integuments* or *scalp*.

With regard to diseases of the spinal cord, similar grounds for diagnosis exist; and the locality of the lesion may often be correctly referred to the *anatomical regions* of the cord, to *certain columns* of its *substance*, to the *white* or *grey nerve-substance* of which it is composed, or to the *coverings*. It is of importance in cases of diseases of the nervous trunks to know which set of the cranial nerves are affected; and in relation to the spinal *nerves* it is necessary to distinguish diseases of the *anterior* from those of the *posterior* roots, as well as the region of the cord from which the diseased roots proceed.

The grounds of diagnosis of the locality of nervous diseases generally may be summed up as follows:—

(A.) The *brain* is presumed to be the seat of lesion when several of the special senses are simultaneously affected; when *Perception, Ideation, Volition*, and *special Sensation*, are affected; when the muscles and general sensory nerves are implicated longitudinally and unilaterally (hemiplegia); when muscles situated so high as those of the face and tongue are involved, and the *orbicularis* of the eyelids does not share in their affection. In these rare cases of bilateral (or transverse) paralysis (*paraplegia*) resulting from some cerebral change, the symptoms at some period of the case have generally referred to the head (by their special character), so that, by a combination of the two classes of observations, the general diagnosis may almost universally be established.

(B.) The *spinal cord* is presumed to be the organ affected when the symptoms of motory and sensory character are distributed transversely or bilaterally, inducing paraplegia or transverse spasms; when the mental functions are unchanged. The precise locality may be estimated sometimes from anatomy of the spinal nerves. If the lesion or disease is high, speech, deglutition, or respiration may be impaired. There is often erection of the penis, and the retention or voluntary discharge of fæces or urine.

(C.) The *nerve-trunks* are presumed to be the seat of lesion when the symptoms are referable to an isolated muscle, or group of muscles, or to a small portion of the sensory surface. When paralysis is the symptom, the irritability of the muscles to electric stimulation is quickly lost, and the symptoms show no disposition to wander from the special localities affected (Reynolds).

The distinguishing characters of *meningeal* from *cerebral* diseases may be arranged in the following tabular form, for comparison and reference:—

CEREBRAL DISEASE.	MENINGEAL DISEASE.
1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, such as paralysis, anæsthesia, loss of memory.	1. It is not till some time after the detection of signs of disease that diminution or loss of nervous function takes place.
2. Cerebral disease is not commonly attended by high-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia, pain, or tenderness.	2. The subsequent diminution or loss of nervous function which succeeds the prolonged existence of "head symptoms" is generally preceded, in cases of meningeal disease, by extremely severe excitement or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.
3. Little vascular excitement attends cerebral disease, nor is there frequently any highly marked general disturbance.	3. In meningeal affections there is usually much local vascular excitement, with general disturbance.
4. Paralysis and Anæsthesia, losses of Volition, Ideation, Perception, and the like, characterize cerebral disease.	4. Spasms, convulsions, pain, and delirium, are the general features of meningeal disease.

In diagnosing the locality of diseases of the brain generally, it is necessary to distinguish, in the first instance, the *intrinsic diseases* of the nervous system; also local diseases from nervous complications of other diseases not of a local kind. It is necessary also to distinguish affections of the *brain, spinal cord,* and *nerves,* as much as possible from each other; and, lastly, to separate diseases of the *meninges* from *cerebral lesions.*

It is chiefly by the history of the case that nervous symptoms peculiar to the *Specific* or *Constitutional* class of diseases are to be distinguished. It is also generally worthy of notice that symptoms referable to altered nervous functions are the earliest indications of intrinsic or local diseases of the organs of the nervous system; and that when general disease exists of a *Specific* or *Constitutional* kind, the nervous symptoms are secondary in relation to the time of their appearance, compared with the earliest manifestation of symptoms of ill-health.

The diagnostic value of *vomiting*, as a symptom of cerebral disease, is one which must be thoroughly appreciated. Regarding this symptom, Dr. Reynolds makes the following remarks:—"The intimate sympathy subsisting between the stomach and the head is a matter of daily observation. Headache from gastric disturbance is as common as vomiting from cerebral derangement. In children especially, the existence of obstinate vomiting is indicative of head rather than of stomach disease. A consideration of the following points (for comparison tabulated) may lead to the discrimination of the pathological significance of this symptom:—

GASTRIC OR HEPATIC VOMITING.	CEREBRAL VOMITING.
1. There is nausea, which is relieved, at all events temporarily, by the discharge.	1. Little or no nausea, and the vomiting continues, in spite of the complete discharge of its contents by the stomach, so soon as anything (liquid or solid) is introduced.
2. The tongue is foul, the conjunctivæ often yellowish, and the headache secondary in respect of time.	2. The tongue may be clean, the conjunctivæ colourless or injected, and the headache primary.
3. Griping pain in the abdomen, diarrhœa, and disordered evacuations frequently attend the *gastric* or *hepatic vomiting*.	3. Obstinate constipation generally attends *cerebral* vomiting.
4. Retching and increased salivation attend *gastric* or *hepatic vomiting*.	4. In *cerebral* vomiting the stomach is emptied almost without effort, and without any increase of the salivary secretion.

Thus, while vomiting may depend upon derangement in the gastro-intestinal canal, it may also depend upon increased sensory or reflex action, and is thus a valuable indication of cerebral disease."

II. **As to the Nature of the Affection.**—The nature of the intrinsic diseases of the nervous system may be shortly stated to be—

(A.) *Acute*, but *non-febrile*, to distinguish them from the nervous symptoms which attend the febrile state of many of the diseases already noticed, as peculiar to the *Specific* and *Constitutional* classes. They are of such a kind as are marked by—(1.) Diminution or loss of functional activity (apoplectic and paralytic diseases); (2.) Increase or excess of action, such as of sensibility (*neuralgia*), of mobility (*convulsions, spasms*), ideation (*delirium*).

(B.) *Chronic* diseases, the character of chronicity depending not only on the time such diseases last, but also on the severity of their course.

Such chronic diseases are marked by—1. Excessive functional activity, as by *neuralgia, hallucination, chorea, hypochondriasis.* 2. Diminution or loss of functional activity—for example, *anæsthesia, paralysis, dementia, epilepsy.* 3. Combinations of these conditions, such as—(1.) Loss of mobility, with increased sensibility, as in *paralysis with pain.* (2.) Loss of mental, with increased motor activity, as in *coma* with spasms. (3.) Loss of sensibility, with increased mobility, as in *anæsthesia* with *reflex spasms.*

It is of great importance to recognize, as Dr. Brown-Séquard points out, the necessity of distinguishing between the symptoms of—(1.) Loss of function; and (2.) Irritation.

The most frequent cause of local paralysis or loss of function is an irritation in certain parts of the nervous centres, or in the trunk or periphery of nerves. Such circumstances may produce paralysis in very different parts in different cases, according to the particular fibres on which the irritation has acted. Thus there are now numerous cases, capable of clinical recognition, where paralysis of the upper and lower limbs, and of the face, as well as of contractions and rigidity, which are traceable to the influence of reflex action.

Symptoms of irritation may be arranged in two groups, according as the irritation acts—(*a.*) In the central parts, as in convalescence from such serious illnesses as *enteric fever*—overtaxing the mental powers, as in the case of weak children; or, (*b.*) In the peripheral parts of nerve-fibres, as in the neuralgic headache of dyspeptic children during the second dentition, when the symptoms frequently resemble those of the first stage of *tubercular meningitis.* Convulsions are not rarely due to the irritation of ascarides; and they also occur concomitantly with the second dentition, just as they occur in the first.

III. **As to the Anatomical Condition.**—Although some of the diseases of the nervous system are marked by excessive severity of symptoms during life, such as *tetanus*, *epilepsy*, *chorea*, *hysteria*, *neuralgia*, and the like, yet no characteristic or constant structural change can be detected in the nervous centres after death, either as a consequence or as a cause of such diseases; and although the belief is daily extending, that no morbid conditions of function can exist without some correspondent change in the organs, yet, so long as we have no means of appreciating such changes, the diseases now noticed must be regarded as "neuroses," "dynamic," or "functional" diseases.

But there are many other diseases of the nervous system which are attended by some physical changes in the organ, expressed by undoubted symtoms during life, and which leave evidence of their existence after death. For example, very different apparent vascularity is discoverable after death in the nervous masses and texture; and there are two very common classes of nervous symptoms during life which evidently depend upon the variable amount of blood in the brain. These symptoms are referable to *active arterial hyperœmia,* and are distinguished by the well-known phrase of "determination of blood to the head;" or they are referable to *passive venous hyperœmia,* commonly called "congestion." The question has been much discussed and experimented on as to whether more than a fixed proportion of blood can find its way into the brain. It was at one time supposed that the amount of blood contained in the closed cavity of the adult cranium could neither increase nor diminish, but was a constant quantity. There can be no doubt that all considerations of the subject lead to the conclusion that the quantity of blood within the cranium is extremely variable at different times and under different circumstances; and (as clearly stated by Dr. Sieveking) there is a peculiar property belonging to the white matter of the brain which has a strong bearing on the question—namely, the great elasticity of the medullary tissue, so much so that the resiliency afforded by this property is a sufficient counterpoise to the rigid structures which envelop the brain, and which do not, as is erroneously supposed by some, remove the intracranial contents entirely from the influence of atmospheric pressure. That pressure is exerted on a large surface, composed of columns or tubes of blood in innumerable small curved vessels which maintain, through the scalp and diploe of the skull, a direct communication with the blood within the cranium; and which is thus directly influenced by atmospheric pressure, while every anatomical arrangement of the parts within the cavity of the skull illustrates provisions made to counterbalance the varying interchange of bulk between the solid and fluid contents of that cavity. Among these may be noticed the ventricular and subarachnoid spaces, with their varying amount of contained serosity, as furnishing most prominent evidence of provision to accommodate the varying amount of fluids within the cranium.

Morbid states of the brain are also due to a poisoned state of the blood; although such a condition cannot be proved in all cases,—such, for example, as occurs in many of the general diseases already noticed, such as *Typhus*, *Variola*, *Rheumatism*, as well as from the action of *Alcohol*, *Opium*, and *Urea*,

IV. **As to Urea in the Blood and Brain.**—It is often of importance to determine whether urea circulating in the blood is contaminating the brain and impairing its functions.

The following instructions are given for the detection of urea in the brain after death, as well as for its discovery in the blood during life:—

"1. *In the Serum.*—Take the serum from a good-sized blister, and evoporate it to dryness over a water-bath. The residue is to be extracted with alcohol, which is a ready solvent of urea. This alcoholic extract is then to be evaporated to dryness, and a little water added, so as to make a syrupy mass, which should be plunged into a freezing mixture, and a few drops of pure nitric acid added to it. If urea be present, the characteristic crystals of nitrate of urea are soon found in the solution, and may be recognized either by the naked eye or by the microscope (fig. 4).

"2. *In the Substance of the Brain.*—Take about three-fourths of a whole brain, and cut it up into small pieces. Then treat it with four successive portions of boiling distilled water, each portion, consisting of about ten ounces, being allowed to stand six or eight hours before the next is added. The brain while thus macerating should be frequently stirred and mashed about with a glass rod. The washings, after being poured off, are to be mixed together and filtered. The filtered aqueous extract so obtained must be evaporated to dryness over a water-bath, and the dry residue, after being powdered, is to be again treated with four successive portions of boiling distilled water, observing the same precautions as before. The washings, after being mixed together as before, are to be filtered, and the clear solution evaporated to dryness over a water-bath; and after being thoroughly dried in a hot water oven, the residue obtained in this manner should be finely powdered, and the powder boiled in five successive portions of ether. The ethereal extract so obtained should be evaporated to dryness at a low temperature, and then treated with a little tepid water, and allowed to get quite cold. It is then to be filtered through paper previously moistened with water, and the clear solution again evaporated to dryness at a low temperature, when a small quantity of the extract procured in this way (which would contain all the urea present in the brain operated upon) is to be placed on a glass slide, treated with a drop of strong nitric acid, covered with a bit of thin glass, and allowed to stand a little time, and then examined under the microscope. A few crystals will then be seen, having all the characters of those of nitrate of urea" (*Clinical Lectures*, by Dr. Todd, 1859).

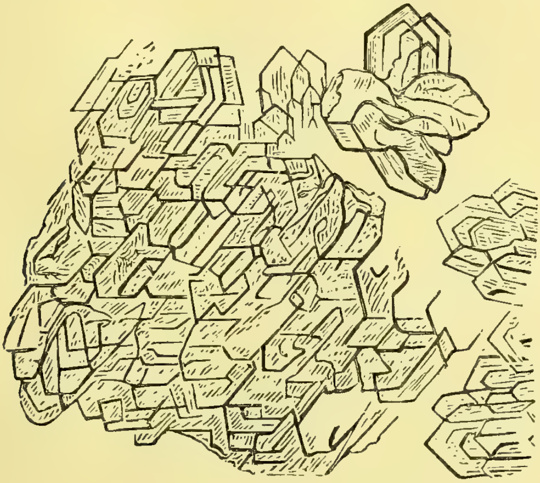

Fig. 4.*

V. **As to the Morbid Textural Changes of the Brain.**—These consist chiefly of inflammation and its *productive* consequences, or in softening, degeneration, or atrophy of the nerve-substance; and in heterologous products, such as result from *tuberculous* or *carcinomatous* infiltration. The nervous centres are also known to waste, harden, and lose weight as age advances, and the nerves participate in these changes. Hardening seems less constant than wasting and loss of weight; but it is a change very generally observed in old people; and its characters are thus described by Dr. Maclachlan:—"The cortical portion is thinner and darker than in adults, and the medullary portion is often void of its glistening white appearance, which gives place to a dull white or grey, and frequently to a pale drab colour in old age." The membranes of the brain are also thicker, more opaque, and more resisting in the aged. This is especially the case with the arachnoid, and is associated with enlargement of the Pacchionian glands or villi. The condensed dura mater acquires immense strength, and adheres firmly to the calvarium, especially along the margins of the longitudinal sinus.

When the brain shrinks, as in atrophy, there is a marked increase in

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[*] Nitrate of Urea (after BEALE). No. II. Urinary Deposits, Plate III.

the cephalo-rachidian fluid, according to the degree of atrophy. Instead of two or three ounces, there may be ten or twelve ounces; and the subarachnoid areolar tissue is then also frequently infiltrated with serum (MACLACHLAN). The ventricles contain more than the usual quantity of serosity, and the lateral ventricles especially are filled with limpid fluid. Flattening of the surface of the convolutions becomes apparent, while the sulci are widened and diminished in depth.

It is of importance to study every disease of the nervous system as presenting—(1.) Disease or changes in tissues; (2.) Damage of organs generally; (3.) Disorder of function (H. JACKSON).

The object has been, in most cases, to discover the seat of the disease, rather than its cause. A *softening*, a *sanguineous effusion*, and an *abscess* may produce exactly the same symptoms if seated in the same part of the brain; whereas when seated in different parts of the cerebral structures, affections of the same nature have little in common, so far as effects are concerned.

For the purposes of diagnosis it may be useful also to arrange in groups the diseases of the nervous system as follow:—(1.) Those attended by a febrile state, and where delirium is often a prominent symptom, such as *encephalitis* and *meningitis*. (2.) Those characterized by a group of symptoms commonly called "*apoplectic*," where loss of *consciousness* and of *voluntary* motion takes place as if by a "*sudden stroke*," such as in the various forms of apoplexy. (3.) Those diseases characterized by *exalted*, *perverted*, or *suspended functional activity;* as by *convulsions*, *spasms*, or derangement of motion—such as *chorea*, *hysteria*, *catalepsy*, *epilepsy*, *hydrocephala*, *sun-stroke*, *diseases of the Intellect.*

Many words in common use also require definition, *e. g.*:—

Convulsion.—The occurrence of universal involuntary muscular contraction, generally of paroxysmal or temporary duration.

Spasm.—Involuntary convulsive actions of less extent. Of these there are several varieties:—

(*a.*) *Clonic Spasm*—Consists in rapidly alternating contraction and relaxation, as in *subsultus tendinum.*

(*b.*) *Tonic Spasms* or *Spastic Contractions*—Consist in contractions having a certain duration, attended with rigidity or hardness of the muscles, as in *common cramps* and *tetanus.*

Epileptoid or *Epileptiform Attacks*—Imply a sudden loss of Perception and voluntary power, with more or less generally distributed spasmodic movement. The movements are quasi-tonic at first, then clonic, and appear to impede the respiratory process. The attack lasts from two to twenty minutes, followed by some exhaustion and sleep (REYNOLDS).

Coma—Denotes the loss of Perception and Volition; in other words, the loss of Consciousness, with the appearance of profound sleep, from which the patient may be partially roused.

VI. **As to Physical Conditions.**—The investigation of certain *physical conditions* materially helps the diagnosis of diseases of the nervous system. The physical conditions to be particularly inquired into are—

(A.) Perversion of the sense of touch and power of discrimination.

(B.) Perversion of muscular power.

(C.) Perversion of body-heat.

(D.) Perversion of the powers of expressing thought (*Aphasia*).

Of these in their order.

(A.) **Perversion of the Sense of Touch.**—The physical diagnosis of perversion of the sense of touch and power of discrimination is based on the fact, that "if two points of a hard substance touch an unmoved cutaneous surface, they can only be perceived separately when the distance between them exceeds a certain limit." Weber has shown that the minimum of distance thus established varies in different parts of the skin. By this experiment he constructed a scale of the sensibility possessed by the several tactile surfaces; and to do so, he used a pair of compasses, the points of which were armed with suitable pieces of cork, to find out the shortest distance at which the points are recognized as separate. A shorter distance than this will give rise to an indistinct impression of a long drawn point; and, finally, on approximating the points still more closely, the perception of the two points becomes completely the perception of one point only.

Dr. Sieveking devised an instrument of this kind suitable for such physical diagnosis of tactile sensibility, which he called the *Æsthesiometer* (fig. 5). He applied it in paralysis, to ascertain the amount and extent of sensational impairment in *paralysis*, as a means of diagnosis between actual *paralysis of sensation* and mere *subjective anæsthesia*, in which the tactile powers are unaltered, and as affording a means of determining whether a case of palsy was progressing for better or for worse (*Brit. and For. Med.-Chir. Review*, vol. xxi., p. 280. 1858).

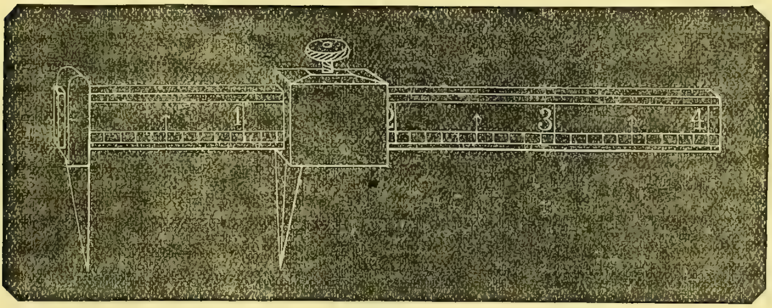

Fig. 5.

Instruments on a similar principle have been devised by Drs. Brown-Sèquard and John Ogle; but the best instrument is said to be that of Dr. Jaccoud, made by Colin & Robert, of Paris, about nine centimeters long, with a moveable scale in the form of an arc of a circle, the whole folding up so as to be easily carried in the pocket.

For those who treat nervous diseases this instrument is as indispensable as is the stethoscope to those who treat diseases of the chest. It not only measures the degree of tactile sensibility, and ascertains its perversions, but it aids in making out the precise site and nature of the disorder, particularly if the lesion be at the base of the brain, and in the spinal cord.

As it is necessary to know the normal distance limits, Weber's results may be stated as a standard for comparison,—premising that a delicate skin and an active mind admit of shorter distances than are here given (VALENTIN'S *Physiology*, p. 493):—

"(1.) The point of the tongue has a more delicate sense of touch than any other part of the body. Here the minimum distance is ·0433 inch. The skin of the middle of the back gives a minimum distance of 2·13 to 2·68 inches, and is the region where touch is dullest. Hence the extremes may differ from fifty to sixty-fold.

"(2.) Assuming that the average for the tongue is = 1, the distance for the terminal phalanx of the index finger is 1·2, and for that of each of the remaining fingers 1·8. At the thumb side of the first and second phalanges, it is 3·3; and on the dorsal surface of the last phalanx, 4·4.

"(3.) The red part of the lips gives 3·1, and the white 4·6. This difference is chiefly due to the unequal thickness of their coverings, and perhaps to their nervous relations. The remainder of the face has a still duller sense of touch. On the outer surface of the eyelids it is 7·9; on the skin of the cheeks, 9·4 to 10·9; and in the inferior frontal region, 12·4.

"(4.) The tactile sensibility of the foot is in every respect inferior to that of the hand. For example, the volar side of the terminal phalanx of the thumb gives 1·5; and that of the great toe, 6·7. The dorsal surface of the hand gives from 4·4 to 14·4; and that of the foot, 12·2 to 25·9.

"(5.) The extremities of the limbs, such as the hand and foot, have a more delicate sense of touch than their middle segments, such as the forearm and leg; while these again are more sensible than the segments connected with the trunk, such as the thigh and upper arm. The two latter belong to those parts which do not possess a high development of tactile capacity. The vicinity of the elbow and the knee-joint is more sensitive, being easily excited to pain.

"(6.) The face has a more accurate sense of touch than the crown of the head or the neck. The dorsal surface of the trunk is inferior to the abdominal in this respect.

"(7.) It would seem that, in the adult, these minimum distances alter very little by lapse of time. At least the author finds that his skin gives about the same numbers as it did eleven years ago.

"(8.) The friction of some parts of the skin gives rise to peculiar feelings of tickling, or to voluptuous sensations. But such parts do not necessarily rank high in the scale of tactile sensibility. Thus the axilla gives 26·9, and the foreskin 10·6, as the minimum of distance.

"(9.) The tactile sensibility is capable of being increased by habit to an extraordinary degree. In this way some blind persons are able to recognize different colours by inappreciable differences in their grain. The Bengalese spinning women can distinguish the threads of the cocoon with a tactile delicacy which is almost incredible. And persons devoid of arms may educate the sensibility of the toes, until it corresponds with that of the fingers of an ordinary individual.

"(10.) In judging of the delicacy of touch, we usually take the minimum distance at which two points can be recognized as the unit from which to start. This fact explains a peculiar illusion, to which attention was first drawn by Weber. When we draw the protected points of the compasses downwards from the cheek to the lips, it seems as if the distance between them gradually increased in consequence of our thus proceeding from a less sensible part to one which is more so."

The following are the rules laid down by Dr. Brown-Sèquard for the use of the *Æsthesiometer*:—

(1.) Fix the two points of the instrument at the distance which is normal to the part of the skin to be inquired into.

(2.) Cover the points of the instrument by pieces of cork, as the slightest prick producing pain interferes with the perception of tactile sensation.

(3.) The points must be applied simultaneously. If the patient feels only one point, when both points touch the skin at the same time, the two points must be gradually separated from each other, and re-applied to the part until both points are felt. This arrangement will indicate the anæsthesia.

If, on the other hand, the two points are each distinctly felt at the normal distance limit, they are to be gradually brought nearer to each other till one point only is felt. This arrangement indicates hyperæsthesia.

(4.) The patient ought not to be allowed *to see* whether one or both points of the instrument are applied, nor should he be made cognizant of the object of the experiment.

(5.) The instrument should first be applied upon a healthy portion of skin, so as to ascertain how far he can discriminate the sensation of one or of two points.

(6.) If the patient *knows* that both points are in contact he may *imagine* he feels both, no matter how short the distance may be between the points. In such cases one point only should be applied; and after he has asserted that he feels both, let him see that one only has been used: otherwise pre-conceived notions will destroy the value of the experiment.

(7.) In anæsthesia of considerable extent, the two points of the instrument may be applied one after the other after an interval of forty or fifty seconds, and yet give the sensation of only one point: this is due to the slowness of the transmission of the impression; and the æsthesiometer in this way may mark the rapidity of the transmission of tactile impressions.

(8.) When the degree of anæsthesia is great, the two points of the *æsthesiometer* are felt only as one, no matter how great the distance between them, provided that they are applied upon the same longitudinal line. Brown-Sèquard has known one point to be put upon the wrist and the other upon the forearm, and yet there was the sensation of one point only.

(9.) Cutaneous hyperæsthesia may be of such an amount that, however close the two points may be, both continue to be felt. In a case of *chronic spinal meningitis*, tactile sensibility was so much heightened that the patient felt both points applied to the thigh at the distance of one millimeter apart; whilst, in health, there should have been a space of from five to six millimeters between them for both to have been distinctly felt.

(10.) To avoid the mistake of finding either *anæsthesia*, or *hyperæsthesia*, when there is really neither, it should be borne in mind that *strychnia* increases tactile sensibility, whilst *belladonna* lessens it. If then it is necessary to find out the exact state of the cutaneous sensibility of any one part of the body in patients using either of these drugs, the state of tactile sensibility in the healthy parts should be first explored, and the actual normal type of the individual under the influence of these drugs be ascertained.

(11.) In certain cases when two points of an *æsthesiometer* are applied, there is a distinct sensation of three points; or two points may be felt when one only is applied. In the instances in which this perversion of tactile discrimination has been met with, there has always been inflammation or congestion, sometimes the result of an intra-cranial tumor at the base of the brain, and especially in one of the cerebral peduncles, or in one of the lateral halves of the annular protuberance. The sites of this phenomenon are the face, neck, and the hand, but particularly the face, for, in nine cases in which it was observed, in six it was limited to the face. When the distance between the two points of the instrument was two centimeters, two, or sometimes only one, point was felt; but at two and a half, or three, and even at four, centimeters, there was a distinct sensation of three points touching the skin (BROWN-SÉQUARD, *Archives de Physiologie*, t. Iʳ., 1868).

(B.) **Perversion of Muscular Power.**—On this point Dr. Clymer makes the following remarks in the American edition of this work:—

> "Muscle is the instrument, and not the producer, of force; the genetic factor of all muscular power being in the nervous system. Hence damage to the nervous centres is followed by derangement of muscular function—diminished or total loss of power being one of the most common consequences of cerebral and spinal disease.
>
> "To measure the strength of paralyzed muscles of the upper extremity, the *dynamometer* of Mathieu, a surgical cutler of Paris, will be found a useful instrument. The usual plan of making the patient grasp the physician's hand gives an imperfect indication, nor does it allow of any correct estimation of changes for better or for worse in the progress of the palsy. This simple instrument consists of an elliptical steel spring, to which is attached a semicircle of gilt brass, upon which a scale is marked. An indicator, with a small cog-wheel at one end, may be moved freely around the arc of the circle by a steel arm, upon one side of which cogs are cut, fitting into those of the indicator. The lower end of this arm touches the elliptical spring, when the indicator points to zero on the scale; a brass sheath on the under side of the scale keeps this arm in place, at the same time letting it move freely (Fig 6).

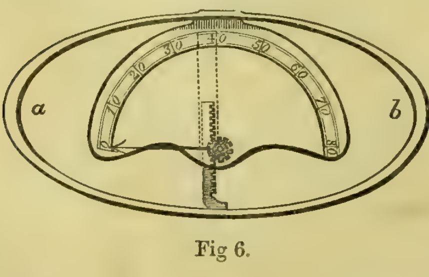

Fig 6.

> "When the dynamometer is taken into the hand and pressed, the two sides of the spring are approximated, and the steel-arm, the cogs being pushed by the lower side of the spring, turns the indicator. When pressure is taken off, the indicator does not return to 0, but remains at the point to which it has been carried by the muscular power of the patient.

"It may also be used to measure tractile force, for if two hooks with cords attached be fastened to the spring at the points *a* and *b*, traction on the cords will approximate the two sides of the ellipse, and thus push the steel-arm so as to move the indicator.

"Dr. W. A. Hammond has found it useful in determining the effect of electricity upon the irritability of paralyzed muscles, and thus enabling the physician to decide whether the proper time for its use has yet come, by observing whether the strength of the muscles is increased or diminished after the application of this agent."

(C.) **Perversion of Body-heat.**—In cases of hæmiplegia the temperature of the paralysed side is raised by half a degree to one degree. It is rarely normal and never lessened. The thermometric equilibrium is restored on the cure of the paralysis. The general body heat of hæmiplegic patients is not as a rule increased, and is usually below the normal—92·5° Fahr. During the last hours of life it may rise to 96° Fahr. Well marked paralytic atrophy is attended by lowering of temperature in the parts affected. Becquerel's discs, in connection with a galvanometer, are the best means to determine differences of body temperature in paralytic parts. They are composed of two thin plates of *copper*, soldered to a thin rod of *bismuth*, contained in a small tube of hard rubber, furnished with a handle. One disc is placed on the sound limb, and the other on the corresponding part of the paralysed limb. Both are in connection, by means of delicate silk-covered wires, with the poles of a galvanometer. If the temperature of both limbs be the same, the needle of the galvanometer remains at rest. If the heat of eith limb is raised, the needle is deflected to the north or south pole, according as one or other limb has the highest temperature. A difference of less than $\frac{1}{100}$ of a degree can thus be determined.

(D.) **Perversion of the Powers of Expressing Thought.**—This physical sign of cerebral disease has been elevated by some writers into a substantive affection under the name of *Aphasia.* It implies an inability to communicate thoughts, ideas, or names of things either by words or by writing, the Intellect being more or less perfect otherwise, and the organs of phonation free of paralysis. Every shade of deficiency or inability characteristic of this affection is to be met with, from temporary loss of memory of certain words, the names of common objects, and their misuse in conversation, up to persistent and complete inability to give expression by words, writing, signs, or gestures, to thoughts or ideas. "In some cases a mere inability to articulate, whilst the patient is able to write; in others a loss of memory of words until they are suggested; and in yet others a total forgetfulness of names—the patient giving everything a wrong appellation" (WILKS). The non-expression of the names of things, or the substitution of wrong ones, are among the most common forms of this inability. "Hand me the —— to stir the ——." "Poker" and "fire" are the words which are incapable of being spoken. "Give me my ——; you know," pointing to his head. "Your hat?" "Yes, my hat." Many other examples are given by Trousseau. The substitution of one word for another is another form of this affection—*e. g.*, "boots" for "head." Forgetting your own name, or the name of the person you go to visit, is another form. In another series of cases there is a sudden loss of the general faculty of speech, without paralysis or incapacity of thought. In a third set of cases there is unilateral palsy or unilateral spasms; and sometimes it occurs after an attack of apoplexy, or after an attack of severe typhus or typhoid fever.

In some cases, however, the thought or idea which leads to verbal expression, as Dr. Gairdner shows, is not absent, so that the faculty remains of communicating by the usual written signs.

The several theories of localization of the brain lesion believed to be present in cases of aphasia are not capable yet of positive demonstration. The disorder may exist in any of the typical forms, and yet no appreciable change be detected in any part of the cerebral substance or its blood-vessels. The following are the general results of *clinical* and *post-mortem* investigation:—(1.) In a large majority of cases of *aphasia* the peculiar inability of speech is associated with unilateral palsy, indicating disease of the *central ganglia*, or immediate surroundings of the *corpora striata*. (2.) In numerous cases, but not in all, there is disease of the third left frontal convolution of the cerebrum, or in the left anterior lobe, very near to this convolution. (3.) The whole of this convolution and the left anterior lobe of the brain may be wholly or partially destroyed, and yet no loss of speech has been observed. (4.) Aphasia may exist with apparent integrity of the left anterior lobe of the cerebrum, and indeed of the whole brain. (5.) In all the cases Dr. Wilks has seen there has been disease in the *central ganglia*—a necessary lesion for the production of *hemiplegia*—and loss of speech seems to accompany, and is usually found in connection with *right* hemiplegia, when a small spot of disease in the left *central ganglia* is sufficient. It may also occur from disease of the *pons Varolii* (*Guy's Hosp. Reports,* vol. xii., p. 174).

As a separate and distinct disease due to a specific lesion, *aphasia* cannot yet find a place in nosology. The phenomena are complex in the extreme; and the connecting link between the mental defect and loss of speech, with material change in the brain, is both clinically and anatomically still incomplete. Mental co-operation is defective in all the cases.

SECTION III.—DISEASES OF THE BRAIN AND ITS MEMBRANES.

ENCEPHALITIS.

LATIN EQ., *Encephalitis;* FRENCH EQ., *Encéphalite;* GERMAN EQ., *Gehirnentzündung*—Syn., *Encephalitis;* ITALIAN EQ., *Encefalitide.*

Definition.—*Inflammation of the brain, or of its membranes, the precise seat of the inflammation not being known before death.*

Pathology.—The most general fact that has been determined regarding this morbid state is that there are two very distinct forms of *cerebritis* to be recognized both anatomically and clinically. While dissection demonstrates the fact that inflammation may exist either in the brain alone or in the membranes alone, yet clinically it is found that *meningitis* generally complicates *cerebritis,* and thus the phenomena proper to each of these morbid states become combined; and especially in that form of the disease in which the lesion is extensive, commonly known as "*general cerebritis*," "*phrenitis*," or "*encephalitis*."

There is little interstitial exudation in cases of this disease, connective tissue is scanty, and the most important changes take place in the nerve-filaments and nerve-ganglionic cells. They swell up, in the first instance, and gradually break up into a detritus, with more or less abundance of the compound inflammatory corpuscle of Gluge.

Causes.—Inflammation of the substance of the brain is a rare disease, and may be caused by any of the morbid poisons associated with the *specific* or *constitutional* diseases, such as *pyæmia, glanders, typhus fever.*

Most cases result from mechanical injuries (*traumatic encephalitis*), not only from direct injuries affecting the brain after the skull has been penetrated; but numerous cases occur where the cranium has remained uninjured, and where there has only been an apparently slight contusion. In such cases concussion has been so great that the brain-substance has sustained a bruise, small vessels being ruptured, so that blood escapes. Such a lesion may not at first induce any signs of injury; but afterwards causes encephalitis. Other cases result from the excitement of insanity and uncontrolled moral feelings. In some instances encephalitis has followed the suppression of a cutaneous eruption, or the accidental inclusion of a nerve by a ligature applied to a blood-vessel (LALLEMAND, quoted by SIEVEKING). In others, caries of the bones of the cranium, and especially of the petrous portion of the temporal bone caused by *otitis.* Intemperance in alcoholic fluids also is a frequent cause of this, as well as of every other disease of the brain. The disease is sometimes brought about idiopathically, by exposure to the sun's rays in very hot summer days, especially in tropical climates. As a secondary disease, encephalitis is produced by cancer, tubercle, and by every other structural disease incidental to the brain textures.

Encephalitis occurs at every age: in childhood during the tendency to hydrocephalus; in adult age from the action of morbid poisons, and from mechanical and moral accidents; and in old age from the natural decay of the frame. Ramollissement of the brain has occurred at the following ages:—In a few cases from birth to fifteen; 39 cases occurred from fifteen to forty; 54 cases from forty to sixty-five; and 60 cases from sixty-five to eighty-seven. The frequency of this disease, therefore, increases with age. Men are supposed to suffer in a larger proportion than women, probably from their greater exposure to the exciting causes.

Morbid Anatomy.—The lesion is generally confined to certain parts; the entire brain is rarely if ever involved. The spots of inflammation vary from the size of a pea to that of a turkey's egg, and are usually of an irregular spherical shape. There may be only one spot or several. Usually there is only one; but if there are many, they are generally small and located in the cerebrum or cerebellum, most frequently in the grey substance, and very near the surface. The brain-substance affected is at first swollen by infiltration and hypertrophy of growth, then it softens, and becomes dotted over with red spots of extravasated blood. One section of the morbid part rises above the level of the cut surface. The softening resolves itself into a red-pulp, of a rusty-brown or yellow colour. It is so diffluent that it may be washed away by a light stream of water, leaving remains of nerve-tubes, blood corpuscles, compound granule cells, and fine granular detritus, in single particles, or in masses. Cyst-like cavities are sometimes the result of such changes, filled with a sero-gelatinous fluid. Sometimes a characteristic cicatrix takes the place of all, which is at first pale, red, and vascular, and which afterwards becomes white and callous.

The Symptoms and Cause of *encephalitis* have considerable latitude.

The symptoms are partly the result of destruction of the part of the brain affected, and partly the result of disturbance of the cerebral circulation, especially in the vicinity of the part, and therefore greatly influencing the functions of the brain.

In twelve severe cases related by M. Paroisse, and resulting from wounds, he states that the symptoms were nearly the same in all, and were as follow:—The men all stated that after the sabre wound they had felt no other inconvenience than local pain of the injured part, and that for two or three days afterwards they had all been able to march five or six leagues a day. On the third day, however, they had all been seized with fever, which terminated on the evening of the fourth day; but from that period they had suffered little, always preserved a good appetite, and prayed not to be put on a low diet. About the seventeenth day they became downcast and dejected, owing, probably, to many sloughs being detached, and much suppuration taking place about this time. On the following day they first lost the sense of smell, and then the senses of sight and taste. With these symptoms, but without fever or convulsions, they fell into an easy sleep; and, as if they had no further strength to contend with the disorder, they died between the nineteenth and twenty-second days from the infliction of the wound.

The symptoms which have been related by Paroisse agree entirely with those observed by John Hunter. He observed that trifling wounds of the membranes of the brain were often followed by severe and extensive inflammation of those tissues, and by very dangerous symptoms; but if the injury had been great, so as to have excised a portion not only of the arachnoid, but also of the substance of the brain, that the symptoms which followed were comparatively slight—a circumstance which he attributed to the brain in the latter case having room for expansion; and he therefore suggested the propriety of extensively incising the arachnoid and pia mater in all cases in which the dura mater alone had been wounded. Large portions of the brain may be destroyed without causing perceptible functional disturbance.

In idiopathic inflammation the case may be acute or chronic. The first stage of the acute form is generally of short duration, and in so far the attack may be said to be sudden; and if there have been preliminary symptoms of ill-health, the symptoms have generally been headache or long-continued derangement of the digestive organs.

The general symptoms of the first stage of encephalitis may be but feebly marked (unless *meningitis* predominates). There is usually, in the first instance, some heat of head and of surface generally. The face is pale, and the pulse low and irregular. The breathing is variable and sighing. There is slight feverish oppression, with headache and vomiting. *The nervous symptoms* are generally highly marked, and so are those of diminished functional activity in cases where the cerebral affection predominates as distinct from the meningeal. They consist of—

1. *Mental Phenomena.*—The patient is sullen, and his faculties become obscured. There is confusion of Thought rather than delirium, which occurs only in a mild form when the patient doses.

2. *Sensorial Phenomena.*—Unless meningitis is present, there is no hyperæsthesia; but there is deep-seated, violent, oppressive pain, frequently described as shooting from the centre to the vertex, the temples, eyes, or ears; and it is out of all proportion to the intensity of the febrile phenomena; and while the febrile phenomena subside (as they very generally do in the course of twelve or twenty-four hours) the local pain does not diminish.

The second stage may be ushered in by convulsions; or, after the first stage has lasted two or three days, the pulse usually becomes rapid and weak. The cerebral nervous functions become inactive; anæsthesia, paralysis, and coma commonly follow two or three convulsive paroxysms, and death follows after the first stage has lasted two or three days.

When the signs of meningitis co-exist and are unusually severe, the pain is deep-seated, and followed, after twelve or twenty hours, by convulsions and coma, and there is commonly *meningo-cerebritis* of considerable extent.

When the *cerebritis* is local or partial, the symptoms are those which are usually regarded as indicating "inflammatory softening," "red softening," as it is commonly called, or "acute ramollissement." Premonitory symptoms are common, and consist of—

1. *Mental Symptoms*—namely, some loss of intellectual vigour, failure of Memory, confusion of ideas, irritability of temper, and a consciousness of weakness—these symptoms becoming gradually persistent.

2. *Sensorial Symptoms,* such as pain of the head, dull, deep-seated, fixed, and protracted; tingling or numbness in one limb or side; imperfection of the special senses; dimness of sight; dulness of hearing.

3. *Motorial Symptoms,* as evinced in the loss of power of one limb or side—the most important of all the premonitory symptoms.

There is more or less fever in proportion to meningitis: there is heat of head, vomiting, and general malaise. The signs of meningitis, on the other hand, may be feebly marked; but there may be convulsions, followed by coma, partial paralysis, with rigidity returning more or less rapidly, and ending fatally in a day or two, or from two to three weeks. Thus there may be the symptoms of meningitis, or of cerebritis, of both together, or alternately; and, occurring with such premonitory symptoms as have been indicated, they leave little doubt of the existence of a partial cerebritis.

Diagnosis.—If symptoms of local brain disease commence after an injury, or if there be disease of the bones of the skull, such as the petrous bone, followed by "*head symptoms,*" as they are called, it is probable that encephalitis has commenced.

Treatment.—So soon as there are any circumstances which may lead to "head symptoms" after injury, it is well to commence treatment at once with local antiphlogistics, leeches, and evaporating lotions, such as *muriate* of *ammonia* in solution with *vinegar*, applied by cloths, which are to be frequently changed. The patient must be made to observe a rigidly abstemious regimen, and rest with the head elevated above the shoulders.

MENINGITIS.

LATIN EQ., *Meningitis;* FRENCH EQ., *Méningite;* GERMAN EQ., *Gehirnhautentzündung*—Syn., *Meningitis;* ITALIAN EQ., *Meningitide.*

Definition.—*Inflammation of the immediate coverings of the brain, the dura mater, pia mater, and arachnoid.*

Pathology and Morbid Anatomy.—The inflammation may be simple and idiopathic, or it may supervene as the result of some specific or constitutional disease, such as of some of the fevers, or of the *syphilitic, rheumatic, gouty, rachitic,* or *scrofulous* cachexiæ. It is also known to occur in convalescence from *pneumonia* and *pleurisy*, or from *acute exanthemata* and *erysipelas,* and from protracted diarrhœas, and especially also as intercurrent with Bright's disease. Three several forms (in addition to the general involvement of all the membranes) may be distinguished:—

(1.) Inflammation of the *dura mater.* This is almost invariably the result of injury or disease of the bones of the skull; and in such cases the injury or disease by which it is caused may be readily distinguished.

(2.) Inflammation of the *pia mater* and *arachnoid.* In diffuse *arachnitis* the arachnoid has seldom any considerable redness or congestion, but is thickened and opaque; while the transparent serum naturally contained in the cavity of the arachnoid being now scanty, or wholly wanting, it has neither the polish nor the moisture which is natural to it in health, so that the membrane appears brown and dry.

The principal phenomena of *arachnitis* are most obvious in the *pia mater*, so that the large vessels of that membrane are greatly congested; but still, according to Dr. Baillie, the redness is not so general nor so continuous as in inflammation of other serous membranes. Also, if the *pia mater* be attempted to be removed, it is easily torn, and separates from the brain in small fragments. The arachnoid covering the *dura mater* seldom participates in this affection. These are the appearances observed in diffuse *arachnitis*, supposing it to terminate by resolution. The inflammation, however, often proceeds, and may terminate by effusion of serum, lymph, or pus.

When serum is effused into the arachnoid cavity, the opacity of the arachnoid gives the serum a gelatiniform appearance; but when that membrane is divided, the serum is found to be fluid, and to diffuse itself in every direction; sometimes, however, it may be turbid, from an admixture of a small portion of free albumen. It is also not unusual to find a few points of lymph, of pus, or of blood, either at the exterior surface or within the arachnoid, effused along with the serum, and almost in juxtaposition with each other. The quantity of fluid effused is variable,—from two to three drachms to as many ounces. The effusion most commonly takes place at the upper surface of the hemisphere, but sometimes at the base, and sometimes into the ventricle of the brain. Lymph is the form which the exudation usually assumes ultimately if life continues, varying in density and thickness, dipping down between the convolutions; and commonly it is most abundant on the upper portions of the hemispheres. Foville says he has met with cases in which the effused lymph covered the whole of the brain, or nearly so, as far as the *tentorium.* The lymph was deposited in the arachnoid sac in two layers—one adherent to the cranial arachnoid, and the other to the cerebral arachnoid; while between them was a stratum of serum. He mentions having had six cases of this description under his care for several years, and that they were all in a state of the dullest stupidity, and apparently labouring under paralysis of every sense. They were like statues, with this difference, that, placed upright, they preserved their balance; if pushed, they walked; and if food was placed in their mouths, they swallowed it. Were these cases of "Paralysis of the insane," as now so-called? (Art. "Méningite," p. 406, *Dict. de Médicine.*) Lymph also may be effused into the arachnoid cavity, but it is generally in small quantity.

Suppurative inflammation may take place either into the subarachnoid spaces or into the arachnoid sac. It is, however, by no means a frequent occurrence. Rostan gives several cases of effusion of pus into the arachnoid cavity, likewise Morgagni, Cruveilhier, and Dr. Bright. Dr. Baillie states that he once saw pus effused into the cavity to such an amount as to cover the entire upper surface of the brain. Two cases are given by Dr. Hodgkin of cut wounds of the head in which pus was found in the arachnoid sac. Dr. Sieveking relates a remarkable instance of purulent exudation of the arachnoid, occurring in a young woman aged twenty-seven, under his care, at St. Mary's Hospital, in whom sudden and unexpected coma supervened, and terminated, after thirty-six hours, in death. She had previously suffered from *otorrhœa;* but on her admission she gave no signs of cephalic disease; nor was any direct connection traced after death between the affection of the ear and the *meningitis.*

The characters of *chronic arachnitis* are—a similar opacity and thickening of the membranes, together with granulations of a pearly colour and more especially along the longitudinal sinus, and also an augmentation of the *glandulæ Pacchioni.* Much serum is effused into the cavity; and the connective tissue by which the pia mater is attached to the brain acquires considerable strength, so that portions of the brain come away with the membranes. The membranes acquire considerable tenacity; and so marked are their characters in some cases, that Dr. Maclachlan notices an instance in which a portion of the arachnoid nearly an inch square was fully an inch thick, in appearance like the boiled white of an egg, and equal to the peritoneum in toughness. The surface of the brain is pale, and sometimes slightly atrophied. Much serum abounds in the arachnoid cavity, in proportion generally to the atrophy of the brain; and the ventricles contain serum (MACLACHLAN). Ossification of the pia mater is extremely rare; but it also becomes thickened, opaque, and injected. Dr. Baillie, however, mentions one case of ossified pia mater, on the authority of Soëmmering; and Dr. Hodgkin speaks of a specimen in the museum of Guy's Hospital.

In *acute arachnitis of the ventricles* the membrane becomes thickened, semi-transparent, pulpy, and sometimes sprinkled with minute spots of blood. It is rare to find lymph effused, but occasionally old adhesions are seen between the opposite surfaces of the ventricle. Pus has also been occasionally found in these cavities. With regard to the effusion occurring in the ventricles in "*meningitis*," it is correctly remarked by Dr. Sieveking that "it offers many relations different from the arachnoidal effusions occurring on the surface of the brain; nor is it quite intelligible why the secretion into the cavity of the ventricles should so rarely be found to communicate with the superficial arachnoidal space." The anatomical relation of these parts may in some measure explain the differences; for it is very doubtful that the arachnoid lines the ventricles of the brain—an arrangement which Kölliker considers impossible. Nevertheless, there is in meningitis generally an increase in the ordinary amount of the fluid in the ventricles, to the extent of several drachms, which may perhaps be regarded rather as a result of contiguous sympathetic effusion than as an extension by continuity of the inflammatory exudation.

A microscopic examination of the smaller vessels of the pia mater in *meningitis* shows that they are studded with the exudation-corpuscles described by Gluge and Bennett, and with oily-like vesicles; and these appearances are found both within and without the vessels (SIEVEKING).

(3.) *Tubercular meningitis*—Syn., *Acute hydrocephalus.* The *meningitis* which accompanies the cachexia associated with *tuberculosis* is of a very distinctive kind, and frequently terminates the lives of tuberculous children. Its essential morbid character consists in the growth of tubercle on the arachnoid, generally in the shape of small miliary granules. They are found most frequently and most abundantly within the fissure of Sylvius, between and upon the convolutions of the brain, and at the base of the brain, and very rarely on the cerebellum. They are generally of the nature of the grey granulations imbedded in the vascular network of the pia mater. The effusion into the ventricles and the softening of their walls are secondary, but not invariable results of the meningeal lesion.

Causes.—Every age is liable to meningitis. Children are often attacked by it whilst teething, under the form described as *hydrocephalus acutus*, and also when labouring under *scarlatina, measles,* or other disease caused by a specific poison. Adult age, as well as the middle periods of life, is still more liable to this affection, both from the greater exposure to the action of the typhus, typhoid, and paludal poisons, to syphilis and to mechanical injuries, as well as to the greater intemperance and greater excitement incident to this age. In old people meningitis is likewise common; and two forms of the disease are met with in the aged,—namely, the *acute* and the *chronic*, both of which present great varieties in their character and progress (MACLACHLAN), and both sexes perhaps suffer in nearly equal proportions.

Meningitis is also apt to occur as the result of *gout* or of *rheumatism*, constituting *gouty* or *rheumatic meningitis* (MACLACHLAN); and it is an occasional if not a frequent consequence of Bright's disease (CHAMBERS, GOODFELLOW, MACLACHLAN).

Meningitis is indeed a disease which most commonly occurs from the action of some specific morbid poison; and there are few agents of that class which are not apt to act on the membranes of the brain. There are many instances also of persons suffering from *arachnitis* after exposure to the heat of the sun, or to what is in common language called the "*coup de soleil,*" and which will be considered under the head of *insolatio.* Intemperance, as well as great mental work and anxiety, is also a frequent cause of the chronic forms of the disease; but the abuse of alcoholic liquors is perhaps the most common predisposing cause; while fits of intemperance occasionally are the direct agents which induce the disease. It is especially apt to be associated with or to follow *delirium tremens* in the aged, and likewise *gout* or *rheumatism.* It is also especially connected with *insanity,* and with every structural disease of the brain; and to these causes must be added mechanical injuries, diseases of the bones, and morbid growths, especially in cases of primary implication of the *dura mater.*

Symptoms and Diagnosis.—*Meningitis* has usually been described in three stages. The symptoms of the first stage are those of excitement, resulting from diffuse inflammation. The symptoms of the second stage are those of compression, marking that effusion has taken place; while those of the third stage are associated with progressive recovery, or with the modes in which death may occur.

But there are symptoms characteristic of several forms of this disease, which may be classed as follow:—

(*a.*) *Simple Meningitis.*—The type of this morbid condition is that in which the membranes covering the convexity of the hemispheres are the parts generally affected. The most important facts to be ascertained in the previous history of the case, before the development of cephalic symptoms, relate to the general health, and especially to any signs of cachexia or diathetic states, such as *tuberculosis, rheumatism, gout,* or of the specific action of implanted poisons, such as *syphilis.* It is important also to ascertain whether any blow on the head has been sustained, or if the patient has been much exposed to the sun; whether any disease of the ear or nose exists; whether application to study has been intense, or to the cares of business.

Premonitory symptoms may be trifling, or absent altogether. The most common are slight but increasing pains of the head, sensorial disturbance, irritability of temper, or restlessness, with some general *malaise.* Rigors quickly supervene, or simple chilliness, with *cutis anserina* and pallor of the surface, quickly followed by febrile re-action. An attack of convulsions may supplant the rigors, especially in children. Such, however, are by no means common in the adult, and are not necessarily indicative of any severe or advanced lesion. The fever is commonly high; the pulse sharp, hard, and frequent; the respirations are irregular, performed with a sigh, and often with a moan.

Niemeyer observes, that in scarcely any other disease of the brain is the fever of a similar character and equal in severity to that of meningitis, and consequently it is of great importance in diagnosis.

If the frequency of the pulse subside after the disease has lasted for some time—if it fall from 120 or 140 beats to 60 or 80, while the other symptoms of fever and the functional disturbance of the brain increase—the evidence is almost pathognomonic of *meningitis.* The skin is hot; the bowels obstinately constipated; and evacuations, when they occur, are dark and offensive. In this stage there is little or no prostration of strength. The headache of fever is supplanted by acute and intense pain; the face flushes and turns pale alternately; the eyeballs stare, and the conjunctivæ become injected.

The functional disturbance of the brain is partly of the character of irritation, and partly of depression, or of complete paralysis.

The purely nervous symptoms, expressed by *mental, sensorial,* and *motorial* phenomena, are thus classified by Dr. Reynolds:—

1. *Mental.*—The temper is extremely irritable. There is marked somnolence, or wakefulness, and the two sometimes alternate for several days. The most marked feature is delirium, commencing early, and of a furious character, the patient screaming and gesticulating in the wildest manner; the expression of countenance is savage and malignant, or sometimes it has the fierce aspect of the brute.

2. *Sensorial.*—Marked and continuous headache (*cephalalgia*) prevails, with exacerbations of a darting violent character, eliciting from the patient, and especially from children, a sharp piercing cry. Pain is increased by movement, and the patient holds the head with the hands; or, if a child, frequently carries them to the head. The headache is increased by sensorial impressions, and hence the eyes are obstinately closed, and the ears, if possible, kept covered with the bedclothes. Double vision, *tinnitus aurium*, *formicatio*, and subjective sensations of various kinds are present. The sensorial disturbances are highly marked.

3. *Motorial.*—Restlessness is incessant, sometimes general or partial. The muscles of the face and limbs twitch involuntarily. There is strabismus, or the eyeball is unsteady, and with a contracted or oscillating pupil. Vomiting is frequent, without epigastric pain or tenderness, and often without nausea.

This stage generally lasts from *one* to *four* days, and its characteristics may be shortly expressed as consisting of—*the combination of great nervous hyperaction, with marked fever, a peculiar cry, cephalalgia, vomiting, and constipation.*

The second stage is one of a transition from the first to the third. The fever diminishes. The pulse sinks in frequency and force, becoming variable in frequency between very wide limits, and in very short intervals of time. Respiration becomes peculiarly irregular. The bowels continue constipated. The tongue becomes furred and dry. The heat of the head persists, but the body generally is cool.

The nervous phenomena present remarkable intermissions during the further progress of the affection, especially in the following points:—

1. *Mental.*—Delirium becomes quieter, or passes into coma; or the patient may appear collected and well.

2. *Sensorial.*—Excitement diminishes and disappears, and drowsiness is the most common feature.

3. *Motorial.*—Muscular twitchings generally are increased on both sides of the body. Convulsions are common in the child, and spasms

often alternate with paralysis. A violent general convulsion may throw the patient at once into

The third stage.—It may come on almost immediately, or a week of transition symptoms may intervene. The face becomes sunken, the extremities cold, and the abdomen retracted. *Sordes* form on the gums and teeth. The pulse flutters, becomes thready, feeble, and uncountable. Great prostration of strength supervenes.

The nervous functions are gradually suspended.

1. *Mental.*—Perception, Volition, and Ideation become lost, so far as can be ascertained by corporeal signs.

2. *Sensorial.*—Anæsthesia is complete.

3. *Motorial.*—There is absolute paralysis to almost every form of stimulus, observed first in the eyelids and eyeballs and then in the limbs. Muscular relaxation becomes complete, as evidenced by the dilated pupil, stertorous breathing, involuntary micturition and defecation.

Generally there is absence of nervous action, and organic life gradually dies out.

The duration of these stages is various. For the most part each lasts a week; but one or more stages may be wanting.

The tongue in the first stage is white; in the second it becomes brown; in the third it again cleans, if the patient does not die. The pulse, likewise, in the first stage is from 90 to 100; in the second from 110 to 130; and in the last stage it either gradually returns to its natural standard, or runs on too rapidly and too feebly to be counted.

The symptoms which have been described are those which especially mark *arachnitis* at the superior portions of the brain. When, however, it occurs at the base, or in the ventricles, some differences are observable, although the condition cannot always be distinguished; but in some cases such a location of the malady is rendered probable if the intellect is less impaired, the passions more excited and the patient lies fretful, impatient, morose, and although somnolescent, he occasionally cries out and grinds his teeth very early in the disease; while the parallelism of the axis of the eye is frequently affected at an early period.

(*b.*) *Tubercular Meningitis in the Child.*—It is an object to ascertain the existence of the scrofulous cachexia in the first instance, as described at pages 16, 17, *ante, et seq.* If such exists, *tubercular meningitis* may supervene on the occurrence of any febrile disturbance, with slight thirst and anorexia; irregular and somewhat quick pulse; vomiting and constipation; clayey evacuations deficient in bile; red and moist tongue; dry and hot skin, and other phenomena of general derangement. The temperature, as measured by the thermometer, will be found to be persistently above the normal amount (Ringer).

The special nervous phenomena are generally feebly marked.

1. *Mental.*—There may be irritability of temper and peevishness, with some slight delirium at night, rarely commencing early in the disease, disturbed sleep and restless manner.

2. *Sensorial.*—Pain in the head prevails, with intolerance of light and sound. Vertigo is also indicated by staggering, or clinging to objects for support.

3. *Motorial.*—Grinding of the teeth prevails, occasional vomiting, unsteady restless movements, and dragging of the limbs. After three or four days of these premonitory symptoms,

The second stage commences, with heat of head and flushings of the face, alternating with pallor. The pulse is irregular, and commonly, when the child is still, it is of little frequency, but it rises rapidly if the child is disturbed, The vomiting ceases, but the constipation persists, with retracted abdomen.

The following special nervous phenomena become more marked:—

1. *Mental.*—The child inclines to lie quiet, and resists being moved, as if pain was increased by motion. There is delirium, which is sometimes fugitive and sometimes persistent.

2. *Sensorial.*—The cephalalgia increases. The expression of countenance bespeaks great suffering, and the face looks aged. A peculiar piercing cry is now and again given by the child. The eyes are closed, and there is a tendency to drowsiness.

3. *Motorial.*—Strabismus and muscular twitchings occur. The pupils are variable, and often oscillate, and the eyeballs are unsteady.

The third stage of tubercular meningitis is ushered in by the general symptoms of approaching dissolution, such as coldness of the extremities, clammy perspiration, and an excessively rapid but feeble pulse.

The special nervous phenomena are first those of exalted spinal action, and then those of general prostration.

1. *Mental.*—Drowsiness passes into stupor, with an idiotic expression of face. There is loss of perception and volition.

2. *Sensorial.*—All signs of activity give place to anæsthesia, and the eyes are half open.

3. *Motorial.*—Death approaches by convulsions, with partial paralysis, *subsultus-tendinum,* clenched hands, retracted head, and automatic movements, giving way to general relaxation.

(*c.*) *Tubercular Meningitis in the Adult.*—The symptoms occasionally assume an apoplectic, sometimes a convulsive form, at the commencement; and the febrile character is generally imperfectly marked. The premonitory symptoms are those associated with the scrofulous cachexia, and *meningitis* may occur at any stage of the lung disease. The following are its features:—

After some remission of chest symptoms, special nervous phenomena referable to the head may supervene.

1. *Mental.*—The patient looks bewildered, with a dull, heavy, expressionless face, often highly characteristic. There also appears to be some *intellectual* incapacity to speak; the patient, seeming to understand what is said or asked, looks at the inquirer for a few seconds, and then turns the head away without a reply. There is often marked somnolence.

2. *Sensorial.*—Pain in the head, fixed to one spot (generally the forehead), is the most striking symptom, of considerable intensity, and is persistent for many days.

3. *Motorial.*—An attack of convulsions may precede every other symptom, but paralysis is rare.

The second stage is of very variable duration, the pulse is highly irregular, while alternate flushings and pallor of the countenance are common, and all the symptoms already noticed become more intense, mild delirium prevails, and the face becomes increasingly stupid-looking.

Paralysis of volition alternates or co-exists with clonic or tonic spasms, strabismus, or prolonged convulsive attacks.

The third stage of tubercular meningitis in the adult is marked by increasing stupor, immobility, and involuntary defecation and micturition.

The general characteristics of *tubercular meningitis* thus consist in the occurrence of fixed pain, vomiting, dulness of intellect, and duskiness of the face, with partial paralysis or convulsions, slight fever, and diminution of the chest symptoms in a patient demonstrably tuberculous. The indications of inflammatory action are only feebly marked, not only during life, but after death; and very often the febrile state which had existed before becomes less noticeable at the onset of cerebral symptoms. Still, heat of head, injected conjunctivæ, and flush of face, denote a condition of vascular excitement; while persistent headache with febrile symptoms in a patient otherwise phthisical, when it can be traced to no other known cause, is strong presumptive evidence of impending *tuberculous meningitis* (HILL). The aid of the thermometer should invariably be sought in diagnosis, (see p. 233, *et seq.*, vol. i.) But there are also cases in which the disease of the brain is marked by the absence of ordinary symptoms. The premonitory phenomena already noticed, for example, may be absent, especially in children; and then the steady persistence of any one symptom becomes of great importance in diagnosis—for example, sickness, headache, constipation, drowsiness, heat of head (WEST).

(*d.*) *Acute Meningitis of the Aged.*—Dr. Maclachlan gives the best account of this form; and the phenomena he describes may be classified as the previously described forms have been. It seldom occurs suddenly, nor with the intense symptoms which prevail in the meningitis of early manhood. It commences insidiously, without rigors, and very frequently some days elapse before it can be recognized, even by persons familiar with the diseases of old age. The pulse is natural, the tongue remains clean, the bowels regular, and there is little or no vascular excitement, local or general, but the symptoms at the outset are purely of a nervous character.

1. *Mental.*—The temper is peevish or irritable, with more or less confusion of thought, inattention, and forgetfulness. "If infirm, and already an inmate of an hospital, the patient commits strange mistakes—takes possession of another's bed, uses the spitoon instead of the chamber-pot, and is frequently found lying outside the clothes, or with his feet where his head ought to be. When addressed, his answers are rational, but still there is a peculiarity in his manner and expression of countenance, an apparent slowness of comprehension, and a vacancy of the eye, that warns the physician of the approach of some cerebral disease" (*The Diseases and Infirmities of Advanced Life*, p. 85).

2. *Sensorial.*—The appetite is slightly if at all impaired; and the skin remains of normal temperature.

3. *Motorial.*—Restlessness prevails, although prostration is apparent. In walking, the gait is unsteady, and in lifting anything to the mouth the hand trembles. In a few rare cases the disease commences with convulsions.

While such symptoms are being developed, after twelve, forty-eight, or sixty-two hours, but generally within twenty-four hours, more or less febrile re-action is expressed by slight increase of temperature merely, and without any marked redness of the face or acceleration of the pulse. The nervous symptoms are now of the following kind:—

1. *Mental.*—Wandering, low muttering delirium and incessant talking become frequent and characteristic symptoms. Maniacal excitement is uncommon. For a day or two the patient may answer questions rationally, though slowly and hesitatingly, when roused from the reverie in which he is generally wrapped. At a still more advanced stage there is coma.

2. *Sensorial.*—Headache is not a prominent symptom. It is so slight or unfrequent that unless the patient is pointedly asked the question, there is never any allusion either to it or to giddiness, or to *tinnitus aurium.* This absence of headache is pointedly noticed by Dr. Maclachlan as a remarkable character; for, even in the most acute pus-forming or false-membrane-forming meningitis, headache may be entirely absent from the beginning. The eyes are suffused, the pupils either slightly contracted or natural. Knitting of the eyebrows, intolerance of light, acuteness of hearing, and vomiting,—common and characteristic phenomena of the disease in early life,—are comparatively rare in the aged. The scalp may be hotter than natural, and, combined with the suffusion and injection of the eyes, is the only physical indication present of vascular excitement within the cranium. The feet are frequently cold, and the surface generally moderately warm. There is usually great thirst, the patient drinks greedily, but seldom asks for liquid. He is apt to refuse food, or takes but a small quantity at a time, when it is presented to him.

3. *Motorial.*—Nervous twitchings and convulsions are observed in the worst cases; and if the patient be raised in bed, these symptoms are frequently induced when otherwise absent, and the head trembles, as well as the upper extremities. When coma prevails, there may be sometimes slight convulsions of the limbs.

The general features of the disease resemble typhus fever. The tongue becomes very dry, and generally brown in the centre. The bowels cease to act without medicine, but are not obstinately confined. Towards evening there is generally increased febrile disturbance, denoted by flushing of the face, where it had been previously pale and dejected; dryness of the skin, greater heat of the scalp, and acceleration of the pulse. In numerous examples, while dulness of intellect and somnolence are the first symptoms observed, in others great and unusual loquacity, with redness of the face and heat of the scalp, ushers in the disease. The progress of the disease is equally inconstant. In not a few cases the symptoms of febrile excitement are wanting; but dulness of intellect and stupor, with more or less insensibility of the limbs and relaxation of the sphincters, are frequently prominent symptoms throughout the whole course of the disease, as if the medullary substance of the brain were congested or suffered compression from early exudation on its surface. Such examples of an apoplectic character are more frequently met with in old persons addicted to fermented liquors, in whom the cerebral vessels are enlarged from repeated engorgement. Excessive venous congestion of the brain prevails in such cases during life; and the symptoms of meningitis are marked, and apt to be confounded with effusion into the brain, or softening of its texture (MACLACHLAN, l. c., p. 87).

(*e.*) *Chronic Meningitis of the Aged* is extremely subdued in its symptoms and insidious in its approach. It is not unfrequent as a result of albuminuria, or of repeated attacks of *delirium tremens;* or it follows *gout* or *rheumatism.* The characteristic phenomena may be arranged as follow:—

1. *Mental.*—It is almost uniformly accompanied with great impairment of the mental faculties. There is very marked loss of memory, slowness of comprehension, periodical fits of passion.

2. *Sensorial.*—Vertigo prevails, with ringing in the ears; and occasional attacks of headache occur, with or without vascular excitement.

3. *Motorial.*—Speech is thick. There is paralytic weakness of the lower limbs, and the gait becomes tottering and feeble. All movements, whether of the upper or the lower limbs, are performed slowly, awkwardly, and with more or less of uncertainty.

The energies of the whole system are reduced. The functions of organic life are impaired. The appetite may be good, but digestion is slow, the bowels being inactive, and the various secretions vitiated or diminished. "Sooner or later the aged invalid takes to bed reluctantly. There he lies uncomplaining, vegetating, the mere wreck of what he formerly was, both in mind and body, gradually sinking, and dying often in consequence of sloughing of the hips and nates" (MACLACHLAN, l. c., p. 93).

Diagnosis.—*Meningitis* is distinguished from *encephalitis* by the headache—always severe, the early delirium, and by the general absence of *hemiplegia.* It must be admitted, however, that disease of the brain and of its membranes is often conjoined, so that *meningitis* is not in all cases a simple affection, but is ultimately conjoined with *cerebritis,* the inflammation of the membranes usually extending to the cortical portion of the brain.

From *delirium tremens* meningitis is mainly distinguished by the character and mode of accession of the delirium, and the tremor, and by the *mental, sensorial,* and *motorial* phenomena.

From *typhus fever* it is mainly distinguished by the absence of the characteristic eruption or mulberry rash (see vol. i., p. 497).

Treatment.—As a general principle, remedies have little influence over those forms of the disease which arise during the progress of any *specific* or *constitutional* disease, independent of the general treatment indicated for that disease. When arachnitis arises from mechanical injuries, the treatment is generally by bleeding, active purgatives, especially by calomel and scammony, and by cold applications to the head. In chronic cases Foville strongly recommends the cold *douche*—

pouring cold water over the head from a pitcher held some distance above it.

This remedy must be used with caution, as being a powerful depressant, yet producing less ultimate debility than bleeding. It acts, doubtless, by cooling down the general mass of the blood; and patients almost always recover consciousness under its influence. To secure a lasting result it must be repeated at intervals of a few hours.

In children I have certainly seen good results from inunction with mercurial ointment. (See remarks under the treatment of "Hydrocephalus.")

In advanced life it rarely happens that the symptoms are so intense as to demand general blood-letting; but in vigorous constitutions this measure is sometimes necessary. Dr. Maclachlan gives a well-marked instance of its usefulness (l. c., p. 90). The cases which demand it are those attended by high cerebral excitement and vascular action. But as a rule local blood-letting is not only infinitely more safe but more beneficial, and it can be repeated from time to time, as the nature of the symptoms may indicate; while general blood-letting can very rarely be resorted to more than once, and that only at the commencement of the attack. The beneficial effects of local blood-letting may be greatly aided by keeping the head well raised, and by the constant application of cold water to the scalp; or the occasional use of bladders filled with crushed ice.

The bowels should be opened as rapidly as possible, unless the patient is feeble, emaciated, or greatly exhausted. The most active and searching purge is obtained by *calomel* and *jalap*; or four or five grains of the *compound extract of colocynth* should be given, combined with two or three grains of calomel, in cases uncomplicated with *gout* or Bright's disease, and followed in a few hours with a dose of *salts* and *senna*. A fair proportion of nutriment must be given, in the form of *milk, strong beef-tea, sago, tapioca,* or *arrow-root;* and the patient should be kept in a quiet and darkened room, and with cold compresses constantly applied to the head.

The more active symptoms being subdued, but not till then, a blister should be placed on the nape of the neck, if coma should ensue. The bowels should be kept open regularly, and the strength supported by mild unstimulating nutriment. When nervous irritability continues during convalescence, *henbane*, or *muriate* of *morphia* may be given (MACLACHLAN, l. c., p. 92).

In the *chronic* form the periodical attacks of headache or of insanity may generally be relieved by a brisk purge; and the head should be kept cool by cloths dipped in vinegar and cold water. If vascular excitement prevails, leeches may be applied behind the ears. The bladder must be kept empty of urine by the catheter, and although wine must be withheld during the active stage of the disease, it is beneficial when the vital energies begin to fail (MACLACHLAN).

The dietetic treatment should be strictly antiphlogistic, the patient especially avoiding all mental excitement; and, if not secluded, he should at least be kept tranquil, not only in body, but also in mind.

TUBERCULAR MENINGITIS—*Syn.*, ACUTE HYDROCEPHALUS.

LATIN EQ., *Meningitis tuberculosa*—Idem valet, *Hydrocephalus acutus;* FRENCH EQ., *Méningite tuberculeuse;* GERMAN EQ., *tuberculöse Hirnhautentzündung*—Syn., *Acuter wasser-hopf;* ITALIAN EQ., *Meningitide tubercolare*—Sin., *Idrocefalo acuto.*

Definition.—*An effusion of serous fluid between the membranes of the brain, or into its ventricles, associated with miliary tuberculosis of the meninges, or at the base.*

Pathology.—The meningitis of acute hydrocephalus may be limited to the membranes of the base of the brain; the effusion into the subarachnoid space and the ventricles of a readily coagulating exudation, containing few pus globules. Nodules of tubercle may generally be detected by their opacity over the translucent arachnoid. When the meninges generally are affected, miliary tuberculous growths are to be seen, and effusion is general in the ventricles. These lesions are part of an acute attack of scrofula, *with* deposit of tubercles in the meninges. They occur generally in children who are of such a scrofulous disposition that in later life they would be likely to suffer from pulmonary consumption; or they are the offspring of consumptive or otherwise debilitated parents—badly nourished, physically ill-developed; but mentally often very sharp and bright. "Too wise and clever to live long," is a common saying regarding such children.

They generally have a fine skin, very perceptible veins, long eyelashes, and a clear bluish sclerotic. (See pages 16, 17, *ante, et seq.*)

The brain affection is often apt to be regarded as the primary disease; but on *post-mortem* examination it will generally be found that caseous degeneration of bronchial, lymphatic, and mesenteric glands, with tubercles in the lungs or intestinal canal, bespeak the general constitutional state of scrofula with deposit of tubercle.

In adults the development of acute hydrocephalus is generally subsequent to acute miliary tubercle of the lungs, or chronic pulmonary consumption, forming an acute complication to these diseases. The disease may also be a sequence to typhus, enteric fever, measles, or scarlet fever (NIEMEYER).

The age of childhood exercises a most remarkable predisposing influence upon this disease. At that period the rapid growth of the brain, the irritation of dentition, and the great susceptibility of the nervous system generally, are all powerful causes of determination of blood to the head. The greatest number of attacks, according to Percival, occur between the second and the fifth year; or, as a more general law, the disease occurs from infancy to twelve years of age. During the first year of life, however, the disease is rare. Children with large heads and precocious intellects, and more especially those of a scrofulous diathesis, are its most frequent victims. One warning may be learned from this disease—namely, that it is said to be most common in the children of parents addicted to drunkenness, and from this cause it often runs in families.

Although it has been stated that hydrocephalus may occur during fœtal life, and is more common in the early periods of infancy and childhood, yet adult age is not altogether free from it; and Golis has mentioned three cases of persons attacked in old age, two of whom were above seventy, while the other, perhaps less advanced in life, suffered from this affection for ten years. It seems sometimes to run in families; at least, Frank mentions a family of seven children, all of whom were born with this disease; and Golis another, in which six children were aborted hydrocephalic at six months; while three others, born at the full period, were attacked shortly after birth.

Unless in the form of *tubercular meningitis*, *acute hydrocephalus* is undoubtedly a rare disease. But it is no less certain that a disorder occasionally occurs, which is really an *acute hydrocephalus*, due to certain changes of nutrition of the nature of inflammation in the membranes or *ependyma* of the ventricles, followed by more or less effusion into their cavities.

This form of *acute hydrocephalus* is rapid in its course, ending within two, three, or four to fourteen days.

The morbid anatomy of such cases has been described by Dr. Beveridge, of Aberdeen. The surface and substance of the brain appears natural, and the fluid in the ventricles may be clear, but a granular layer of lymphy exudation covers the floor of the ventricles. This layer of exudation appears thin, soft, and grey in colour, and is easily overlooked on account of its resemblance to the parts which it rests upon. It is composed of granules, exudation corpuscles, and imperfectly formed cells. It may be seen covering the optic thalamus and corpus striatum. It is most abundant towards the fore part of the ventricles, where, in the angle between the rounded anterior end of the *corpus striatum*, and the *crura* of *the fornix*, it accumulates to the greatest extent, so as sometimes almost to fill it. At or opposite the foramen of Monro it causes a very peculiar appearance. There the exudation, so far from closing the opening, prolongs it on either side into the ventricle, converting it into a sort of short canal, which opens by oval apertures with sharply defined borders, placed not vertically, but so obliquely as to be nearly horizontal, quite clear of the edge of the fornix, and visible at once on opening the ventricle from above; so that, instead of its being necessary to look underneath the apex of the fornix to see the foramen, the openings of it appear like two oval eyes between the fornix and corpus striatum, and are in full view, without disturbing or touching the parts in any way. This appearance gives the impression as if a soft granular layer had settled down on the floor of the ventricle, filling up its angles and interstices, but prevented from covering the foramen of Monro by the steady set of a current through that opening (*Med. Times and Gazette,* vol. ii., 1868).

Premature or excessive mental exertion and blows on the head may be, and often are, alleged as the exciting cause of this disease. It does not always follow, however, that such is the case. The blow may only be a means of directing attention to the child, after which the discovery is made that symptoms of cerebral irritation exist, which had already commenced.

Morbid Anatomy.—A yellowish opaque exudation is usually conspicuous over the base of the brain, and within the *sub-arachnoid spaces*; and is generally plentiful in the meshes between the *pia mater* and *arachnoid*. It is especially abundant about the *chiasma* of the *optic nerves*, the larger fissure of Silvius, and cerebral fissures towards the convexity of the brain. The *pia mater* is generally covered with whitish granulations, like particles of sand or a hemp seed, most abundant and distinct in the vicinity of the blood-vessels. (See Bastian, *Ed. Med. Jour.*, April, 1867.)

The ventricles, especially the lateral and the third, are sometimes considerably distended with serous fluid—occasionally quite clear, but sometimes clouded with flocculi. The walls, especially the fornix and commissural parts, are generally so softened that they break down at the least touch, as the result of maceration, rather than of inflammatory softening, which may spread indefinitely far beyond the immediate vicinity of the ventricles. The larger the amount of fluid the more bloodless and pale is the brain, and the whiter the softened parts (NIEMEYER).

Symptoms.—Acute hydrocephalus is divided into three stages; the first stage, according to Dr. Cheyne, being that of increased irritability; the second, that of diminished sensibility; and the third, that of convulsions or palsy.

The first stage may be either sudden in its attack, or be preceded for several days by insidious and indefinite symptoms, such as giddiness, so that the child stumbles or falls at play; by a furred tongue, constipated bowels, and perhaps offensive breath. The child shows some change in manner, declines play, desires to sit apart, and rest the head. At length the senses of sight and of hearing become morbidly acute; he starts at slight noises—complains of intermitting headache—rests his head on his nurse's lap—occasionally complains momentarily of his head—and then, after a time, rises up and plays again.

As this stage advances, the pulse rises, the skin is hot and dry, the urine scanty, the stomach irritable, the bowels constipated, perhaps painful, the stools black and offensive, while the brow is knit, and the pupil of the eye contracted or expanded. The most remarkable feature, however, is a great fretfulness of temper, so that the child is not merely pettish, but quarrelsome. If he sleeps, his sleep is short, uneasy, moaning; he also grinds his teeth, rolls his head, and when he wakes up it is with a scream. He is generally sleepy, and dreams a great deal. "We are led to suspect some deeply-seated evil from the frantic screams and complaints of the head and belly, alternating with stupor, or rather lowness, and unwillingness to be roused" (CHEYNE). These symptoms may extend over several weeks; and the child becomes emaciated by fever and impaired digestion.

In much of this description some of the phenomena of *meningitis* may be recognized, and, doubtless, a great number of the acute cases are associated with this morbid state already described (page 149, *ante*), and especially with meningitis at the base, for at the commencement the lesion is localized there, and the characteristic peculiarities in the sequence of its symptoms are so distinctly traceable to the functions associated with these parts as to give grounds for very certain diagnosis. The numerous nerves starting from the base of the brain and passing through the foramina, so that (1) irritation, and (2) paralysis in the parts supplied by the *nerves of the eye*, the *vagus*, and *medulla oblongata*, direct attention to the child. Among the first signs are *contraction*, and then later, *dilatation of the pupil, ptosis of the upper eyelids, vomiting, slowness, and subsequent frequency of the pulse, peculiar changes of respiration*, and depression of the abdomen (NIEMEYER, BUDGE).

The second stage commences when effusion has taken place. The pulse, instead of being rapid, is then as slow, perhaps slower, than natural—sixty beats a minute, or less; but this is chiefly when the patient is in a horizontal position, for if he attempts to sit up, it again becomes rapid. The sickness is also abated; nevertheless, the child lies in a state of stupor and of great unwillingness to be moved, with his eyes half-closed, dull, and heavy, or perhaps staring or squinting; the pupil being still contracted begins to expand—first one and then the other—so that he often suffers from double vision. Loud noises do not now disturb the child, nor light influence the eyes. The stupor, however, is still interrupted by exclamations or shrill piercing screams, which are reflex, while the tremulous hand of the little sufferer is incessantly engaged in picking his nose or mouth. As the disease thus advances, the capillaries of the brain are compressed by the effusion, loss of consciousness begins, epileptiform convulsions supervene, and paralysis of the extremities. For a time the inspirations are imperfect, and then the lungs are filled by a deep sighing inspiration; *coma* becomes deeper, the face assumes a dark colour, and appears pinched, with sunken eyes; and often tetanic contractions of the body cause a painful feeling to those unacustomed to such a disease.

In the third stage the patient either sinks or recovers, and temporary appearances of improvement excite deceitful hopes. If the event is unfavourable, the pulse again rises, the eye becomes red and dim, and the child, delirious, is often attacked by partial or general convulsions, or one limb or one side may be palsied. From this point the powers of life gradually sink, till at last death closes the scene; but for days and days the child may linger on the very brink of the grave. If the the patient should fortunately recover, the stupor subsides, the countenance becomes more natural, the bowels more regular, the secretion of urine perfectly restored, and at length his health, though long broken, is gradually re-established. The duration of this acute form of the disease is estimated at about three weeks, each stage averaging about a week.

The pulse becomes very frequent just before death, and the skin copiously perspires, urine and fæces being passed involuntarily.

Diagnosis.—The vomiting in this affection is characteristic, and has been described as cerebral vomiting (p. 132, *ante*). It is not traceable to errors of diet, nor does it occur after eating, but when the child is raised up, or the head is elevated. When the child is also seen to bend the head backwards, or bore it into the pillows, with the muscles of the neck contracted, and perhaps the cervical glands swollen, the circumstances are very suggestive of the onset of acute hydrocephalus.

Acute hydrocephalus is to be distinguished from enteric fever by the screaming, rolling of the head, grinding of the teeth, and by the absence of the peculiar state of the tongue which marks the latter disease. There is always intense marasmus, although food may be taken.

There is a morbid state resembling the disease which is neither *acute* nor *chronic*, to which Sir Thomas Watson has given the name of *spurious hydrocephalus.* To three great men of the past we owe our knowledge of this condition—namely, to Gooch, Abercrombie, and Marshall Hall. In children, from a few months to two or three years of age, of small make and of delicate health, from exposure to debilitating causes, this morbid state not uncommonly supervenes. It is indicated by heaviness of the head and drowsiness. The child lies on its nurse's lap, unable or unwilling to raise the head. It seems half asleep, one moment opening its eyes and the next closing them again, with a remarkable expression of languor: the eyes are unattracted by any object put before them, and the pupils remain unmoved on the approach of light. The breathing is irregular, sighs are occasionally expressed, and the voice is husky. The tongue is slightly white, the skin is not hot, but sometimes colder than natural. In some cases there is now and then a slight and transient flush. Diarrhœa has often existed for some time, or the child has been severely purged by medicines, or having been weaned, has ceased to thrive since its change of food. The peculiar green colour of the stools, so frequent in this disease, seems to be imparted to them in the lower portion of the intestine, the fæcal contents of the upper portion being of a pale drab colour, while the bile in the gall-bladder is of a yellow colour. When this condition of things occurs in a more elderly child, it has been generally brought about by depletion, by loss of blood, or by medicines. As the more marked symptoms are sometimes ushered in by extreme irritability and a feeble attempt at re-action, cases of this kind (which Dr. Marshall Hall named "the hydrocepha-*loid* disease") have not unfrequently been mistaken for acute hydrocephalus, and treated accordingly, the patient being generally leeched out of its life. In very young children the diagnosis is sometimes very difficult between congestion and exhaustion, between fulness and emptiness.

Sir Thomas Watson, from whose lectures these characteristic notes are taken, suggests the following test:—"As a guide to diagnosis, take notice of the state of the unclosed fontanelle. If the symptoms proceed from plethora, or inflammation, or an approach to inflammation, you will find the surface of the fontanelle convex and prominent, and you may safely employ and expect benefit from depletion. If, on the other hand, the symptoms originate in emptiness and want of support, the surface of the fontanelle will be concave and depressed; and in that case leeches or other evacuants will do harm, and you must prescribe better diet,—ammonia," brandy in arrow-root, milk from the mother's breast, if possible, and all such means as will tend to foster and nourish an infant.

Prognosis.—Most physicians are agreed that if acute hydrocephalus shall unequivocally declare itself, it will be rapid, and almost inevitably fatal.

Treatment.—Tuberculous meningitis in the form of *acute hydrocephalus* can only be successfully combated in the first or earliest stage. As the first symptoms are those characteristic of inflammation of the brain, and especially of its meninges, and as we can only conjecture to what it may turn, *leeches* behind the ears ought to be applied in the first instance. If this local abstraction of blood proves beneficial, it may be repeated during subsequent relapses. At the outset of the disease laxatives and ice compresses may also be used (NIEMEYER).

There is, perhaps, no class of cases in which the sanative powers of judicious blood-letting become so apparent as in children in whom the disease has been observed early and carefully watched. When the inflammatory symptoms are less decided, and the headache slight, and the disease drags on slowly, a single venesection is admissible. Dr. Alison relates the following highly illustrative case, confirmed now by the teaching of Niemeyer :—

> "A boy aged eight years, of rather delicate habit, and who had complained occasionally for *some weeks* of headache and disordered bowels, temporarily relieved by laxatives, calomel, antacids, and a careful regimen, but recurring, and attended with gradually increasing febrile symptoms, and *shooting* pain of head; impatience both of light and sound; pulse gradually rising to 108, distinctly sharp, and beginning to intermit, and then nausea and vomiting, not referable to any medicine taken, but gradually increasing, until it recurred every time that he sat up in bed, for nearly twenty-four hours; and a slight but quite perceptible squint showing itself. The full action of laxatives and one application of leeches, as well as cold to the head, having failed to make any impression on this course of things, he was bled at the arm (which in such circumstances and at that age has often appeared to me distinctly preferable) to *twelve ounces*, and the blood was sizy. I do not know what further evidence we could have had of the existence of such inflammation within the cranium as would infallibly, if let alone, have gone on within a few days to delirium, stupor, dilated pupil, slow pulse, succeeded by very frequent pulse, convulsions, and death, and have left after it the usual appearances of the *acute hydrocephalus* (of Whytt, Cullen, and Abercrombie, now described), dryness of the membranes on the surface of the brain, distension of the ventricles of the brain with serum, and some of the marks of inflammatory action either on the membranes or surface of the brain. Instead of this, however, I am quite certain that from the time of the bleeding at the arm this boy *never once vomited*, that the intermission of the pulse was *never again observed*, nor did he again complain of lights or sounds. The pain of the head, although less violent, continued in the evening of the same day, and *twelve leeches were applied* within eight hours after the bleeding; and from that time he never once complained of this symptom, nor admitted that he felt it; and *from this moment he recovered perfectly*, and much more rapidly than he had done from much slighter febrile attacks previously; neither has he suffered from that time to this (now thirty years) any return of serious disease" (*Edin. Med. Journal*, p. 777, March, 1856).

In short, all the symptoms vanished in twenty-four hours, most of them during the flow of blood, and never recurred,—a change, under the circumstances, so sudden and sanative as is rarely if ever seen after the use of any other remedy for the same combination of symptoms. With reference to the senative influence of general blood-letting in children for inflammatory diseases, if performed at the outset, Dr. West observes, that such depletion is as important a remedy as in the adult; nor will the most energetic employment of any other antiphlogistic measure enable us to dispense with blood-letting. In a healthy child two years old, a vein (if easily found) may be opened in the arm, and *four ounces* of blood allowed to flow, if faintness be not earlier produced, without our having any reason to apprehend that the plan adopted is too energetic. The immediate effect produced is greater than that which follows local depletion, and the quantity of blood abstracted is less (West, *Diseases of Children*).

Bleeding in children and in adults exercises an influence quite different—because under totally different circumstances—not yet inquired into; but certainly blood-letting at ages under adult life is soon repaired compared to its reparation after that period. Hasse recommends very small doses of *morphia* ($\frac{1}{24}$th of a grain) in the early stages.

The first thing to be done by way of medicine is to purge the patient. The purgative is not of great moment, provided it acts freely. Some prefer two to five grains of *gamboge,* others five grains of *calomel* with thirty grains of *compound jalap powder.* Such doses are to be followed up by a *black draught,* or the *sulphate of magnesia.* So difficult is it to obtain the action of purgatives in this disease, that doses of three times the strength for adults have been in some instances required; but such large doses are never to be given until the inefficiency of smaller ones has been ascertained, (Sir Thomas Watson's *Lectures,* No. xxvi.)

The stools are generally black, or extremely offensive; and, this state of the bowels corrected, the disease, if sympathetic, often ceases. If, however, the head be not relieved, some leeches should be applied to the temples, and the head should be shaved and surrounded with some cold evaporating lotion, such as a towel dipped in cold spring water, or in *vinegar and water,* or in solution of the *muriate of ammonia, with vinegar.*

If the disease be advanced, no efficient treatment has as yet been determined. The symptoms do not yield to the lancet like those of simple inflammation. *Mercury* has also been used to a great extent, but with little success. In urgent cases, for instance, *mercurial ointment* has been rubbed on the back and thighs, even in very young children, to the extent of half a drachm to a drachm three or four times in the twenty-four hours. *Calomel* also has been rubbed on the gums to the extent of three or four grains every four or five hours, and it has likewise been given by the mouth in doses of two grains every third or fourth hour. *Mercury* given in these large doses, it must be remarked, seldom produces salivation; for Dr. Clark says he never saw that effect in children under three years of age except in three cases. But the remedy is not successful, and more generally produces *spinage-like* stools, and irritates the alimentary canal. In France the mercurial treatment has been so unsuccessful that some practitioners have even tried a most opposite remedy, namely, *quinine,* but the result has been equally fatal. *Blisters, moxas,* and other modes of cauterization have been used as auxiliary treatment, but without apparent benefit.

During the course of the disease the diet should be slops and light puddings.

In congenital hydrocephalus the unassisted efforts of nature seem incapable of effecting a cure, and it is extremely problematical if medicine is of any use. When, however, the case is deemed hopeless, the propriety of evacuating the water by means of an operation may be entertained. Golis has given the names of twenty-seven writers who have expressed themselves in favour of it, especially if the fluid be slowly evacuated, and at several repetitions of the operation; yet he himself, along with seven or eight others, proscribe it altogether as cruel and useless. But it has been successful; and when the operation is performed, it seems an axiom that the fluid should be allowed to escape gradually, for otherwise extreme faintness and collapse may be expected. In such cases small doses of ammonia, or a few tea-spoonsful of brandy and water may revive the little patient. Should re-action take place, however, at a subsequent period, a few leeches and a cold lotion ought to be applied to the head. It seems also determined that the younger the child the greater are the chances of success; for if it lives a few years the sutures of the cranium, though open at the top, are united by bone towards the base of the skull, and thus present a mechanical obstacle to their closure; consequently, the operation is more apt to fail. If this disease, though existing at birth, should not develope till later in life, *blisters, mercury,* and *iodide of potassium* to salivation are the remedies mostly relied on.

INFLAMMATION OF THE BRAIN.

LATIN EQ., *Inflammatio cerebri;* FRENCH EQ., *Cérébrite;* GERMAN EQ., *Entzündung der Hirnsubstanz;* ITALIAN EQ., *Inflammazione del cervello.*

Definition.—*Inflammation of the brain substance, with or without implication of the membranes, usually partial, and in many cases dependent on local injury or foreign deposit.*

Pathology.—Acute inflammation of the substance of the brain is rarely uncomplicated and primary. As a rule, it results as a consequence of previous diseases, especially *specific fevers, exanthematica, diseases of the ears, extravasations of blood, tumors or tubercles of the brain, alcoholic poisoning, or external injury.* The most intense in degree, and at the same time often the most limited *cerebritis,* is from injury. The whole of the brain substance is rarely if ever affected, but one limited portion or several separate portions may be affected simultaneously in the white, grey, or membranous parts. The affected part acquires first increased vascularity, which increases to a bright red tint, deepening as the disease advances, until it assumes a reddish brown, and occasionally even a brownish or green shade. In keeping with the inflammatory process the parts affected lose cohesion, and become softer than natural, and may proceed to the formation of pus—a result more common in chronic or subacute forms of cerebritis rather than in the acute, and most apt to occur in persons of scrofulous and unhealthy constitution, or as the result of injury or local disease, especially of bone (CRAIGIE).

Vascular turgescence and extremely red congestion of the brain, as seen in *typhus, exanthemata, epilepsy, delirium tremens, tetanus, convulsions, hydrophobia,* bring the lesion very nearly, if not altogether, to the condition of inflammation.

Morbid Anatomy.—As to hyperæmia of the brain, characteristic of or approaching to inflammation, it is invariably found that the amount of blood in the vessels and the amount of cerebro-spinal fluid are in inverse ratio. Vessels greatly distended with blood are accompanied by a decrease of arachnoid fluid. It is only in cases where the finest vessels of the brain are distinctly seen injected with red blood, out of proportion to the congestion of central vessels in other parts, that hyperæmia can be said to exist. Distension of the large blood-vessels (which are veins), always seen on the surface of the brain when the calvarium is removed, is a normal appearance. The first fine ramifications of vessels are those supplying the *pia mater;* passing thence into the substance of the brain. It is in such vessels that hyperæmia is to be recognized, mainly by their number visible and their distension with red blood. In the normal condition they can scarcely be recognized by the naked eye. But the congestion of acute cerebritis goes a stage farther. The colouration is general, effusion of blood actually takes place in spots or foci, and the consistence of the texture is changed.

Various results follow the inflammatory process, just as in inflammation of other textures; and in the brain there are chiefly three to be recognized, namely, (1.) Red softening; (2.) Yellow softening; and (3.) Abscess.

It is in the sub-acute or chronic inflammation of the brain that these results are most common. It is the long duration of the process which gives rise to those peculiar modifications to which these names have been given.

A green shade in the colour of the morbid part of the brain is suggestive of the formation of pus; and in proportion as the colour passes from red to yellow or grey, before greenness sets in, associated with pulpiness or a semi-fluid condition, the lesion of red or of yellow softening is to be recognized. It is rare to find perfect purulent matter in a distinct cavity. Effusion of serous fluid generally attends the softening (MORGAGNI, ROSTAN, LALLEMAND, BOUILLAUD, BRIGHT, CRAIGIE). The lesion may be either on the convoluted surface, to the extent of two or three square inches, or at the *septum lucidum* or *central ganglia,* the *cerebellum* or *cruces cerebri;* or the whole brain softened to a pulp.

Symptoms.—In general cerebritis they may be almost negative, or a little more than a gradual failure of all bodily and mental power, as in cases of *typhus fever.* If one part of the brain should be affected more than another, there will be a special paralysis; otherwise the symptoms are little more than weakness of the limbs, strange feelings, and interference with the intellectual faculties, with perhaps giddiness, sickness, headache, and other symptoms common to all febrile disorders (WILKS).

Usually the symptoms of *cerebral irritation* precede those of depression. *Cerebral irritation* expresses itself more prominently, sometimes in the *sensory*, at another time in the *motory*, or at another tlme in the *mental* functions.

Sensory symptoms are manifested by *headache*, which is a frequent but not essential symptom of all cerebral diseases. How *headache* occurs is still unexplained. Increased sensitiveness to impressions on the part of the senses may have an explanation similar to what may account for headache—namely, that the *brain* generally, as well as organs of special sense, may have such hyperæsthesia, that irritations or impressions may annoy or excite, which in other normal circumstances would not have been noticed. Light causes inconvenience; a slight sound, or irritation of nerves by touch, cause unpleasant sensations. The excitation is morbid *ab initio.*

Expression of cerebral irritation by *motorial symptoms* consist of restlessness, sudden starting, gnashing the teeth, crying out, twitching of single muscles, and general convulsions.

Mental Phenomena.—The expression of cerebral irritation by *mental states* mainly consist in a rapid change and loose connection between the thoughts, so that clear thinking is impossible. Ideas are thus confused, so that the patient has false notions about himself and the world generally. He is, in a word, "delirious." So real and intense is the delirium that it cannot be distinguished from true perceptions. Hence he has *hallucinations* and *illusions. Dizziness* is a form of hallucination. It is a vivid representation of the movement of bodies surrounding the patient, or of the body of the patient himself—all being really at rest at the time (NIEMEYER). Conditions directly opposed to these are indications of cerebral depression.

The extreme local lesion of actual *cerebritis* is indicated by *partial anæsthesia, partial paralysis*, and *loss of certain mental functions.*

Treatment.—What has been said regarding *encephalitis* and *meningitis* can only be repeated here.

RED SOFTENING OF THE BRAIN.

LATIN EQ., *Cerebrum fluidum rubens;* FRENCH EQ., *Ramollissement rouge;* GERMAN EQ., *Rothe-Erweichung des Gehirns;* ITALIAN EQ., *Rammollimento rosso.*

Definition.—*Softening of the texture of the brain, as a result of inflammation, to the consistence of pulpiness or of cream-like consistence.*

Pathology.—From what has been stated under the preceding subjects, there are several states to be distinguished within the cranium connected with inflammation, namely,—(1.) "Inflammation of the brain substance," with or without implication of the membranes, usually partial, and in many cases dependent on local injury, or specific deposits, or growths, or specific fevers; (2.) Inflammatory red softening, or accute ramollissement, as contra-distinguished from (3.) "White softening of the cerebral substance," the result of an *atrophic* process and impaired nutrition; and (4.) Abscess.

The red *diffuse* inflammation of the substance of the brain appears to have many degrees. In the first degree the substance of the organ, when cut into, exhibits more bloody points than usual, so that the medullary portion appears as if sprinkled with blood, while the colour of the cortical substance is increased in intensity. If the inflammation assumes a higher degree, the most markedly red appearances generally only partially affect one of the convolutions of the brain, or a small portion of a hemisphere; and the inflamed part varies from a bright rose to a deep red dusky colour. This increase of colour is supposed by many pathologists not to arise from any greater vascularity of the part, but from blood escaping from the vessels and becoming effused or infiltrated into the substance of the brain, forming so many apoplectic spots (BOYER). The inflamed part is generally swollen, and sometimes considerably so, and is generally softer, though it may appear firmer than usual.

As the lesion advances the exudation ultimately assumes an appearance like pus, or becomes more or less softened, approaching in character the condition of pus as it appears to the naked eye. Without suppuration, however, having actually taken place, the mere effect of inflammatory exudation amongst the brain-tissue is to soften its texture and increase its specific gravity, whereas in "white non-inflammatory softening of the brain" the specific gravity is diminished. When the softening results from the exudation merely, before it has undergone any subsequent change, there are traces of congestion, which give to the affected part a red appearance; and the microscope shows, as Bennett first demonstrated, a large amount of exudation-corpuscles interspersed among the broken-down nerve-matter, as well as coating the inner and outer surface of the minute blood-vessels. Wherever exudation-matter exists, although there may be no palpable indication of changed consistence in the tissues, yet the specific gravity will be found increased; and without microscopic examination we cannot tell whether or not congestion of the brain-substance exists, combined or not with the exudation-corpuscles of red softening.

The inflammations of the substance of the brain have thus much in them that is peculiar, depending on the nature of the tissue; and, independent of physical appliances (such as the tests for specific gravity and the microscope), the unaided senses cannot enable us to appreciate the exact pathological significance of morbid changes. It is only in a small number of cases that the red colour of the brain is characteristic of inflammation; for in by far the greater number there is no increase of redness; and on account of the frequent occurrence of such cases, *ramollisement* has been described by many authors as a distinct idiopathic disease. The liberation of phosphoric acid and of fatty acids seems to be associated with yellow softening; and there is decided acid re-action of the fluid contained in the soft part (ROKITANSKY).

The varieties of softening, as characterized by their colour merely, cannot be regarded as essentially different: they are peculiarities due to the nature of the tissue and the effects of the morbid process upon its component parts. It appears to be the *liquor sanguinis,* rather than the *corpuscular* part of the blood, which takes the chief part in cerebral inflammations where softening is the prominent phenomenon. The characteristic of the part thus affected is, that it is generally whiter or greyer than the natural colour of the brain, and also softer than its natural substance; and, accordingly, a rough way to appreciate the presence of this softening is to allow a gentle stream of water to flow upon the suspected part; if softening exists, the softened parts will be gradually washed away. This softening, however, has many degrees, and in some cases can be only accurately determined by a microscopic examination. In its extreme form the softened portion of the brain is absolutely diffluent, so that it can be poured out of the cranium with as much facility as a thickened cream or a thin jelly can be poured from one cup into another. Sometimes nearly a whole hemisphere has been thus destroyed (BRODIE, MACLACHLAN). In this semi-liquid state much serum can often be expressed from it.

Extreme softness of the brain, unattended with inflammatory coloration, may be well seen in those cases where the brain has been extensively injured, as by sabre wounds. Many such cases are related by our army surgeons. In cases of *hernia cerebri,* also, not only does the protruded portion become softened, with red particles of blood intermixed in it, but in acute cases, which terminate fatally in a few days after the injury, "the medullary structure, intervening between the base of the protruded part and the *anterior cornu* of the lateral ventricle, had entirely lost its natural structure, and become soft and pulpy. Around this disorganized mass, and extending across the *corpus callosum* into the medullary substance forming the roof of the opposite ventricle, the brain had undergone a change from its natural colour to a greyish-blue white, while it still retained its natural consistency" (STANLEY). It was remarked, also, in this case, during the last three days of life, that a very considerable quantity of fluid constantly oozed from the centre of the protrusion, whence it trickled down the cheek in a continued stream. Such cases show the acute and rapid nature of the softening process as an inflammatory phenomenon, but without any obvious discoloration. In fact, most of the phenomena connected with the inflammatory process in the nervous substance are characterized by the apparent want of increase of the red part of the blood.

Symptoms.—Although the anatomical conditions of the brain-substance in acute *ramollissement* may not be associated with an appearance of much red blood, yet the clinical history of the disease seems closely to resemble that of cerebral hæmorrhage, and it is very often impossible to tell whether the physician has to deal with a case of *apoplexy,* as commonly understood, or a case of *cerebral softening.* The premonitory symptoms peculiar to softening appear to be absent in a half, or more than a half, of these cases (ROSTAN, DURAND FARDEL). In some instances, however, the premonitory symptoms afford strong probability of softening, and are of much value: the absence of them, however, cannot be regarded as equivalent to the absence of softening (REYNOLDS).

The attack itself may be gradual or sudden. Thus, after the progressive development, during some hours or days of such premonitory symptoms as have been mentioned in the previous affections of the nervous system, the patient gradually becomes apoplectic, or he may at once appear to become so suddenly and instantaneously, without the premonitory symptoms. In the latter case, however, the attack is due to congestion; it gradually passes away, and the patient recovers intelligence for a time, but the confirmed symptoms of softening remain. They are as follow:—

1. *Mental Symptoms.*—Transient excitement or mild delirium may precede the abolition of Perception; and when this does occur it is highly characteristic. Coma is frequently developed abruptly, and is often of the following peculiar character:—The patient lies still, as if in a profound sleep, but immediately gives the hand or puts out the tongue, if told to do so, Intelligence remaining intact. The loss of Perception and Volition, however, is not recovered from. Dulness and obscuration of Thought and Perception prevail often to a marked degree.

2. *The Sensorial Symptoms* are not so well marked as the motorial. Hyperæsthesia has erroneously been considered pathognomonic of ramollissement, perhaps because it is more common in softening than in any other apoplectic disease. Numbness and a sensation of cold are not at all unfrequent.

3. *The Motorial Symptoms* are of two kinds—namely, paralysis, and spasmodic contractions of muscles. The face-muscles act unequally, producing deviation of the features, sometimes very slight, at other times highly marked. Speech is almost constantly impaired, and after slight recovery it continues to be so. Paralysis is commonly limited to one side, sometimes to one limb, but in rare cases it is general. The spasmodic contractions are either of a tonic or of a clonic kind, rigidity or occasional spasm being found in either the paralyzed or non-paralyzed limbs,—most commonly in the former.

The physician, however, will not derive much information from the mere recognition of the presence of single symptoms: it is by a close observance of their combinations that exact diagnosis will be insured. The following combination of symptoms are those which may with most probability be referred to softening:—

(1.) Imperfect coma, partial loss of Perception and Volition, with rigidity of the limbs; (2.) Perfect coma without rigidity; (3.) Paralysis without loss of Consciousness; (4.) Paralysis with hyperæsthesia; (5.) Rigidity, coming on after the return of Perception and Volition.

The after-symptoms of softening are also strikingly different from those of apoplexy. The morbid phenomena do not suddenly disappear, nor is there the gradual improvement which takes place after apoplexy. Enfeeblement of the mental powers most commonly persists, and the motorial phenomena remain. Slight apoplectic-like seizures occur, convulsive movements and rigidity increase, and some little febrile excitement becomes developed, which in severe cases generally assumes a typhoid type, with brown tongue and rapid pulse. From such a condition recovery is rare.

The duration of life in *ramollissement* of the brain is various; but in 109 cases the disease terminated within the periods indicated in the following table:—

DURATION OF LIFE IN CASES OF SOFTENING OF THE BRAIN.

1 died in 12 hours.	2 died in 12 days.	1 died in 35 days.
1 „ 15 „	3 „ 13 „	1 ,, 36 ,,
1 „ 24 „	3 „ 15 „	1 ,, 47 ,,
1 „ 32 „	1 „ 16 „	1 ,, 49 ,,
5 „ 2 days.	2 „ 17 „	1 ,, 60 ,,
9 „ 3 „	4 „ 18 „	1 ,, 65 ,,
5 „ 4 „	5 „ 20 „	1 ,, 68 ,,
4 „ 5 „	3 „ 21 „	1 ,, 190 ,,
7 „ 6 „	1 „ 22 „	1 ,, 220 ,,
8 „ 7 „	1 „ 23 „	1 ,, 5 months.
8 „ 8 „	1 „ 25 „	2 ,, 6 ,,
3 „ 9 „	1 „ 29 „	1 ,, 1 year.
5 „ 10 „	4 „ 30 „	2 ,, 3 years.
4 „ 11 „		

It thus appears that *ramollissement* of the brain is more frequently an acute than a chronic disease, the greater number dying before the twelfth day, while at the end of a month only sixteen cases out of the 109 were living.

In the thirteen cases which have been collected of ramollissement of the *cerebellum*, the impariment of Intellect was trifling, while motion was greatly affected in all except one doubtful case. In ten cases there was palsy with or without contraction of the muscles of the opposite side of the body; in two others, convulsive actions of both sides of the body; and in the last case observed by Rostan the palsy was on the same side. In this case the disease depended on an exostosis of the petrous portion of the temporal bone. In no instance is it said that any sexual desire troubled the patient.

Diagnosis.—The great difficulty in the diagnosis of acute ramollissement is to distinguish it from apoplexy. In most cases it is not possible to make the distinction. "On the other hand," writes Dr. Maclachlan, "there are numerous cases of acute softening which can scarcely be mistaken for cerebral hæmorrhage. In such a combination of symptoms as the following—and I do not group them artificially, but as I have repeatedly observed them—the probabilities are, that the case is one of acute softening, and not sanguineous apoplexy:—After several days' suffering from headache, giddiness, drowsiness, dulness of comprehension, tingling or numbness in the toes or fingers, followed by sudden hemiplegia, without loss of consciousness, the probabilities are, that the symptoms are due to softening instead of hæmorrhage. If to these symptoms succeed pains in the palsied limbs, and diminution or exaltation of the cutaneous sensation, while the symptoms maintain a variable rather than a fixed character, whether the palsied limbs are contracted or relaxed, or alternately contracted, the chances are still greater that the case is one of softening; and the diagnosis may be considered as established should the paralytic symptoms, and with them the associated stupor, preserve this vacillating, impulsive peculiarity, there being periods of amelioration, followed by increasing coma and increasing palsy" (l. c., p. 193).

Prognosis.—The prognosis in every case of encephalitis is grave; but, as far as we can judge, even acute cases recover, and live for many years afterwards, notwithstanding the unfavourable opinion expressed by Rostan. Inflammatory *ramollissement* of the brain is looked upon as an essentially fatal disease, and the patient seldom survives the formation of an abscess; but it is apprehended that the pus may be occasionally absorbed, and that the opposite walls may unite by granulations, and leave an areolar-tissue cicatrix. Nevertheless, there is some evidence, also, that inflammatory ramollissement is capable of being cured. The *post-mortem* evidence of this fact is the disappearance of one or more layers of the cortical substance, probably by absorption, while the pia mater adheres to this part of the brain. The evidence of the cure of ramollissement in the grey matter of the *corpora striata* and other central parts is the presence of a number of "holes," resembling Parmesan cheese, of a red colour when there has been transudation from the blood-vessels, and of a fawn colour in other cases. The part is atrophied and softened; while the holes may be filled with a limpid fluid, sometimes lined with a membrane (Dr. Simms, *Med. and Chir. Trans.*, vol. xix., p. 413).

Treatment.—In diffuse inflammation of the brain arising from mechanical injuries there can be no doubt that bleeding and antiphlogistic treatment generally are most beneficial, when employed with a wise discretion and at an early period. When, however, inflammation occurs during the progress of a general disease of the system, it is necessary that such measures be employed with the greatest caution, and in the majority of such instances they are better omitted altogether; for we find in many cases of typhus fever, in which the brain is probably partially impaired in consistence, that the patient recovers under a stimulant treatment.

In acute idiopathic *ramollissement* of the brain the treatment can hardly be said to be yet determined; but there is good reason to believe that general bleeding is only to be practised with benefit when the disease is associated with general congestion of the brain.

Most advantage is to be derived from the use of tonics, and of a nutritive diet. As a general rule, *cerebritis* does not admit of so copious depletory measures as *meningitis.* In acute cases, where the premonitory symptoms of congestion are obvious and urgent, bleeding may be carried to a considerable extent, consistent with the nature of the case and the individual; and after a reasonable quantity of blood has been taken without producing nausea or fainting, the bleeding may be again repeated till some decided impression is made. In many cases, on the other hand, cupping, or bleeding by means of leeches to the temples or back of the ears, may be more useful than venesection. Such cases are indicated by the signs of determination of blood to the head, heat of the scalp, suffusion of the eyes, redness of the face, with or without inordinate action of the carotids. Such cases are also more likely to be benefited by the cautious repetition of this treatment than by general blood-letting, which can very seldom be required in softening of the brain occurring in persons turned of fifty (MACLACHLAN).

Even when the pulse may seem to warrant bleeding, and at a time when bleeding may seem safe and proper, it is advisable, before having recourse to this measure, carefully to examine the state of the heart, the state of the arteries at the wrist, and the condition of the corneæ. By such examination, assisted by the previous history of the case, the condition of the cerebral arteries will best be indicated, and the readiest clue gained to the probable nature of the cerebral affection. If the heart's action and sounds are feeble, or if signs of valvular insufficiency are present, it is probable that the softening is anæmic, atrophic—a consequence of partial inanition, and not of partial cerebritis. This conclusion is still more probable if the radial arteries are rigid, and if the *arcus senilis* is fully developed. To bleed under such circumstances is unwarrantable.

In severe seizures resembling apoplexy the bowels should be opened by an enema of castor oil and turpentine. In less urgent cases, where there has not been loss of consciousness, or where it has been restored and the patient is able to swallow, a purgative of colocynth and calomel, with or without croton oil, may be substituted for the enema. Perfect rest in the recumbent posture must be enjoined, with the head slightly elevated, and all constrictures of dress removed from the neck. The scalp should be kept cool by rags dipped in cold vinegar and water, or iced water. If much re-action supervene, leeches may be applied to the head; and should the pulse be weak, twenty or thirty drops of the *spiritus ammoniæ aromaticus* may be administered. The catheter may require to be used; and the bladder must always be examined, in case the urine may be retained. Nourishment must be given in small and oft-repeated quantities.

If sanguineous effusion has actually happened, *calomel,* to the extent of permitting the mouth to be made slightly tender, is believed by not a few to encourage the absorption of the fluid. It can only, however, be given in healthy subjects, free from gouty or renal disease; and Dr. Maclachlan recommends that it may be given in the form of *three* or *four* grains of blue pill and two of James's powder every evening for ten days or a fortnight, the effects being carefully watched, so that the gums should not be more than touched.

To allay the pains in the palsied limbs *opiates* are of great value. They procure sleep and moderate tetanic rigidity of the flexor muscles. *Muriate of morphia* may be given in doses of one-sixth to one-fourth of a grain at bedtime; and the doses may after a time be increased, if necessary; and may be combined with extract of colocynth or croton oil, to counteract the tendency to constipation (MACLACHLAN, l. c., pp. 195, 196).

In chronic cases local bleedings from the temples, or from behind the ears, combined with moderate purgatives every three or four days, are demanded in most cases, with a blister applied now and again to the nape of the neck; and as much as possible the currents of blood must be diverted from the brain. Headache and stupor are generally relieved by full feculent evacuations. The diet ought to be strictly that of a vegetarian, and as little stimulant as possible (WOOD).

In the more chronic, and although ultimately fatal, forms of the disease, life is evidently prolonged by mild tonics, attention to the bowels, and by a liberal and nutritious diet, with such a graduated allowance of alcoholic beverages as the case may require. Beyond this the medical treatment of ramollisement of the brain is still a problem, with only a few unsure data to guide us for its solution.

YELLOW SOFTENING OF THE BRAIN.

Latin Eq., *Cerebrum fluidum flavens;* French Eq., *Ramollissement jaune;* German Eq., *Gelbe Erweichung (des Gehirns);* Italian Eq., *Rammollimento giallo.*

Definition.—*Yellow softening of the brain, as a result of death of a portion of its tissue.*

Pathology.—This form of softening is usually in the greater hemispheres, in their medullary portion. The lesion may vary in size from that of a bean to that of a hen's egg. In the extreme condition of softening the cerebral substance is changed to a moist gelatinous pulp, varying in colour from greyish white to yellow. The surrounding portion of brain is generally reddish, from dilated vessels or hæmorrhagic infarction. The yellow colour of the softer portion generally depends on the changes which go on in the blood extravasated in such capillary hæmorrhages. The softened and disintegrated brain-substance becomes charged with the colouring matter of the blood; eventually the lesion is converted into an areolar net-work, filled with a yellow chalky milk-like fluid.

The medullary structure above the lateral ventricles is very often the seat of this species of lesion. When the softening approaches near the external surface of the hemispheres there is usually flattening of the convolutions, with a sensation of pulpiness or fluctuation beneath.

This yellow softening is in reality a form of gangrene.

Symptoms.—It comes on suddenly, and as if without any preliminary inflammatory or hæmorrhagic stage, and at once proceeds to complete disorganization of a portion of the brain, which so dies. The entire duration of this form of softening may not exceed three or four days. The effects of the several forms of softening on the system are not well defined, and cannot be considered as in any way distinctive, so that those already indicated embrace all that can be detailed. Softening in the *crura* is generally speedily fatal, death usually taking place within two or three days, and is rarely prolonged to the sixth day. Within the substance of the *medulla oblongata* it is still more rapidly fatal.

Much confusion exists regarding the nature of the several softenings of the brain and spinal cord. It has always been difficult practically to distinguish one kind of softening from another. Professor J. H. Bennett, of Edinburgh, describes six conditions under which softening—as seen *post-mortem*—may arise, as follow:—

(1.) *Exudation or inflammatory softening,* from exudation which has been infiltrated among the elementary nerve structures.

(2.) *Hæmorrhagic softening,* from a mechanical breaking up of the nerve texture by hæmorrhagic extravasations, either in large masses or infiltration by small isolated points.

(3.) *True fatty softening,* from fatty degeneration of the nerve-cells, independent of exudation.

(4.) *Serous or dropsical softening,* from the imbibition of serum, which loosens the connection between the nerve-tubes and cells.

(5.) *Mechanical softening*, from violence in exposing the nerve-centres.

(6.) *Putrefactive softening*, occurring especially in warm weather.

Only the first three, and perhaps also the fourth, may occur during life.

Dr. Craigie considers softening of the brain to succeed at least four morbid states, as follow:—

(1.) The consequence of the blood-stroke (*coup de sang*).

(2.) The consequence of effusion of red blood, which separates and breaks down the delicate texture of the part in which it is effused.

(3.) As a consequence of the process which accompanies or follows hydrocephalus.

(4.) It may take place in the cerebral substance surrounding tumors. Whatever therefore may be, the circumstances under which softening is developed during life, or the form it assumes after death, the lesion is not so much a substantive disease as the effects of a morbid process taking place under several different conditions of the brain.

Diagnosis.—Whenever *hemiplegia*, complete and absolute, occurs *suddenly*, *without* loss of consciousness, softening of the brain may be diagnosed, and that the softening is of the *colourless kind* (RÉCAMIER, TROUSSEAU, TODD). Whenever, on the contrary, complete loss of motor power is attended by loss of consciousness, especially when coma is sudden, *hæmorrhage* may be diagnosed, and that to a considerable amount. When the intellect is effaced to some extent, but not entirely,—when there is obtuseness but not complete loss of sensibility,—whilst there is absolute loss of motor power, *capillary hæmorrhage* co-exists with softening (RÉCAMIER, TROUSSEAU).

Treatment must be by restoratives and food, and perhaps wine. Blood-letting and lowering treatment are now not to be thought of.

ABSCESS OF THE BRAIN.

LATIN EQ., *Abscessus cerebri;* FRENCH EQ., *Abscès du cerveau;* GERMAN EQ., *Abscess des Gehirns;* ITALIAN EQ., *Ascesso.*

Definition.—*Suppurative inflammation of the brain, terminating in a collection of pus.*

Pathology.—Collections of purulent matter have often been found in the substance of the brain (*Apostema cerebri*).

Suppuration of the brain occurs in three forms, and in by far the greater number of cases no trace of redness can be seen in any part of the brain; and probably this want of vascularity may account for the rare development of pus-corpuscles in the fluid of what appears to be abscesses, and which otherwise looks like puss. Pus is not always present in undeniable abscesses of the brain, but molecular granules, exudation-corpuscles, and pyoid bodies, evidently the result of the exudation-process, are generally the sole objects which the microscope can detect (LEBERT, BENNETT). Exudation in the softened state of the brain-substance, resembling pus, may be thus—(1.) Collected into the form of an abscess; (2.) It may be infiltrated into the substance of the brain; or (3.) It may be detected on the convolutions, in the shape of a ragged ulcer, varying in size from a fourpenny-piece, or less, to that of half-a-crown. It is in this latter form and site of the suppurative process that well-formed pus-corpuscles can most frequently be detected.

In abscess of the brain the surrounding substance is generally of the natural colour, except in a very few cases in which it succeeds to apoplectic effusion, when the walls of the cavities are dyed by the previously extravasated blood. Dr. Baillie says, when the abscess is of large size, the weight of the pus breaks down the neighbouring parts, and they look simply as if they had been destroyed, or very much injured by the pressure; and also when the abscesses are small there is an ulcerated appearance of the cavity in which the pus is contained. In other cases the usual membrane of an abscess forms. This membrane is at first extremely delicate and easily torn; but as the patient continues to live, the membrane lining the abscess becomes of greater consistency, and even of considerable density, so that in some cases it is fibrous, fibro-cartilaginous, and even ossified, thus laying the basis for the formation of bony tumors of the brain.

Recent abscesses of the brain form irregular round cavities, filled with yellow, greyish, or reddish thick fluid; and the walls of the abscess consist of ragged masses infiltrated with pus. In the immediate vicinity of the abscess there is usually inflammatory softening, and beyond that *œdema* of the brain.

Such abscesses increase till they open into a ventricle, or reaching the meninges, extensive meningitis may occur. In other instances ulceration may take place through the cerebral membranes as far as the calvarium or skull bones. The ulcer may perforate the bone, or open into the cavity of the tympanum. If the ulcers be incorporated by new connective tissue formations, they have a regular form and smooth walls. If it has lasted a long time, the corpuscle is dense, and the contents thickened by re-absorption of the fluid part, the remainder being converted into a cheesy chalk-like mass (NIEMYER).

Of eighty instances of abscess of the brain, collected by Professor Lebert, twenty-two of them, or above a quarter, were cases of scattered abscesses in various parts of the brain; the remaining fifty-eight were cases in which solitary abscesses were found in some part of the encephalon, distributed as follows:—

Left hemisphere,.........	in 23 cases.	Cerebellum, ...	in 12 cases.
Right ,, ...	in 18 ,,	Pituitory body,...........	in 2 ,,
Corpora striata, ...	in 2 ,,	*Medulla oblongata*,......	in 1 case.

In the case of multiple abscesses there were never more than five, sometimes three or two only.

The abscesses generally occupy the white substance, and only affect the grey matter by extension. In form the abscesses are generally oval, and vary in size from a pea to the size of a hen's egg or larger; so that one entire hemisphere has been found converted into a pouch filled with pus. The pus is generally of a greenish colour, of considerable density, and rarely containing blood. Sometimes it is very fetid.

It is generally granular, not containing many well-formed corpuscles; and the older the abscess the more the pus-corpuscles are found to be degenerating. Pus is generally found infiltrating the cerebral tissue surrounding the abscess; and in the zone beyond, the tissue is softened, while, if the process be recent, a zone of vascular redness surrounds the whole. Connective tissue ultimately grows in a condensed form round the site of abscess, and closes the whole with walls of considerable thickness, (1 to 4 *millim.*) Often these walls are very vascular. There does not seem to be any evidence that these encysted abscesses are ever cured.

The lateral ventricles are the parts into which the abscesses most frequently burst. Thickening of the *ependyma* follows, and scattered spots of inflammatory softening may be found in the neighbouring tissue. In some cases the abscess makes its way outwards through the petrous portion of the temporal bone or the aural passages.

The most frequent cause of cerebral abscesses is internal *otitis*—a lesion which often results from *scarlet fever, typhus fever, small-pox, measles,* or *scrofula.* The ear affection which causes the discharge is usually either (1) disease of the *tympanic* cavity or of the *dura mater* investing the temporal bone. The inflammation which terminates in abscess of the cerebral substance is usually the effect of inflammation of the membranes, and in some instances of the discharge being suddenly checked, and the chronic external inflammation being suddenly converted into an acute internal disease. The inflammation is generally observed to succeed quickly the suppression or disappearance of the external discharge. It is generally observed in cases of scrofula (Craigie). Abscesses also occur as the sequelæ of inflammations of distant parts, as of *pneumonia pericarditis, enteritis.* They may also occur as so-called metastatic abscesses, *pyœmia,* and as a result of traumatic injury.

The symptoms of abscess of the brain are likewise extremely obscure. In a case treated for disease of the nose, the man made no complaint of his head, and was able to sit up in bed, and to assist himself in every way. He died suddenly in the night. An abscess of considerable size was found in the left hemisphere above the ventricle. In other cases pain, delirium, coma, palsy, and sometimes convulsions, were the symptoms observed. The convulsions were observed most frequently to occur when the abscess formed in the *tuberculum annulare* or in the *medulla oblongata,* or so close to these structures that the growth of pus would affect their functions simply by its pressure (Baillie, *Morbid Anatomy,* p. 450).

The latent character of brain abscess is important in diagnosis. Sudden headache is the symptom which most frequently excites attention; and it is generally accompanied by febrile symptoms and vomiting. The patients become heavy, morose, and may be delirious, with contraction of the pupils and photophobia. Difficult articulation, numbness, formication, and convulsive attacks may supervene. While the intellect may suffer comparatively little, sensibility suffers more frequently; and headache, generally at first diffused, is more or less intense, and subsequently becomes unilateral. Coma occurs frequently, is often temporary, and paralysis occurs in about one-half the cases. The paralysis is generally local; but it may assume the form of general muscular debility. The duration of such cases fluctuates from two or three weeks to two months (LEBERT, SIEVEKING, in *Med.-Chir. Review*, 1857, p. 526).

Schott has analyzed forty cases of abscess of the brain. He finds *otitis*, *pyœmia*, and injuries to be the most frequent causes. In cases of *otitis* the abscess formed mostly in an imperceptible manner; and in cases of injury the symptoms were very transitory. Encephalitis was present in acute cases. (*New Syden. Soc. Year Book*, 1862, p. 79).

APOPLEXY.

LATIN EQ., *Apoplexia;* FRENCH EQ., *Apoplexie;* GERMAN EQ., *Apoplexie* —Syn., *Hirnschlag;* ITALIAN EQ., *Apoplessia.*

Definition.—*A disease essentially characterized by the sudden loss, more or less complete, of Volition, Perception, Sensation, and Motion, depending on sudden pressure upon the brain (the tissue of which may be morbid), originating within the cranium.*

Pathology.—The literal meaning of the term *apoplexy* conveys the idea of a *sudden stroke;* and it has been usual to confine the term to the results produced by extravasations of blood into the nervous tissue of the brain, a portion of which is thus destroyed. More comprehensive pathological doctrines teach us to give a wider signification to the term. It is now used to characterize a group of symptoms irrespectively of the anatomical conditions upon which they may depend. These symptoms consist of—(1.) Premonitory warnings, extending over variable periods (seconds, weeks, months, or years), marked by sundry derangements of the nervous functions, such as loss of memory, dulness of sensation, or diminished power; (2.) The individual is more or less suddenly deprived of volition and perception in their relation to sensation and motion. Consciousness is thus more or less lost, and paralysis is more or less complete. The patient may fall to the ground completely insensible, or he may only stagger and cling to some object for support. The respiration and circulation may be unaffected, or the former may be stertorous and the latter laboured. Some group of muscles, a side of the body, or the whole body, is paralyzed, flaccid, motionless; or it may be rigid with tonic, or convulsed with clonic spasm. From this state the patient may never recover. Life becomes gradually extinguished, or the sufferer may recover partially or entirely; in the former case leaving some mental, motorial, or sensorial faculty impaired for weeks, or for the whole of after-life.

The essential phenomena of an apoplectic seizure consist in the severance of the brain-functions, namely, volition and perception, from motion and sensation: the other symptoms that occur are additional phenomena, depending on secondary changes subsequently induced in the part, or its vicinity, which has been the primary seat of lesion. These essential phenomena of the *apoplectic state* are found to be due to a variety of local lesions, or complex morbid states, and not to any constant lesion. One or more of the following local lesions or complex morbid states may induce the apoplectic condition, namely,—

(1.) *Congestion of the brain*, or what is commonly called *determination of blood to the head—congestive apoplexy.*

(2.) *Hæmorrhage*, or *extravasation* (a.) *into the substance of the hemispheres* or *cerebellum*, (b.) *into the ventricles*, or (c.) *into the arachnoid cavity.*

One or other of these lesions constitutes apoplexy in the common acceptation of the term—*sanguineous apoplexy* or *cerebral hæmorrhage.*

(3.) *Sudden serous effusion in large quantity* is equally efficient in bringing about the apoplectic state commonly called *serous apoplexy.*

(4.) *Local cerebritis*, or *softening of the brain*, produces, as already shown, symptoms, in the first instance, of an apoplectic kind. So also do

(5.) *Tumors of the brain*, or *meningitis.*

(6.) *Tuberculous meningitis.*

(7.) *The progress of various specific and constitutional diseases from blood-poisoning.*

(8.) *Anæmia*, as in the *hydrocephaloid disease of children, disease of the heart, and vascular obstructions.*

To the phenomena produced by the first three of these conditions only has it been common or usual to apply the term *apoplexy.*

This disease was well known in the Greek and Roman schools of medicine, and is of too frequent occurrence, and of too striking a character, to have escaped observation even in the rudest ages of society. Patients have died with undoubted apoplectic symptoms when nothing has been found but congestion of the vessels of the scalp, of the membranes of the brain, and of the brain itself, but without the extravasation of a particle of blood. More generally, however, a greater or less quantity of blood has been effused either into the cavity of the arachnoid, into the substance of the brain, or into some of the ventricular cavities. Thus it is that the lesions found in cases which die of undoubted apoplectic symptoms vary much. Sometimes the evidently congested state of the brain during life leaves no trace visible after death. Such cases have been described as "*nervous apoplexy*" (SANDRAS).

Although much stress is laid upon the fact that "a sudden pressure upon the brain is necessary to produce the apoplectic state," yet it is difficult in all cases to account for the proximate cause of the disease by such an explanation; for when the quantity of blood extravasated has been not larger, for instance, than a barley-corn, it is difficult to account for all the phenomena by mere pressure. The ideas or theories which have thus been formed to account for the apoplectic state may be shortly stated as follows:—

(1.) The result of sudden pressure, effected by causes within the cranium.

(2.) From a peculiar morbid state of the nervous matter of the brain, and which predisposes to the extravasation of blood (DRS. ROBERT WILLIAMS, WOOD, SIEVEKING)—an apoplectic orgasm, as it has been called by some, but which is probably of the nature of softening (ROCHOUX).

(3.) A morbid condition of nerve-matter and minute blood-vessels, also probably of the blood itself (BOUILLAUD, PAGET), and in connection with chronic renal disease (BRIGHT, BURROWS, CHRISTISON, KIRKES).

Cerebral hæmorrhage generally takes place from the smaller arteries or capillaries of the brain, and there is generally structural disease of the arterial walls, as well as a morbid condition of the brain-substance surrounding the diseased vessels, combined with increased pressure of blood from some temporary cause—temporary plethora—such as occurs during prolonged and luxurious meals.

When the quantity of blood extravasated is small, the disease is seldom fatal from the first attack; but the rapidity of the fatal issue appears to bear some relation to the vicinity of the hæmorrhage to the *medulla oblongata;* and the effusion of blood into the ventricles is also generally most rapidly fatal. But in all cases impairment of general nutrition is the result of injury to the brain from apoplexy; and a gradual diminution of mental power is seen in almost all apoplectic cases, corresponding to a general atrophy of the brain; and degeneration extends into the spinal medulla of the filaments communicating with the apoplectic effusion (TÜRK).

In examining apoplectic cases it is not unusual to find a cavity scarcely bigger than a barley-corn in the substance of the brain, the evidence of the primary attack, and containing a clot of blood variously changed. If the blood be effused among the membranes, it may be altogether absorbed, and not a trace of disease be found. In severe cases still greater quantities of blood are effused; and if the apoplexy destroy the patient in a few minutes or a few hours, the quantity of blood effused will sometimes fill the whole cavity of the arachnoid, or extensively rupture the substance of the brain, forming a cavity as large as a nut or an egg, or even lay the ventricles into one cavity.

It is rare that sanguineous effusion occupies both cerebral lobes, or the whole extent of the membranes of the brain, although such instances are occasionally seen. More commonly it is limited to the substance of one hemisphere, or to the membranes covering it, or to the cavity of a ventricle. When the membranes of the brain are affected, the more immediate seat of the hæmorrhage is usually that portion covering the convexity of the brain. This varies, however, so that the portion covering the base, or that investing the cerebellum, or, indeed, any other part, may be its seat.

The superficial membranes of the brain are not the only membranes of that organ which are the seat of apoplectic effusion. Hæmorrhage may take place from the membrane lining the ventricles, and which sometimes bleeds so profusely as not only to fill the lateral ventricles, but even to enlarge their cavities. As death in these severe cases is usually sudden, the walls of the ventricles are generally healthy; but in some very few instances the *septum lucidum* has been found ruptured, and the ventricles have freely communicated. The smaller ventricles are in a very few instances also the seat of apoplectic effusion. Dr. Abercrombie gives a case in which the third and fourth ventricles were filled with blood. The patient was not at first insensible, but gradually became so, and died in a few hours.

The appearance of the blood effused into the membranes of the brain varies according to the time which elapses before the patient dies, thus affording opportunity for its examination. If that event takes place in a few hours after the attack, the blood is still fluid, or is found in black clots, while the membranes, except being infiltrated with blood, are as yet healthy. The substance of the brain, likewise, has no other appearance of disease than that of being flattened, from the presence of the extravasated blood. If the patient, however, survives a few days, the membranes show marks of inflammatory action. They are injected, thickened, and although dry and pitchy-like in the immediate neighbourhood of the clot, have yet some serum effused in other parts of the space they enclose. The convolutions of the affected part of the brain are likewise now not only flattened but softened. Thus various processes immediately commence in the blood after it is extravasated, as well as an interchange of processes between the blood and the surrounding parts. The most obvious of these phenomena are the formation of a coagulum or clot; its solution, and the formation of blood-crystals; the gradual absorption of the more fluid constituents; the formation of an organized membrane round the clot; continuous absorption of the exudation; induration of the surrounding cerebral parts; contraction of the cavity, and ultimately the formation of a cicatrix. These changes are effected with various degrees of rapidity, depending on the site and extent of the extravasations, the healthy state of the nervous texture, and of the patient constitutionally.

When effusion has taken place into the substance of the brain, if the patient has died in the fit, or shortly after, the hæmorrhagic cavity is found filled with half-coagulated blood, its walls irregularly softened, and dyed to the extent of some lines deep with the colouring matter of the blood; and a small stream of water directed upon this part at once removes the extravasated blood, and also a layer of softened cerebral matter. Again, if the patient has survived a week, the blood is found coagulated, and the serum set free; but the presence of the clot has caused inflammation, so that the walls of the cavity are not only discoloured, but more decidedly softened, and are softer in proportion as they are nearer the clot. If life be prolonged till the fifteenth day, the serosity is absorbed, but the walls of the cavity are still of a deep red. About the seventeenth day Virchow has discovered blood-crystals, or hæmatoid crystals, in the cavity. These blood-crystals were first discovered by Sir Everard Home, and have been more recently described, and their nature explained, by Funke, Kunde, Lehmann, Beale, Parkes, and Sieveking. It appears that these crystals do not form from clotted blood until the blood-corpuscles have become ruptured by endosmosis. Their contents then escape and crystallize as the solution gradually becomes concentrated (Beale). Thus another guide to the age of the clot exists in the presence of these crystals. About the thirtieth day, if the patient lives so long, the clot is isolated, and a membrane forms, by the new formation of connective tissue growing from the neuroglia. At first this membrane is muciform, fragile, intermixed with particles of cerebral matter, and also with some of the colouring matter of the clot; but by degrees it becomes more consistent, thick, and hard, and encloses the clot, which diminishes, and some serum is probably secreted by the new membrane surrounding it. Eventually a new formation of delicate connective tissue, coloured yellow by the pigment of the clot and by serous infiltration, covers the walls of the clot-cyst, and breaks up the clot into a fine net-work. The cyst has also been found fully formed, organized, and nearly empty, by the thirteenth day (MACINTYRE); and by the seventeenth day after extravasation it has been found to contain sanguineous fluid (MOULIN, SIEVEKING). The cerebral walls surrounding the cyst, previously softened, now become indurated, and are stained yellow, from the usual changes which the extravasated blood with which they are penetrated undergoes—a colour, however, which they ultimately lose. The cavity thus formed may be filled at length with serum only; or, the serum being absorbed, the membranous cyst may ossify, and may be thus converted into a bony tumor. At other times the opposite sides of the cavity unite by a kind of areolar membrane, enclosing pigment, which thus forms a species of apoplectic cicatrix, but possessing so little power of conducting nervous influence that the patient seldom recovers from his palsy. Such is a short outline of the effects of hæmorrhage into the substance of the brain. The size of an apoplectic cavity varies from a barley-corn to that of an egg, and their number is as variable as their extent. Sometimes we find but one, sometimes two, and in a very few instances three or more cavities. When many apoplectic cavities exist in the brain, it is rare to find them all in the same state. Some are old and almost obliterated, others are fresher, and others again quite recent, their different stages marking a distinct and different period of attack. An account of a most interesting case of this nature is given by Dr. Fuller, in which there were *six* clots, each of a different date, and in different stages of discoloration, and corresponding to each of six well-marked apoplectic seizures in the course of nine months (*Diseases of the Chest*, p. 602).

Some of the most exact data we possess regarding the pathology of apoplectic seizures are to be found in the *Transactions of the Pathological Society of London.*

In *fourteen* cases in which lesions of the brain connected with apoplectic seizures have been shown at the Society, the position of the blood effused was as follows:—(1.) Superficial meningeal effusions, five cases; (2.) Within the substance of the hemispheres or the *central ganglia*, four cases; (3.) In the *pons Varolii*, two cases; (4.) In the *pons Varolii* and *crura*, one case; (5.) In the *pons Varolii* and *cerebellum*, one case; (6.) In the *pons*, *crura*, and *cerebellum*, one case.

Superficial or *ventricular* extravasation occurred in five cases; that is, the blood lay in a more or less coagulated mass under the arachnoid over the hemispheres, and was most abundant at the base and about the roots of the nerves. In two of the cases there was some evidence of previous *meningeal* disturbance. This evidence consisted of adhesion of the cerebral arachnoid and pia mater to the cerebral convolutions along the anterior margin of the middle line of the hemisphere: the history of the other case recorded the occurrence of two previous apoplectic seizures. In it *post-mortem* evidence of previous irritation also existed, in the adhesion of the *dura mater* to the calvarium.

The conditions as to general health of those in whom these attacks occurred were as follow:—One was convalescent from a uterine disease, the nature of which is not stated; another had chronic bronchitis and asthma; in a third intemperance was predominant; and in the fourth there had been two previous attacks of apoplexy. In three of these cases the condition of the heart is recorded as being larger than natural —in one of them weighing 11¾ ounces, in another 15 ounces, where the hypertrophy was general. In the case of the repeated attacks the heart was fatty, as well as the coronary arteries and those of the pia mater. The kidneys also were fatty. In all of them the attack was sudden—followed in one by instantaneous death, in the others by insensibility, collapse, and blanched surface. In one case the pupils were dilated; in another, where the *septum lucidum*, *corpora striata*, and *optic thalami* were broken down, there were paralysis and rigid contraction of the right arm. Death followed in thirty-four hours; another died in twenty-two hours; one on the third day, and another on the fourth. In none of these cases is there noticed any lesion of the brain-substance itself. In all, the hæmorrhage appeared to proceed from the tomentose or vascular surface of the pia mater. In this variety of meningeal hæmorrhage the vessels of the pia mater become inordinately injected, and the effusion consisted of blood, or merely blood-serum (DR. WILLIAMS). While this effusion of blood or serum lies on the convoluted surface generally, it occasionally, as in this instance, is effused into the ventricles, so that the brain was compressed both from the peripheral or pericranial surface and from the ventricular or internal surface. There was no connection of these cases with external injury, the most common cause of extravasation between the pia mater and brain. They all appear to have been the result of morbid states of the vessels of the pia mater. Cases of this kind are recorded by Craigie in the *Edinburgh Medical and Surgical Journal*, vol. xviii., p. 487; also by Morgagni, *Epist.* iii., 2 and 4, quoted by him from Valsalva, *Epist.* xi., 19 (see Craigie's *Pathological Anatomy*, p. 730).

Extravasation into the Substance of the Hemispheres or Central Ganglia.—In four cases some part of the cerebral substance contained the extravasated blood, more or less free, or surrounded by a cyst. In two of the cases the clot was surrounded by a cyst, in one of which, death taking place thirteen days after the seizure, the cyst was found nearly empty, disintegration and absorption having thus early taken place (MACINTYRE). These cysts seem to be formed of fibrilloid tissue like condensed fibrin, more or less hyaline, or stained with colouring matter of the blood; and in one case, where death occurred twelve weeks after the fit, blood-crystals were found in abundance in those parts of the wall of the cyst, and most abundantly on the yellow parts. In this case, at the end of twelve weeks, the cyst was filled with slightly turbid yellow fluid, a small portion of coagulum only remaining (BRISTOWE). In the case where the extravasation occurred into the *corpus striatum* it protruded into the lateral ventricle of the right side, and nearly filled it, and the nervous substance around was broken down and soft. Death occurred in half an hour (GIBB). The position of the clot in the fourth case was above the lateral ventricles, separated from the longitudinal fissure only by the grey matter. Some ecchymosis existed in the neighbourhood, and there was yellow discolouration on the surface of the left *corpus striatum,* where two cysts existed with fibrous walls, the remains of old extravasations (OGLE).

The conditions as to general health of the patients are not stated. One case occurred during convalescence from an attack of *delirium tremens,* after having been apparently benefited by a dose of morphia amounting to *one* grain of the *acetate.* There was extensive cardiac, pulmonary, and gastric disease in this case, and the patient lived thirteen days after the seizure. A bloated florid look were the only circumstances to indicate the general state of the case where the extravasation occurred in the hemisphere above the lateral ventricle. In three of the cases the heart is specially noted as fatty, its weight in one instance being thirteen ounces, in which case, also, the liver was enormously enlarged, containing sugar. In all the cases the arteries at the base of the brain were diseased. The symptoms were generally those of a fit, in one case like syncope, when the extravasation was into the ventricles and *corpora striata,* death following in half an hour, preceded by a feeble pulse, the pupils being slightly dilated. In the other case, in which there was *post-mortem* evidence of previous extravasation by the remains of cysts on the surface of the left *corpus striatum,* together with a thickened and opaque arachnoid with sub-arachnoid fluid, a fit occurred with paralysis of right side, a feeble and quick pulse, followed by convulsions, contracted pupils, and incoherent speech, breathing becoming stertorous, death following by coma on the sixteenth day.

Extravasation into the Pons Varolii and other parts.—Five cases are noted in which the extravasation took place into the *pons Varolii.* In two of the instances the *pons* alone was implicated. In one, the *pons* together with the *crura cerebri;* in another, the *pons* together with the *cerebellum;* and in a third, the *pons* together with the *crura* and *cerebellum* were implicated. Of the cases where the *pons* alone was implicated, a clot the size of a pea occupied its centre. It occurred in a lunatic, taking his walk out of doors in his usual health. Sudden paralysis indicated the seizure, and death followed in twenty-four hours. In the other case (age thirty-four) the *pons* contained a clot as large as a filbert shell, seated in the middle line, the blood having burst through into the fourth ventricle. Surrounding the clot the nerve-tissue was softened and shreddy. The seizure was sudden, with right hemiplegia and anæsthesia. Paralysis of the left side remained, with feeble circulation, and death followed in five days. In the instance in which the *crura* as well as the *pons* was affected, the blood was partially coagulated, breaking up the nervous matter, and distending the parts into a bag. In the instance in which the *crura* and *cerebellum* as well as the *pons* were implicated, the clot was similarly enclosed in a bag of nervous matter. In the other instance the lesions in the *pons* and *cerebellum* were confined to the left side.

These cases were distinguished by rapid and extensively increasing difficulty of breathing—in two amounting to *stertor*—and by contraction of the pupil; features placid; convulsions of pectoral muscles; coma becoming more and more perfect; and death followed in from four to twelve hours. In three of the cases extensive disease of the vessels, both large and small, especially towards the brain, is noted.

These cases have been related, from the supposed rarity of the lesion as to its seat. And although the greater number of the cases are deplorably meagre in the details recorded in the Society's *Transactions*, yet they are exceedingly interesting on the whole, when thus classified, establishing as they do several facts, not accurately determined before, relating to the pathology of cerebral apoplexy or cerebral hæmorrhage. They confirm the observation that, when the extravasation is extensive, although superficial, the result is rapidly fatal by the coma, resulting from compression over a large surface; that when effusion is sudden and extensive in *certain parts* of the brain, such as in the *corpora striata*, or *optic thalami*, or the *pons Varolii*, the result is rapid death; and generally they confirm the belief that the more nearly the extravasations approach the *medulla oblongata*, the more rapidly fatal is the result. The symptoms which were associated with irritation of the parts connected with the *pons Varolii* were peculiarly well marked in three instances. So delicate are the sensibilities of these parts in the exercise of their functions, that it has been taught that blood is rarely found effused in them as a lesion after death, because simple vascular injection of such parts was considered sufficient to disturb the functions of the brain so much as to produce the fatal result before such changes can be effected, (Craigie's *Prac. of Physic*, p. 309, vol. ii.)

The cases are much too few to found any general conclusion upon relative to the numerical frequency of lesion in any particular part of the brain, associated with the apoplectic seizure.

Site of Effusion.	Andral.	* Tacheron.	† Serres.	‡ Lerminier.	Path. Soc. of London.	Total.
Level of corpora striata and into them,	202	...	...	...	...	202
Corpora striata, ...	61	3	...	1	1	66
Thalami optici, ...	35	2	...	2	...	39
Portions of hemisphere above centrum ovale, ...	27	9	2	3	3	44
Lateral lobes of cerebellum, . .	16	...	...	...	...	16
Before corpora striata, . . .	10	...	...	...	...	10
Mesocephalon, ...	9	1	2	...	...	12
Spinal cord, ...	8	...	...	...	...	8
Posterior lobes, ...	7	2	2	...	...	11
Middle lobe of cerebellum, . .	5	...	...	...	...	5
Peduncle of brain, ...	3	...	...	...	...	3
Peduncle of cerebellum, . . .	1	...	...	...	...	1
Corpora olivaria, ...	1	...	...	...	...	1
Pituitary gland, ...	1	...	...	...	...	1
Pons Varolii, ...	...	2	3	...	5	10
Meningeal hæmorrhage, . . .	...	...	...	...	5	5
TOTAL, . .	386	19	9	6	14	444

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* *Recherches Anatom. Pathog.*, par C. F. Tacheron, tome XIII., Paris, 1823, Ordre XIV., An. 31.

† *Annuaire Med.-Chir.*, p. 324, sect. XI.

‡ Ibid., p. 213.

It is well established (notwithstanding statements to the contrary, founded on experiments of a certain kind) that the brain-substance is liable to compression from vascular injection generally, and increased turgidity of its vessels. There are also parts of the brain so constituted anatomically that hæmorrhage more readily occurs in them than in other parts. The anatomical constitution which favours this consists in the increased provision for the transit of blood-vessels. For instance, through the white perforated spot at the commencement of the fissure of Sylvius, the sylvian or middle artery of the cerebrum sends its numerous branches of various size into the substance of the brain. These in the first instance penetrate the *corpus striatum*, which lies immediately over this *anterior perforated spot*. The *corpus striatum*, and the parts at its level, from their anatomical position and relations, are thus the most vascular parts of the whole brain; and most pathologists agree in considering these to be the parts most liable in the substance of that organ to effusion of blood, more especially when the vascular system of the brain is overloaded (BONETUS, MORGAGNI, ROCHOUX, ANDRAL, CRAIGIE. Opposed to this belief were—HOWSHIP, LERMINIER, SERRES, TACHERON). A summary of the facts recorded may be stated, as in the preceding table, page 184.

The result of these observations shows that the comparative liability of parts of the brain to extravasations of blood may be stated in the following order, commencing with the parts most frequently implicated:—

(1.) *Corpus striatum, opticus thalamus*, and hemisphere at the level of these parts.
(2.) The *corpus striatum* alone.
(3.) The hemisphere above the *centrum ovale.*
(4.) The *thalamus opticus* alone.
(5.) The lateral lobes of *cerebellum.*
(6.) The *mesocephalon.*
(7.) Posterior lobe of *cerebrum.*
(8.) Before the *corp. striat.*
(9.) *Pons Varolii.*
(10.) Middle lobe of *cerebellum;* meningeal hæmorrhage.
(11.) Peduncles and olivary body.

Much more extensive records, however, are required to determine these points accurately; and Rokitansky does not consider as yet that we can state such a result in other than general terms.

The ages of those whose cases are recorded varied from thirty-three to sixty-nine, the average age being fifty-four. The disease is known to occur even in children.

Circumstances under which the Lesion occurred.—In two of the cases the arteries are stated to have been healthy. In three of the cases no mention is made in regard to them; but in three there was hypertrophy of the heart, amounting in one case to fifteen ounces. In all the others (nine in number) the arteries were in a diseased condition; and in seven cases there was evidence of concurrent disease in other organs.

Thus far these cases rather tend to confirm the observation of Paget in 1850—namely, that sudden death from apoplexy is most generally associated with fatty degeneration of the minute cerebral blood-vessels;

while, at the same time, the associated morbid states of other organs are such as to lead to the belief of a more general morbid state, and perhaps more especially of the nervous matter—an apoplectic orgasm which predisposes to the extravasation of blood from diseased blood-vessels—the belief now most generally entertained regarding the nature of the apoplectic lesion.

Another interesting feature in the pathology of the lesions demonstrated to the Society is the organization of a wall round the extravasation, and the future changes of the blood-clot, as shown by its examination at various periods after the extravasation. Cysts existed in two of the cases, in one of which, on the thirteenth day after the seizure, the cyst was found nearly empty, the absorption having thus early taken place (MACINTYRE). On the other hand, again, at the end of twelve weeks a portion of coagulum still remained enclosed in a cyst, and blood-crystals existed in abundance on its walls (BRISTOWE). Even at a still longer interval bloody fluid has been found in a cyst otherwise nearly empty, as recorded by a French physician of the name of Moulin, who mentions such a morbid state existing seventeen years after the extravasation (SIEVEKING). The age of the clot may thus, in some measure, be indicated by the cyst, the condition of its contents, and especially as to blood-crystals. Virchow has recorded their existence in the cavity by the seventeenth day; and as shown by Beale, they do not form from clotted blood until the blood-corpuscles become ruptured by endosmosis. The contents of the blood-cells then escape and crystallize, as the solution gradually becomes concentrated.

Symptoms.—Whatever may be the pathological doctrines taught regarding the morbid state of the cerebral parts in apoplectic states, we are able practically during life to do little more than merely recognize the apoplectic state itself. The diagnosis between the *congestive*, the *hæmorrhagic*, and the *serous* or merely morbid nervous state can only be arrived at approximatively by a careful comparison of symptoms closely observed, such as are detailed in the treatise of Dr. Russell Reynolds, already noticed, and from which the following statements are condensed:—

I.—**Symptoms of Apoplexy from Congestion.**—The face, scalp, and conjunctivæ are increased in vascularity; the skin generally is of a dusky venous hue, and the surface is warm. There is fulness of the jugular veins, with increased pulsation in the carotids. The tongue is foul, and nausea prevails, with constipated bowels. Respiration and the pulse are both laboured, and the extremities are cold. Such are some of the general symptoms which indicate the approach of an attack of the congestive form of apoplexy.

The symptoms peculiar to the brain itself are,—

1. *Mental.*—The activity and power of the intellect are diminished. General confusion of thought prevails, with deficient memory. Any attempt at mental exercise increases the expression of these signs; so does the recumbent position and emotional disturbance. Sleepiness, also, with laboured respiration, is common, especially after meals; and there is a general tendency to inaction of body as well as of mind—a "not-to-be-disturbed" sort of desire is experienced. Such mental pheno-

mena, however, are not permanent; and while there is a readily induced state of general confusion, there is no persistent, special, or permanent loss of power of intellect.

2. *Sensorial.*—The senses generally are obtuse. The hearing is dull; and heavy rumbling noises are constant auditory illusions. The sight is dim, or *amaurosis* is complete, and often black or variously-coloured spots are seen floating in the field of vision; flashes before the eyes, or other spectral illusions are more or less constant. The patient may see only half of an object, or halves of objects of different colours. Attacks of dizziness also occur, with a sense of fulness and oppression in the head; numbness and weight of the limbs; dull and heavy *cephalalgia.* There may also be occasional feelings of formication and numbness in certain limbs, momentary loss of memory for some words and figures, or temporary paralysis, confined to certain groups of muscles. These symptoms, however, are only of occasional occurrence, and change their localities. While the absence of pain is not now considered of much diagnostic value, yet the occurrence of severe acute pain is generally indicative of something more than congestion.

3. *Motorial Symptoms.*—Little jerkings of the muscles, and irregular or sluggish movements of the eyeballs, are occasional.

These precursory symptoms having generally been more intense for a few minutes or hours, an attack takes place, distinctly apoplectic. These "warnings" which precede an attack of apoplexy may, in some cases, depend on *thrombosis* of small vessels, or on small capillary hæmorrhages. Thrombosis and embolism (lesions to be afterwards described) now take a prominent place in cerebral pathology, both as regards cerebral softening and hæmorhages. By occlusion of a cerebral artery, or of capillaries, clots in blood-vessels are capable of producing apoplectic symptoms, even when the brain-substance is otherwise sound. There is always paralysis of motion in the side opposite to the obstructed vessel, general sensibility, as a rule, being unimpaired; sometimes it may be lessened, but is never altogether absent. Loss of consciousness is also less complete, and the mind is less affected after cerebral embolism than after hæmorrhage. Aphasia sometimes happens. The middle artery of the brain is the most frequent seat of embolism, also the anterior, basilar, and vertebral. Apoplectic symptoms, hemiplegia, and death has also followed plugging of the carotid (HASSE); and of the inominata, right common carotid, and left internal carotid, and middle cerebral (MARKHAM). The seizure commonly occurs during some muscular exertion, such as lifting a heavy weight, pulling on a pair of boots, blowing the nose, straining at defecation, or the like; or even upon a simple change of posture, such as stooping, or suddenly assuming the erect attitude.

The special nervous symptoms of the attack in the *congestive form* are—

1. *Mental.*—Some evidence of the existence of Perception may generally be obtained by loud noises, speaking to the person by name, or pinching him. If, however, Perception is quite extinct for a few seconds or minutes, it soon again partially returns, and there is confusion of Thought, with little Volition as to the direction either of Thought or movement.

2. *Sensorial.*—Except during the first few moments of the attack,

when sensation generally is gone, the changes are slight. Sensation,—indicated at least by reflex action (the limbs being withdrawn if pinched),—is generally present.

3. *Motorial.*—There is more or less paralysis of all the limbs to a slight degree, and for a short time. It is very rare to have either hemiplegia or paraplegia. Short or involuntary evacuations do not occur unless there have been some convulsions. There is no rigidity of the limbs, but clonic spasms are not unfrequent. Generally, and in the course of a few minutes, the symptoms begin to abate rapidly, and they rarely last even for an hour. With the return of consciousness paralysis disappears, and sensibility rarely remains deficient.

The manner of appearance and proportion between the three groups of nervous symptoms is of great diagnostic value; and, as indicative of *congestion*, rather than of *hæmorrhage* or *softening*, any one of the following combinations of groups of symptoms are of importance to be noticed :—

1. *The simultaneous development of the three groups of nervous symptoms. There being either—*

2. *Distinct loss of Perception, profound coma, and general paralysis, without rigidity or convulsion.*

3. *Imperfect loss of Perception, with general paralysis.*

4. *General paralysis, incomplete in degree, and sensation unimpaired, or but little affected;* or—

5. *Paralysis complete in degree, but without stertor or rigidity.*

II. **Symptoms of Apoplexy from Hæmorrhage.**—*A. Into the cerebral substance of the hemispheres.* Very different statements are made as to whether or not premonitory symptoms are present; and the practical point in diagnosis which such discrepancy of statements has taught is, "*that the non-existence of precursory symptoms in a given case is in favour of the belief that hæmorrhage rather than congestion is the cause of the lesion or softening.*

The attack is generally sudden, and rapid in its development. Sometimes after a few hours there may be sudden aggravation of the symptoms, generally due to a renewed extravasation. The patient, if standing, generally falls instantaneously, often with a cry, as if knocked down, and constitutes the "Stroke of Apoplexy," commonly so called. It is the nervous symptoms, however, which are of the most importance.

1. *Mental.*—Loss of Consciousness (of Volition and Perception) is commonly complete at the outset. For a few seconds at least the patient is utterly deprived of intellectual power, which in slight cases partially returns in a few minutes. In severe cases, however, Perception does not return till after some hours, and with vague ideas of things; expressions of Thought are confused, amounting to delirium; *and after the first few minutes or half hour has passed, the degree of intellectual obscuration may be taken as an approximate measure of the amount of extravasation,* although it is to be remembered that there are some exceptional and rare cases on record of hæmorrhagic apoplexy in which the mental faculties were very slightly, if at all, impaired. After some days the intellectual powers are often entirely restored; but in many cases confusion of Thought and partial loss of Memory remain. After recovery

of consciousness the power to *Think* and to *Will* is generally unimpaired, so that if the patient is asked to give the affected hand, they show their desire to fulfil the request by taking the paralysed hand in the sound one, whose nerves and muscles are under the control of the Will in order to accomplish the act (NIEMEYER). If the case does not terminate fatally, the well-marked character of the recovery is a strong presumption that the symptoms resulted from hæmorrhage, and were not due to softening of the cerebral substance.

Destruction also of certain portions of the brain in the *medulla* of the *cerbrum* does not cause any perceptible disturbance of function, small hæmorrhages there may escape recognition during life.

2. *Sensorial.*—Sensibility is usually less commonly affected, and less intensely, than mobility. In most cases with hemiplegia there is *anæsthesia* of half the body; but after a time this usually passes off partially or entirely. When cutaneous anæsthesia is complete, although the surface so affected may be limited in extent, the occurrence indicates severity of lesion. In slight cases there is generally only numbness and tingling of the tips of the fingers. Evidence of Sensation may be obtained when there is no proof of distinct Volition. During the profound coma of the attack at the commencement, the dilated pupil and the half-opened eye indicate that the retina has lost its impressibility; and if hearing and smell are similarly affected, the persistence of such symptoms are signs of evil omen.

3. *Motorial.*—As the *corpus striatum* and the *thalamus opticus,* with the parts of the brain on that level, are the most frequent seats of hæmorrhage, any destruction of those parts or of the *pedunculi cerebri* induces paralysis of the opposite half of the body, called *hemiplegia. Paralysis* is present in the immense majority of cases of apoplexy, its characteristic form being *hemiplegia;* but sometimes it is general, the proportion of cases being as ·84 to ·16 *per cent.* When the paralysis is general, the hæmorrhage is rarely limited to the substance of the hemispheres. Paralysis of half the body, characterized by its limitation to the muscles of the extremities, to those muscles of the face going to the angles of the mouth, and to the nose, and to the muscles that protrude the tongue, is due to destruction of the *corpus striatum* and *optic thalamus* of *one hemisphere.* Such patients can generally chew normally on the affected side; they can wrinkle the forehead, open and close the eyelids, and move the eyes in any direction. But they cannot use the paralyzed arm or foot, the mouth hangs down on the *affected side,* and the nostril is contracted. Occasionally the cheek flaps on expiration; *while* on the sound side the angle of the mouth is drawn up, and the nostril dilated. When the patient protrudes the tongue, its point is directed to the paralyzed side, because only the muscles of the opposite side push forward the root of the tongue, and elongate that organ. During profound stupor the deviation of the face indicates paralysis on one side. In less severe cases the condition of the limbs as to Volition is the guide. The tongue commonly deviates to the paralyzed side; and any extreme movement of the face, such as crying or laughing, renders the inequality of action more apparent. The *orbicularis oculi* generally escapes paralysis, or is less affected than the other facial

muscles. The loss of motion is commonly absolute at first, especially in the arm, which is generally more profoundly affected than the leg, the one being more completely paralyzed than the other. Stertor, with involuntary defecation and micturition, are common. *Involuntary contractions* of a tonic or clonic kind are extremely rare from hæmorrhage limited to the cerebral substance. *Reflex* movements continue, and the respiratory acts of the thoracic muscles are duly performed.

The more common combinations of symptoms by which the existence of cerebral hæmorrhage limited to the medullary substance might be inferred are—

1. *Profound coma, with hemiplegia on one side, of marked intensity, and without rigidity.*
2. *Paralysis of both sides, but one more profoundly affected than the other—a rare occurrence in limited hæmorrhage.*
3. *Slight coma, but paralysis hemiplegic and complete.*

A large apoplectic clot which destroys the *corpus striatum* or *thalamus* leaves a *hemiplegia* that never disappears; but small clots in those parts by which nerve filaments and unbroken and ganglianic nerve-cells are unimpaired, but only temporarily pressed apart, leave paralysis which is only temporary.

Extensive apoplectic clots at other parts of the cerebrum not unfrequently leave paralysis which sooner or later disappears again, the capillaries of the motor centres being relieved of pressure by the partial re-absorption of the extravasation, and so become again permeable to blood; or that the collateral œdema in the vicinity of the broken-down part of the brain which extended to the motor centres has disappeared, with cicatrization and contraction of the apoplectic clot (NIEMEYER).

B. Hæmorrhage into the Ventricles cannot in some cases be distinguished from arachnoid extravasation, or in others from effusion into the cerebral substance only, especially when in the vicinity of the ventricles. The cases, however, which are less doubtful are marked by the following characteristics, in addition to the general signs of apoplexy:—

1. *The Mental State.*—Coma is very profoundly marked at the commencement, and remains of equal intensity; or the patient, after partially recovering from a slight seizure, is again suddenly plunged into profound coma, from which there is no recovery. This second attack is presumed to indicate the rupture of the hæmorrhage either into the ventricles or the arachnoid cavity, from its original site of extravasation in the medullary substance of the brain near the ventricles or near the surface.

2. *Motorial Symptoms.*—*Paralysis* is complete in degree, and is developed simultaneously on both sides; or after having been hemiplegic for a short time it becomes general; when the coma of the second attack above noticed comes on, stertorous breathing is strongly marked. Involuntary evacuations follow. The pupils remain dilated. Deglutition is dangerous and difficult. When the paralysis is general and the coma profound, it is almost a sure sign that hæmorrhage has taken place to a considerable extent into the ventricles. *Rigidity* or *tonic contraction* of the muscles is present in many cases of hæmorrhage, and, in nineteen

out of twenty-six cases, occurs in the paralyzed limbs; and in about four out of twenty-six cases may be seen in those of the healthy side (DR. RUSSELL REYNOLDS). Its presence is a sign of extensive hæmorrhage, with laceration of the brain. The most frequent combination of symptoms indicative of hæmorrhage into the ventricles may be shortly stated to be *profound coma, with general paralysis and rigidity.*

C. Arachnoid Hæmorrhage occurs when the extravasation bursts through the pia mater and arachnoid into the space between the membranes, and such cannot be distinguished from the *ventricular extravasation* just noticed. If, however, the extravasation is immediately subarachnoid at first, and of limited extent, it may be approximately diagnosed,—*First,* By the nature of the premonitory symptoms having partaken of meningeal inflammation, such as by severe pain in the head, with impaired Intelligence and power of movement. *Second,* The attack is less sudden than in cases of congestion or of central hæmorrhage, and the symptoms are progressively developed.

The following are the combinations of symptoms which indicate the occurrence of subarachnoid hæmorrhage:—

(1.) *Complete and profound coma without paralysis; or with general paralysis very slightly developed in intensity.* (2.) *Complete loss of consciousness without paralysis; but combined with rigidity or clonic contractions of the limbs.* (3.) *Paralysis of hemiplegic distribution as regards the limbs, but without deviation of the features, the muscles of the face not being implicated.* (4.) *An apoplectic attack without anæsthesia.* (5.) *Imperfectly developed coma with general paralysis.* (6.) *An apoplectic attack, of which the symptoms are somewhat interchangeable or remittent.*

It is generally believed that the apoplectic fit is due to pressure or bruising of the nerve filaments and ganglious cells of the entire brain by extravasation. Niemeyer, however, refers the apoplectic fit to sudden compression of the capillaries—that is to anæmia of the brain substance.

In all large hæmorrhages such anæmia of the brain is a constant *post-mortem* appearance; and during life it is indicated by a remarkably increased pulsation of the carotids.

This symptom is sometimes regarded as a sign of increased pressure or flow of blood into the head; whereas he considers that it really indicates an obstruction at its entry, on the same principle that if a string is tightly tied round the end of the finger, the artery of the finger passing to its end will have its pulsation visibly increased.

Hence it is that large effusions of blood, exudations, transudations, and large tumors encroaching on the skull space are attended with increased pulsation of the carotids.

Where this symptom exists, without hypertrophy of the left ventricle, and without increase of pulsation of other arteries, it must be held as diagnostic of brain disease encroaching on the cavity of the cranium.

Causes.—Among the most frequent causes of apoplexy, especially in some constitutions, is an intemperate use of fermented liquors—a class of substances which powerfully excites, mainly by inducing paralysis of functions, thus allowing the evidences of excitement to appear as the prominent phenomena (ANSTIE). Alcohol also acts specifically on the heart and arteries, causing not only temporary energetic actions of those parts, but also organic alterations in their structure. In the latter case the powers of the heart are often permanently augmented, while the coats of the arteries, thickened, thinned, or ulcerated, have their elasticity destroyed, and thus the tendency to hæmorrhage in the brain is increased. The excessive use of narcotics, as opium or tobacco, is also supposed to predispose to congestion of the brain, and consequently to cerebral hæmorrhage.

The following conclusions have now been arrived at from a comprehensive view of numerous cases:—(1.) That in by far the greater number of cases cerebral hæmorrhage is due to degenerations of the cerebral arteries as its predisposing cause. In the smaller arteries these consist of fatty metamorphosis, or simple atrophy, with the various forms of consecutive dilatation; while in the larger arteries of the base there is *arteritis*, issuing in ossification, or fatty degeneration, or *atheroma*, or passive calcification. (2.) A not entirely rare cause are true aneurisms of the large cerebral arteries. (3.) Hypertrophy of the left ventricle will only favour cerebral hæmorrhage when it permanently increases the normal tension of the aortal system; but this is not the case in compensating hypertrophy of valvular disease of the heart. (4.) In about one-seventh of all cases of apoplexy, neither predisposing diseases of the heart nor of the vessels could be demonstrated (EULENBERG in Virchow's *Archiv.*, vol. xxiv., p. 329; and *Syden. Soc. Year-Book* for 1862, p. 81).

The connection of apoplexy with advanced disease of the kidney has occasionally attracted the attention of pathologists; and the late Dr. S. Kirkes recently brought forward some facts in proof of the frequency with which the kidneys are found diseased in fatal cases of apoplexy; and which, to a certain extent, explain the connection between the renal and cerebral affections (*Med. Times and Gazette*, 1835, p. 515). In all the cases except one the heart was found enlarged—an enlargement due to the prolonged disease of the kidney—and generally in the form of hypertrophy of the left ventricle, without valvular disease. The primary disease is that of the kidney; then the heart and then the blood-vessels become diseased in consequence of the primary affection. The disease of the blood-vessels is chiefly in the form of the well-known yellowish-white thickening and deposit within the coats of the blood-vessels; and Dr. Kirkes believed that the disease of the vessels is in a great measure the result of the continual over-distension and straining to which the arteries are subjected by the unwonted energy with which an hypertrophied heart propels the blood along them:—the full force of the hypertrophied ventricle, independent of valvular disease, will be exercised upon the arterial current.

Extremes of temperature are likewise powerful predisponents to apoplexy. In summer the fluids of the body tend to produce turgidity of the vessels in some constitutions, and the tone of the capillaries is impaired; while in winter the cold drives the blood from the periphery of the body to its central organs, and consequently to the brain. Sudden and great vicissitudes of the weather, as they rapidly exhaust the nervous power, are more frequently fatal than the uniform continuance of its extremes.

The powerful effects of *moral causes* in producing this fatal disorder are well known. *Mechanical obstruction* is also a frequent occasion of apoplexy. If an obstacle, for example, be opposed to the course of the blood, as when the valves of the heart are diseased, the blood accumulates in the capillary system generally, and consequently in the brain; or errant clots of fibrin choke up the minute cerebral vessels. Apoplexy is still more common when the aorta is diseased, the force of the heart, unchecked by the elasticity of that vessel, acting directly on the brain, so that its vessels often give way from this cause. *Mechanical violence,* also, may produce apoplectic effusion. Thus, concussion of the brain, however slight, always produces more or less congestion of that organ; and, if severe, effusion may take place below the *dura matter*, or between the membranes, or into the substance of the brain, which may be extensively ruptured, or the *septum* lucidum torn.

Apoplexy has been known to occur even in childhood. Billard gives the case of a child three days old that died apoplectic from effusion into the left hemisphere and about the lateral parts of the *corpora striata.* Serres saw a similar case in a child three months old. Apoplexy, however, is extremely rare till puberty, and only a few cases are met with before twenty. It is not unfrequent between thirty and fifty, while after fifty it is one of the most frequent causes of death. There are many circumstances which favour the disposition to apoplexy in old age. At that period the capillary system becomes impaired in most organs, and thus the veins are filled with a greater quantity of blood, or they become congested. The cerebral arteries also are often diseased; the heart has frequently acquired an abnormal power, driving the blood with great violence and with an increased momentum towards the brain, while the lungs have their functions so impaired that the blood is only imperfectly oxygenated; and all these are causes of congestion, and of tendency to rupture of the vessels of the brain.

Both sexes are liable to this affection, and in nearly equal proportion. Those most liable to attack are the florid in complexion, of short-necked conformation, with prominent eyes, broad chests, and protuberant bellies, and sometimes enormously fat, especially if high livers, sedentary, and indolent. Many thin persons, with spare long necks, however, frequently die from apoplexy; but it is probable that in these cases the heart or large vessels have been diseased.

The act of digestion, or rather fulness of the stomach, appears to predispose to apoplexy. Numbers are attacked after dinner. Sleep, also, associated with a temporary congestion of the vessels of the brain, is another predisposing circumstance. Thus, of 176 cases examined by Gendrin, 97 had been attacked during sleep. In the sleep of a healthy person the brain is anæmic (DURHAM).

Many diseases, or conditions of the body, predispose to apoplexy, as mania, epilepsy, suppressed hæmorrhoids, amenorrhœa, and especially the "*turn or change of life.*"

Diagnosis.—See previous subject, under "Softening."

Prognosis.—Apoplexy is always a grave disease, and the more grave in proportion, generally, as the respiration is stertorous and the deglutition difficult. Each succeeding attack is more dangerous than the former. The practitioner should also be guarded in his prognosis till after the first week or ten days, lest inflammation should come on, or a fresh attack destroy the patient. Popular opinion (and it is useful in pronouncing an opinion to know what are popular beliefs) supposes the patient to suffer three apoplectic attacks—the first being mild, the second followed by paralysis, while the third is fatal. It is only in a few instances that this number is exceeded. If a patient does not die in the fit of apoplexy, a more or less severe *encephalitis* will set in after a few days, as a result of the traumatic injury done to the texture of the brain.

Increased frequency of pulse, with other signs of fever, headache, sparks before the eyes, delirium, twitchings and contractions of the paralyzed parts, indicate "symptoms of reaction," which may be moderate and finally disappear, leaving the patient more or less permanently paralytic. If inflammation in the vicinity of the clot progresses, however, inflammatory softening will be the result, the end of which has been described in previous pages.

In the congestive form of apoplexy, if active and judicious measures of treatment are employed, the recovery may be rapid and complete; but if this is not the case, there is a liability to a more aggravated form, from which nothing is more variable than the time of recovery. In a very few instances the patient is restored in a few days, or in a few weeks, or in a few months, but more commonly the lesions of motion are permanent, or nearly so. In general, however, some slight improvement takes place even in the worst cases, so that the patient recovers some use, first of his leg, and then, perhaps, of his arm, so that he is able to walk with a straight leg and a dragging foot. The use of his arm returns more slowly and more imperfectly. This recovery is often preceded and accompanied by very severe pains, especially of the upper extremity, marking the still irritated state of the brain. The limb, however, uniformly wastes, and its vital powers are so impaired that, if inflamed, the inflammation seldom terminates by resolution, but has a great tendency to gangrene, while cicatrization is slow and difficult.

If, on the other hand, after a severe expression of hemiplegia, the patient begins to recover the use of his arm more rapidly and completely than he does the use of his leg, it is a fatal sign (TROUSSEAU, TODD, BAZINE). Also, when the fingers of the paralyzed hand continue flexed into the palm, through contraction of the flexors after cerebral hæmorrhage, the use of the hand will remain permanently lost, and no hope of improvement can be held out (TROUSSEAU).

The commonest occurrence is the perfect recovery of the mental faculty, and the progressive but much more gradual return of motion and sensibility. Paralysis of motion sometimes persists in groups of muscles, such as those of the tongue, the forearm, or hand. Sensibility is generally first restored, then the motion of the lower extremity, then the arm, and lastly that of the fore-arm and hand. Discordant opinions are entertained relative to the influence of the electric structures on the paralyzed muscles. The late Dr. Marshall Hall was of opinion that the muscles of the non-paralyzed limb were the more irritable. The late Dr. R. B. Todd entertained precisely the reverse opinion. These were the highest authorities of their day in this country. Duchenne draws attention to two conditions of paralysis after apoplexy—namely, one directly dependent on the central lesion, the other the mere result of inaction for some time. In the former case there is contraction of the muscle as the result of increased spinal action, uncontrolled by the central ganglia of the brain—in the latter form the muscles are flaccid. Active tonic contraction of the muscles, distinct from simple shortening of the flexors, he considers as indicating inflammatory action in the walls of the cyst; but this distinction is one which requires corroboration or contradiction by subsequent observation (DR. RUSSELL REYNOLDS).

No doubt the principal adverse circumstances attending recovery from apoplexy are, that although the patient appears to be doing well the first few days after the attack, yet towards the close of the first week the brain, irritated by the presence of the clot, inflames and softens, and thus induces another and a fatal attack of apoplexy. Should the patient, however, survive this dangerous period, he may continue to live many months, or years, according to his age; but he is generally at length cut off by a fresh attack of apoplexy, or his brain ultimately inflames and softens, and he dies in a typhoid state, as already described.

Although it is the general rule that the patient, on recovering from the attack, has the good fortune to recover all the faculties of his mind, yet his Memory is not unfrequently impaired, often to such a degree that he has forgotten all dates, the names of his friends, or even the names of things. Broussonet, Professor of Medicine at Montpellier, had entirely lost the remembrance of all substantive nouns, and another case is on record in which the patient lost the recollection of all his adjectives. In some instances the power of association is also so destroyed that although many remember both names and things, they are unable to connect the thing with the proper word, so that they call that which is cold, hot; or speak of night when they mean day; or call a coffee-pot a wash-hand basin (*aphasia*). Others, again, have forgotten how to read, and the power thus lost either returns suddenly, or they are obliged to learn *de novo.*

The attention generally is very greatly impaired, and the patient is no longer able to transact business; or if he begins a sentence, is unable to finish it, or he repeats the same idea over and over again. The emotions and passions also are little under control. Some weep like children, others laugh immoderately, and all are easily terrified, or otherwise easily influenced through the emotions.

All these circumstances must be remembered in giving a prognosis regarding an apoplectic patient.

Treatment.—The patient, if seen during the fit, may be bled if the tendency to death is by *coma*, and if the pulse be full, or hard, or thrilling; if the vessels of the neck be congested, the heat of the scalp increased, and if the face be full and turgid. The state of the heart ought to be examined into first. If its action be vigorous, its impulse strong, and its sounds loud but normal; and the heat of the skin preserved, blood-letting is still more required. Slow and deep respiratory movements, with stertor, add greatly to the necessity of immediate venesection, if there be no signs of commencing œdema of the lungs. The beneficial action of the remedy is shown by the pulse becoming softer, more subdued, and more regular. The bleeding may then be allowed to proceed till an obvious impression of this kind is made, or even until the pulse begins to flag. Large bleedings, however, are to be avoided. Ten ounces will generally be sufficient, if taken at the first outset of the attack; but if the pulse does not improve, and other symptoms remain unrelieved, sixteen to twenty ounces may be taken. The indications for blood-letting being thus strongly pronounced, "we ought not to hesitate to open a vein, regardless of the age of our patient" (MACLACHLAN). The bleeding ought to be permitted to flow from a large opening, in order to relieve the congestion, to check, if possible, a further effusion of blood, and to divert its active flow from the head. The quantity taken should be proportioned to the degree of stertor, and to the powers of the patient. The head and shoulders should be raised while the blood is flowing.

On the other hand, in some cases, bleeding during an apoplectic fit hastens a fatal result—collapse occurring immediately after venesection, from which the patient never recovers.

If, therefore, the pulse be small and slow, feeble, or almost imperceptible, the skin cold and clammy, with a tendency to death by syncope; if the heart's action be feeble or weak, and the pulse irregular; if the patient has been of intemperate habits, or is suffering from organic disease of the heart and arteries; or if there is a gouty or rheumatic history, then no advantage is to be gained by the abstraction of blood at this time and in this way. Blood-letting is therefore contra-indicated, under the following circumstances:—(*a.*) anæmia, (*b.*) aortic valvular disease, (*c.*) in cases commencing with syncope. In such case the use of stimulants must be had recourse to in order to prevent paralysis of the heart.

If the blood-letting is not followed by some degree of consciousness, it may be inferred that the amount of blood effused is considerable, and that the patient in all probability will not recover. Still, an additional chance of recovery may be given by applying to the head cold cloths, or crushed ice in a bladder, leeches to the temples, and mustard cataplasms to the feet; also by placing a drop or two of croton oil on the tongue, and by throwing up a cathartic enema of castor oil or other purgative.

At one time every case of apoplexy was treated by blood-letting; and statistics prove, of such indiscriminate practice, that the more freely the blood was taken away the greater was the mortality (COPMAN). Some physicians are opposed to any considerable bleeding during the fit, considering that the bony structure which contains the brain removes all atmospheric pressure so entirely as to cause that organ at all times to contain an equal quantity of blood. A space within the head for a very sensible expansion and contraction at each pulsation of the heart is opposed to such a belief, while *post-mortem* examination shows the brain to contain very different quantities of blood, being sometimes gorged and sometimes blanched of that fluid; but the amount contained in the vessels and the amount of cerebro-spinal fluid are always in inverse proportion—that distension of vessels is accompanied by decrease of arachnoid fluid, and conversely, when the vessels are less full, the meshes of the arachnoid contain more fluid. These facts distinctly show that some arrangement exists for regulating the quantity of blood sent to the brain; and we ought, therefore, in a disease of this moment, to follow the dictates of a long experience rather than the conclusions of a fallacious reasoning.

Blood-letting, therefore, in apoplexy is not to be adopted as a matter of course. In the majority of cases the hæmorrhage is not of a kind likely to be arrested by blood-letting (TODD).

When, also, the symptoms of cerebral hæmorrhage are slight, blood-letting is not to be thought of; and Trousseau at last came to believe that in *all* cases, grave or slight, patients did better without either blood-letting or purgatives. He considers the part played by congestion of the brain to have been much exaggerated. Instead, therefore, of bleeding and putting patients on low diet, Trousseau recommends that the patients be fed and made to get up, if they can. He considers also that blood-letting is not called for even in the stage of re-action.

When emetics are now spoken of, it is generally with the view of condemning them. The physiological phenomena which attend their action increase the determination of blood to the head, as is now well known; and the action of vomiting may be fatal in such cases. After the patient has in some degree revived, and the congestion removed, some time for the absorption of the blood effused should be allowed before deciding upon the future treatment. Any very large depletion after that point is gained would rather facilitate extravasation than prevent it. A few hours, then, having elapsed, the conduct of the practitioner should be guided by the occurrence or not in the patient of *pain of the head*, which may be taken as a measure of the fulness of the brain, and its tendency to inflammation. If, therefore, there be pain in the head, ten to twelve leeches should be applied from time to time till that symptom is entirely relieved; or, supposing the pulse to be full and strong, and the patient free from headache, yet, under these circumstances, leeches should be applied to the head, to subdue that re-action which so generally takes place from the fourth to the seventh day.

The further treatment of the case is by moderately purging the patient, both as a means of relieving the head and of improving the secretions of the alimentary canal, which are often black and fetid. Active and searching purgatives generally do good. Five grains of *calomel*, with a drachm of *compound jalap powder*, given as soon as the patient can swallow, and followed up by *black draught*, or by an ounce of *sulphate of magnesia* with *camphor mixture* every four or six hours, and continued, according to its effects, for a greater or less length of time, are the best means we have for promoting recovery, and for preventing a relapse.

These prescriptions are recommended on the supposition that the attack has been associated with simple plethora. In many cases, however, it is a consequence of hypertrophy of the heart, without valvular disease. And in such cases less blood should be taken, and eight to ten minims of *digitalis* may be added to each dose of the purgative medicine. If the power of swallowing is in abeyance, then three or four drops of croton oil should be put on the back part of the tongue, and stimulating enemata thrown up the rectum. The following are recommended by Dr. Tanner:—

1. *Enema of Turpentine and Castor Oil.*—℞. Olei Ricini, Olei Terebinthine, ā ā ℥iss.; Tincturæ Asafœtidæ. ʒii.; Decocti Avenæ, ℥xii.; *misce*, fiat enema. To be thrown up the rectum by means of a long stomach-pump tube.

2. *Croton Oil Enema.*—℞. Olei Ricini, Olei Terebinthinæ, ā ā ℥i.; Olei Crotonis, ♏ vi.; Decocti Avenæ, ℥iv.; *misce*, fiat enema.

Although turpentine is objected to by some on account of the intoxicating effects which it is sometimes apt to produce, it is nevertheless an efficient remedy where torpor and insensibility exist. It is of great importance to empty the rectum and lower bowel. In cases where the ability to swallow is lost, a drop or two of croton oil, with five grains of calomel, rubbed up with fresh butter, may be laid on the back of the tongue.

If the intestines are distended by gases, an enema of castor oil and rue may be given:—

3. *Castor Oil and Rue Enema.*—℞. Confectionis Rutæ, ʒi.; Olei Ricini, ℥i.; Tincturæ Asafœtidæ. ʒii.; Decocti Avenæ, ℥vii.; *misce* (*Practice of Medicine*, 4th edition, p. 653).

All apprehension of a relapse being at an end, the patient is in general most willing to believe that the palsy which may remain is a mere local disease, and to submit to any treatment for its removal. The ancients applied the actual cautery to the extremities, to the coronal suture, or to the occiput, but without any beneficial success. The moderns have had recourse to blisters, to friction, to electricity, and to strychnine; but every attempt to act locally on the muscular system is prejudicial so long as any central irritation exists. Such remedies are neither theoretically nor practically useful. (See "Paralysis.") Active or passive exercise of the muscles are remedies highly beneficial.

Dietetic Treatment.—The diet of the patient should be low, till all apprehension of a relapse is past, and limited to milk, boiled vegetables, light puddings, and fish. At no subsequent period ought he to indulge in a full animal diet, or to drink undiluted wines. At the same time, too, lowering a regimen is to be avoided, as thereby the irritability of the system and the heart's action generally is increased. All the causes of the disease already fully referred to should be avoided, counteracted, or overcome. The diet and the bowels should be carefully regulated, and the patient placed under the best possible hygienic influences. Niemeyer recommends a sojourn at the waters of Wildbad, Gastien, Pfäfers, or Ragatz, where he considers that gradually both cerebral and spinal paralysis often improve. The *induced current of electricity* is also beneficial. It seems to improve the nutrition of the paralyzed muscles, which tend to atrophy from lung disease, and paralysis tends also to get worse from diminished excitability of the nerves. Local faradisation by induced currents of electricity gives artificial exercise to these muscles, and thereby improves their functional and nutritive properties.

HÆMATOMA OF THE DURA MATER.*

Definition.—*Sanguineous encapsuled flattened masses, composed of fine layers of fibrin, spread to a greater or less extent over the under surface of the dura mater, accompanied by repeated small extravasations of blood, which are converted into pigment. By repetition of the process several layers come to be deposited one upon the other. Numerous and large blood-vessels form in these layers; and from these vessels renewals of the hæmorrhages occur. The disease is chronic, and terminates, after continued cephalic suffering, generally suddenly, with symptoms of apoplexy.*

Pathology.—These tumors of bloody formation seem to occur in connection with more or less inflammation of the *dura mater.* The effusion of blood between the *dura mater* and the arachnoid is not in most cases a primary occurrence, but the productive results of inflammation first occur; and the new growth is developed into a fibrous membrane traversed by a copious network of new-formed blood-vessels. It is from the rupture of these vessels that extravasations proceed (VIRCHOW, WEBER). The lesion is sometimes described as due to *intra-meningeal* apoplexy, with false membranes on the *dura mater;* but the false membranes which are the result of the chronic inflammation precede the apoplectic phenomena. The *hæmatoma* often attains a considerable size. It may be from four to five inches long by two and a half inches broad, and one half to three quarters of an inch thick. It is generally of a flattened circular form, with a central elevation, and usually located near the sagital suture. The long diameter is parallel to the falciform process. The tumor generally occurs on one side only; or, if bilateral, one is more developed than the other. The walls of the sac are generally of a rusty colour, from the altered colouring matter of the blood, and the contents of the sac are partly fresh fluid or partially coagulated blood, in the form of dirty red-brown clots. The cerebrum below is flattened, and may even show a depression. The affection appears to occur only in the adult, and generally after the age of fifty. In recent cases very fine layers of fibrin are found to a greater or less extent spread over the surface of the *dura mater.* By repetition of the inflammatory process, numerous layers of fibrin become deposited one upon the other; and much more numerous and larger blood-vessels form in these layers than are to be met with in the *dura mater* itself. From these new-formed vessels the hæmorrhage proceeds which gives rise to the formation of the *hæmatoma*, and its cystic enclosure is formed by the extravasation taking place between the layers of the false membrane (VIRCHOW).

The lesion is comparatively rare, and was at one time believed to have been the result simply of an apoplectic effusion of blood at the periphery of which the fibrin had been precipitated, and the whole encapsuled. The lesion is now believed to be the remains of repeated chronic inflammations of the *dura mater*, with hæmorrhagic exudations. The blood comes from the large thick-walled capillaries which form in the *pseudo*-membrane deposited in the *dura mater* during the chronic inflammation, and the blood is effused between the layers of this exudation.

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* This disease is not mentioned by the College of Physicians, in their nomenclature, as distinct from inflammation of the *dura mater.*

Old age, disorders of the intellect, especially general paralysis, chronic alcoholism are the kind of cases in which the lesion is found; and sometimes it seems traceable to external injury. In such cases years may intervene between the injury and the symtoms of hæmatoma (GRIESINGER). Of several specimens of the lesion preserved in the museum of the Army Medical Department at Netley Hospital, one occurred in a soldier thirty-three years of age, with tropical service, a history of epilepsy, fever, and lunacy, and finally death with apoplectic symptoms; a second occurred in a soldier twenty-seven years of age, with a history of epilepsy; a third in a soldier thirty-eight years of age, also with a history of epilepsy, tropical service, chronic alcoholism, injury by a fall into the trenches at St. Mary's Barracks, Chatham, and death from paralysis.

Symptoms may extend over several months, and consist in general weakening of the Memory and of the Intelligence; the occurrence of giddiness; and of continuously intermittent general or local pain in the head. At a later period an aggravation of all these phenomena occurs, with transitory losses of consciousness from the momentary arrest of cerebral circulation. Somnolence and apathy prevail, with weakness, and generally one-sided paralysis of the extremities, which may soon disappear.

Treatment—Is more or less expectant. The newly formed membranes tend to undergo retrograde change, and thus finally disappear. To effect this end is therefore the object of any rational treatment, which must be based on the special history of the individual case, especially as to the previous existence or not of syphilis or injury.

SUNSTROKE.

LATIN EQ., *Solis ictus;* FRENCH EQ., *Coup de soleil;* GERMAN EQ., *Sonnenstich;* ITALIAN EQ., *Colpo di sole.*

Definition.—*An affection of the nervous system, associated with vertigo, and sometimes with headache, or the gradual accession of listlessness and torpidity, with a desire to lie down* (LONGMORE, BARCLAY). *These febrile phenomena may culminate in more or less sudden and complete insensibility, without the power of sense or motion, the breathing rapid, and getting more and more noisy as death approaches. Convulsions of the extremities usher in a complete state of coma, in which the patient gradually dies. The approach of death is indicated by the failure of the heart's action, the fluttering of the pulse, the irregularity of the respiration; and the fatal event may supervene within five minutes to a few hours after the disease has become fully expressed. Death is either by* syncope, apnœa, *or by a combination of the two. In cases which recover, various sequelæ are apt to supervene, such as forms of paralysis, more or less complete, choreic movements, melancholia, and other forms of disorder of the Intellect.*

Pathology and Symptoms.—This singular and fatal affection of the nervous system has been described under a great variety of names—e. g., *Heat apoplexy, Heat asphyxia, Coup de soleil, Insolatio, Ictus solis,* and lastly, *Erythismus tropicus.* Notwithstanding that sun heat and the sun's rays have been considered the main agent in producing this disease, it is not less true that the full expression of the disease not unfrequently occurs at midnight. The name implies a common, and certainly a most powerfully exciting cause of a disease which has been variously and erroneously described as of the nature of apoplexy, or of some form of continued fever.

A very great variety of views have been put forward regarding the pathology of this remarkable disease; and the following account of its nature is based upon the very interesting accounts which have been given by many Indian medical officers, and especially by Deputy Inspector-General Longmore, C.B., and now Professor of Surgery in the Army Medical School, when on duty with the 19th Regiment at Barrackpore; by Surgeon Butler at Mean Meer; and by Dr. Barclay, of the 43rd Light Infantry, while on the march from Jubbulpore to Calpee.

Instances of the occurrence of *sunstroke,* and the circumstances under which it has been observed to occur, will best convey any idea of the nature of this disease.

One of the earliest accounts of *sunstroke,* in which its nature was distinctly recognized, is that given by Mr. Russell, of the 73rd Regiment, while in medical charge of the 68th, in May, 1834. The regiment had then recently arrived in Madras—a fine corps of men in robust health. The funeral of a general officer being about to take place, the men were marched out *at an early hour in the afternoon, buttoned up in red coats and military stocks, at a season, too, when the hot land winds had just set in,* rendering the atmosphere dry and suffocating even under the shelter of a roof, and when the sun's rays were excessively powerful. The funeral procession forthwith advanced, and after having proceeded two or three miles, several men fell down senseless. As many as eight or nine were brought into hospital that evening, and many more on the following day. Three men died—one on the spot and two within a few hours. The symptoms observed (and they were alike in the three cases) were, first, excessive thirst and a sense of faintness; then difficulty of breathing, stertor, coma, lividity of the face; and in one whom Dr. Russell examined, contraction of the pupil. The remainder of the cases (in which the attack was slighter, and the powers of re-action perhaps greater, or at all events sufficiently great) rallied; and the attack in them ran on into either an ephemeral or more continued form of fever. The symptoms of these cases did not more nearly resemble each other than did the *post-mortem* appearances. The brain was healthy in all; no congestion or accumulation of blood was observable. A very small quantity of serum was effused under the base of one; but in all three the lungs were congested, even to blackness, through their entire extent; and so densely loaded were they, that complete obstruction must have taken place. There was also an accumulation of blood in the right side of the heart, and in the great vessels (*Medical Gazette,* "Graves' Clinical Lectures," vol. i., p. 181).

A nearly parallel example is related by Sir Ranald Martin as having occurred in the experience of Dr. Miligan, of the 63rd Foot, from the exposure of his corps to the sun during a military funeral at Madras. The greater number of the men were in the prime of life; but there were some old soldiers who had served twenty years and upwards in the West Indies, and who were much broken down by service and intemperate habits. The entire corps had just arrived from the Australian colonies, where spirituous liquors can be had on easy terms. The regiment landed at Madras in the month of May; and from the date of the "untoward circumstance" of the funeral, the hospital became filled with cases of fever. Two men dropped down and died on the very day of the funeral, and for several days afterwards the fever cases augmented considerably (*The Influence of Tropical Climates*, p. 205).

The dreadful march of the 43rd Light Infantry from Jubbulpore to Calpee furnishes the most instructive and melancholy examples of *sunstroke.* It must be remembered that the regiment had previously marched throughout the greater part of the length of the Madras Presidency—a march extending from the 24th December, 1857, to the 17th of April, 1858, when the regiment arrived at Jubbulpore. During this first march the regiment enjoyed great immunity from sickness, owing to the sanitary precautions which had been taken. The march, however, told seriously upon the men in general. They lost condition, and in a great measure their robust appearance, and were in urgent need of rest. But after a rest of five days only, they had to commence the march from Jubbulpore to Nagode, a distance of 163 miles, arriving there on the 8th of May. The heat on the march was excessive, and it told very much on the health of the men, already exhausted as they were by a previous march of almost unexampled length. After having been *four months and thirteen days in the field, and after they had marched* 969 *miles*, a fatal case occurred; and from that date cases of *sunstroke* gradually increased in frequency. At Nagode the regiment remained eight days; and although the indications of exhaustion in the altered looks of the men, their loss of flesh, and their evidently failing strength, were so obvious that they forced themselves on the observation of every one, *yet the men were ordered to continue their march to Banda.* The periodical hot winds were blowing at the time day and night, with scarcely any intermission, and the heat of the weather was almost unbearable. After leaving Nogode they were obliged to encamp for four days in the bottom of a deep and narrow ravine, with nearly precipitous sides upwards of a mile in height. The heat in this valley was insufferable, and exceeded anything the regiment had ever been exposed to before, or that they were exposed to afterwards. The thermometer varied during the day from 115° Fahr. to 118° Fahr. in the largest tents, and in the smaller ones it reached 127° Fahr. Night brought but little relief. On one occasion Dr. Barclay observed the thermometer standing at 105° Fahr. at midnight. Such was the overpowering effect of the heat in this "valley of the shadow of death," that even some of the natives were struck down, and died with all the symptoms of *sunstroke* in less than an hour. The number of cases among the men of the regiment, especially during the first day, was very great. They were carried into the hospital tents at every hour of the day and night; and although a large proportion of them recovered, two officers and eleven men were buried under one tree in the neighbourhood of the camp. Marching was resumed on the 24th of May. On the 27th the regiment arrived at Banda, a distance of about 100 miles from Nagode, having lost during the march two officers and nineteen men.

The health of the regiment improved daily during a short stay at Banda; but the men were still in a miserable state of exhaustion when the regiment again began to march for Kirwee on the 3d of June. At Kirwee five men died from *sunstroke,* and on the return to Banda two more died. The regiment now marched for Humeerpore on the 28th of June, *en route* for Calpee. The weather was again excessively hot, and the men suffered very severely. They were by this time completely worn out and prostrated. There was scarcely a man in the regiment whose strength was not reduced to a level with that of a child; and the officers were not in a very much better plight. Many men broke down altogether, and had to be carried as it could best be managed—in *doolies,* in sick carts, or on baggage and commissariat carts after they were filled. It was painful to see many others who, a few months before, had been in robust health and full of vigour, staggering from weakness as they endeavoured to keep up with the column, throwing themselves down completely exhausted at every halt, and scarcely able to rise from the ground when the "*assembly*" sounded. Calpee was reached on the 5th of July, and between the 28th of June and the 7th of July one officer and five men had died.

These details are given for the purpose of showing *the effects of protracted exposure to intense heat in a body of men in the field debilitated by fatigue and want of rest.* It may now be shown how the effects of protracted exposure to intense heat in a body of men may operate upon them when in *quarters;* as described by Deputy Inspector-General Longmore. He records sixteen cases of *sunstroke* as having occurred in the 19th Regiment between the 23d of May and the 14th of June, 1858, when they were quartered at Barrackpore. The period was marked by an unusually elevated degree of temperature, and generally by great dryness of the atmosphere. The quarters were of a temporary and imperfect nature, some of them being merely hired *bungalows.*

Of the sixteen cases, five occurred in non-commissioned officers, and eleven in privates of the regiment, the proportion attacked being greatly more among the former than among the latter; their occupations, especially when on orderly duty, caused them to be more exposed than the men with whom they lived. Simple exposure to the external atmosphere and the solar rays were not of themselves sufficient to induce the disease. Of the five non-commissioned officers attacked, two were on duty at the time as orderly serjeants; one had had fever for several days, but had not been exposed to the sun on the day of attack; one had been slightly exposed; the fifth not at all. Of the privates, eight were attacked by the disease within doors, and three while on sentry. No cases occurred of sudden sunstroke—*i. e.,* of insensibility instantly induced by the direct rays of the sun in a man previously healthy.

The characteristic feature with regard to atmospheric temperature, when Mr. Longmore's cases were observed, was the little variation of it night or day. There was no rain; and the ground and buildings became so heated that, long after sunset, the radiation of heat maintained a high temperature within doors. Prolonged high atmospheric temperature was recognized as the essential cause of the attack; but nervous depression from solar exposure, fatigue, and previous illness were associated with that prime or essential cause.

But a most important element of causation is still to be mentioned—namely, *the influence of vitiated air when men are congregated without sufficient ventilation.* One-third of the cases, and nearly half the deaths, occurred under such circumstances; so that, *in quarters,* the predisposing causes of *sunstroke* may be comprised in the following conditions:—(1.) Prolonged atmospheric heat, with a dry and rarified state of the air; (2.) Nervous exhaustion; (3.) A contaminated atmosphere (LONGMORE, TAYLOR); while (4.) An increase of the average prevailing temperature sufficed to act as the more immediate exciting cause for the development of *sunstroke.*

The experience of Surgeon Butler at Mean Meer has led him to write, that "when the thermometer ranges beyond 98° Fahr. in crowded barracks, cases of apoplexy almost invariably occur." Dr. Crawford also, in writing on *sunstroke,* notices an electric condition of the atmosphere as influencing the respirability of the air, and refers especially to that peculiar state "in which the hairs of a horse's tail repel each other, in which the hairs of the head stand on end, in which a man exposed to its influence becomes irritable, headachy, and restless, without knowing exactly what is the matter with him. Such a state of atmosphere will generally be found to exist in localities where cases of *sunstroke* occur, whether such localities be the crowded barrack, in the still more crowded cantonment, the tented field, or the march in column through the still valley, the deep gorge, or the thick forest." Dr. Barclay notices that cases of *sunstroke* occur with increased frequency immediately before a thunder-storm, and that they cease as soon as the electrical discharge has taken place.

From the accounts given by these several observers it seems clear, as Dr. Barclay observes, that the symptoms of the disease are liable to be greatly modified by accidental causes, and that those phenomena which are most prominent under one set of circumstances are either absent altogether under another, or so very much less urgent as scarcely to attract observation—that the disease, in fact, varies in several important points according to the nature of the circumstances in which it occurs.

The phenomena which at any time have presented themselves under the various circumstances detailed by Mr. Longmore, Dr. Barclay, and others, may be summed up as follows:—When careful observations are made, the affection seems gradually induced by protracted exposure to extreme heat in a dry and rarefied air, combined with a vitiated atmosphere from defective ventilation, or with physical exertions of an arduous character, implying excessive fatigue of extreme duration, so as to bring about great debility and weariness of the body. Sleep at last cannot be obtained, or it is greatly interrupted, and of short duration. Deterioration of the general health is thus progressive, while altered looks and loss of flesh indicate extreme exhaustion. The skin becomes rough and scaly, and perspiration ceases. The heat of the surface increases to an intense degree; but accurate records of the temperature of the body in cases of *sunstroke* are not yet in existence. The bowels tend to become obstinately constipated. The urine becomes copious, and the calls to pass it are frequent; or even incontinence may prevail (LONGMORE, BARCLAY). Under such circumstances those morbid phenomena intervene which are noticed in the definition.

Most observers are agreed that intemperance cannot be charged with being the immediate exciting cause of the disease, although it is a universally acknowledged predisposing cause. The men of Dr. Barclay's regiment were extremely temperate, robust, and well-formed men; and Mr. Longmore gives a no less favourable account of his men as to temperance. The drunkard, indeed, is rarely to be found in the ranks under circumstances favourable to the development of *sunstroke*. He is either in prison, or skulking at some depôt, or in some hospital, where he may be left for want of transport; or, if with a regiment, "the pains" come on opportunely, to save him from fatigue and danger. The vascular system of a habitual drinker soon shows unmistakable indications of over-stimulation in the suffused eye, the bloated countenance, the profound sleep which follows the slightest indulgence, and the subacute meningitis which sooner or later supervenes in India (CRAWFORD). Such are not the kind of cases which furnish the deaths from *sunstroke*. When exposed to the stimulus of a tropical sun, such cases may sink under it, as they would under any other great excitement; and a debauch or an ephemeral fever will alike predispose a man to an attack of *sunstroke*, inasmuch as both may bring about that state of nervous depression which seems essential to the occurrence of this disease.

In Dr. Barclay's experience the deterioration of the health of his men in the field was progressive. For a long time before the occurrence of the first case of *sunstroke* every one had suffered more or less from "prickly heat," the severity of the affection being, as a rule, in proportion to the amount of perspiration from each individual; and when the heat of the weather became still more intense, one of the first symptoms of its producing an injurious effect was the gradual disappearance of this cutaneous eruption, the skin becoming rough and scaly, and the perspiration ceasing. In many cases the interruption of perspiration appeared to be complete—not the slightest feeling of dampness being perceptible in any part of the dress at any period of the day. On this point Dr. Simpson observed to Dr. Morehead,—"Every man seized with sunstroke, and who could answer questions, informed me that he had not perspired for a greater or less extent of time,—sometimes not for days,—previous to being attacked, and that he had enjoyed good health as long as he perspired; but that on the perspiration being checked, he felt dull and listless, and unable to take much exertion without making a great effort" (*Researches on Disease in India*, p. 617). The heat of the surface became at the same time much increased. The bowels became obstinately constipated. The appetite gradually failed; and a feeling of nausea was generally complained of, the sight of food often exciting loathing. In other instances there was nearly complete anorexia.

The urine became copious and limpid, and the calls to pass it frequent; so much so that Mr. Longmore especially notices a frequent desire to micturate as constant in all the cases in which there was an opportunity of ascertaining the point; and he justly remarks—"If this symptom should prove to be a general precursor of the attack, it might be rendered valuable as an indication of the approaching danger, which, by early and proper care, might then probably be averted; and its presence at a time when heat-apoplexy was prevalent would make the surgeon alert to obviate the more serious symptoms which might be expected to follow" (*Indian Annals*, vol. vi., 1860, p. 399). Sleep from the first was much interrupted, and the periods during which it could be obtained became gradually shorter, until at length no sleep could be got during the night. The pulse was frequent, sharp, and small. The tongue white posteriorly, but seldom foul or dry. Thirst was seldom very urgent. Vertigo was frequently complained of, but headache rarely; and, as a general rule, there was no complaint of pain. The general complaint was extreme debility, weariness, and prostration on any exertion; vertigo, nausea, and in many cases incontinence of urine, more particularly after exposure to the sun, "I cannot hold my water," being a very general answer to the first inquiries of the medical officer. These premonitory symptoms were attended with rapid and progressive emaciation. No one during the march (in the ravines of Northern Bundelcund, so well described by Dr. Barclay) became the subject of *sunstroke* without having previously suffered from some or all of these premonitory phenomena. Such phenomena, however, prevailed in a much larger proportion of cases in which symptoms of *sunstroke* did not supervene at all; and they seem to stand somewhat in the same relation to *sunstroke* that the premonitory diarrhœa does to *cholera.*

The attacks of *sunstroke* came on generally when the men were in their tents, during the day sometimes, but in several instances during the night, and also on the line of march. During the night the patient had generally laid himself down, often seemingly asleep, or trying to induce sleep, when the attention of his comrades would be directed to him by his hurried and heavy breathing, and on attempting to rouse him he was found to be insensible. In other instances he started up suddenly, and attempted to escape from the tent, staggering about, and struggling violently when laid hold of, evidently much alarmed, and anxious to escape from some imaginary object of terror; but in a very few minutes he became insensible.

In one or two instances the first symptom of the disease was reputed to have been an uncontrollable burst of laughter, without any apparent cause, and in sad enough circumstances,—insensibility and death speedily following. In a few instances the patient would come to hospital himself, or with the assistance of his comrades, exhibiting some of the symptoms (premonitory) already detailed, when insensibility or a tendency to sleep would gradually supervene. The time of the day at which most of the cases occurred is important in connection with the elevation of temperature. Ten out of sixteen cases occurred between the hours of 2 and 5 o'clock P.M., and five cases between 5 and 9·30

P.M. (Longmore). In Mr. Cotton's experience at Meerut the seizure usually happened towards evening. The thermometer generally indicated the maximum temperature of the twenty-four hours to be about 4 P.M.; and the variation in the thermometer-range was very slight from 2 P.M. to sunset; and even for some hours after sunset the temperature scarcely at all declined (Longmore).

Dr. H. S. Swift records, in the *New York Journal of Medicine* for 1854, some interesting particulars regarding sixty cases of sunstroke observed at the New York Hospital during that year. The seizure in all was sudden, in the midst of work, and was characterized by pain in the head, a sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially of the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appeared of uniform colour, generally blue or purple. In one case everything was red, in another green, in another white.

In three cases the attacks occurred between 8 and 11 A.M., in forty cases, between 11 A.M. and 4 P.M., and in seventeen cases between 4 and 9 P.M. The general result being that the majority of cases occurred during the *maximum* of the day's temperature.

When the disease was fully expressed, the symptoms were constant and regular. The patient lay on his back, without sense or motion, breathing rapidly, and as death approached, more and more noisily, from the vibration of the *uvula* and the *velum pendulum palati;* and although such *stertor* was present in most of the fatal cases, yet it never approached in degree to that which is common in true apoplexy. The eyes were *fixed, and turned slightly upwards,* becoming gradually more and more glassy, as if from the formation of a film over the cornea; the *pupils greatly contracted* (generally to the size of the head of a pin); the *conjunctivæ pinky, the colour gradually becoming deeper;* the congestion at first deep-seated, and the first symptom of it a pinky zone around the cornea, the superficial vessels afterwards becoming affected.

In Dr. Barclay's cases *the face was invariably pale;* the surface dry, harsh, and burning to the touch, far beyond what he ever felt in any other disease.

In Mr. Longmore's cases the heat of skin greatly exceeded that occurring in pneumonia, and was without parallel in his experience.

Dr. Clymer has collected together a number of records of body-temperature in this disease, some of them observed after death; and in other instances, as they are not noted, it is presumed the observation is during life. Separating the two series of records, the observations stand as follow:—

After death.	Observer.
99°, 104°	Dowler.
105°, 110°	,,
108°	H. C. Wood.
109°	,,
112°	Levick.
105·5°	,,

During life, or at moment of death.	Observer.
111°, 112°, 113°	Dowler.
109°	,,
106°	,,
108°	H. C. Wood.
109°	,,
109°	,,
104°	,,
106°	,,
109·5°	Levick.
109°	,,
106°	,,
109°, 108°, 108·8°	Bäumler.

Sudden and remarkable elevations of temperature are also recorded by Wunderlich. In 1867 Schneider observed a temperature above 104° Fahr., in a fatal case, two and a half hours after admission to hospital. Helbig records three similar cases. Ferber observed a temperature of 104° Fahr. in a case which recovered. Bäumler (in *Med. Times and Gazette*, August 1, 1868) observed a temperature of 109·22° Fahr. in a fatal case, one hour after admission. Levick records a temperature of 109·04° Fahr. in a man fifty-five years of age, who recovered from "Heat-fever" in the Pennsylvania Hospital (*Report* for 1868), and a similar record in a man aged forty, in the same place and year. He communicates also a number of similar observations—amongst which, one by Dowler is said to have reached as high as 113° Fahr. Temperatures of 103°, 104°, and 105° Fahr. are not uncommon a few hours after the attack commences (WOODMAN). See also Dr. Levick (in *American Journal of Medical Science*, vol. xxxvii., p. 40), who publishes cases with temperatures as high as 109·04° Fahr., *ending in recovery.*

The *heart's* action was very rapid and sharp (BARCLAY), excited and irregular (LONGMORE), the impulse and pulsation in the carotids being perceptible to the eye from a considerable distance. The pulse was frequent and sharp, and at first moderately full, giving the idea of a thinner fluid than blood circulating beneath the finger. Frothy mucus, sometimes clear, at other times of a brown colour, was in most instances ejected from the mouth and nose for some time before death, and often in large quantity.

When the disease was about to terminate fatally, the heart's action soon began to fail, the pulse to flutter, and the breathing to be irregular; and in a period varying from a few minutes to a few hours, death closed the scene. In nearly all the fatal cases there was occasional convulsive muscular movements of the extremities up to the time of death. These generally ushered in a state of complete coma, in which the patient gradually sank (LONGMORE).

In a large proportion of the cases, however, from the commencement of the attack to its termination in death, the patient never moved a limb, or even an eyelid; and a comparatively small number of the cases on the march from Jubbulpore to Calpee were from the first attended with convulsions. These generally began in the upper extremities, or in the muscles of the face; and in some cases they did not extend farther, the patient either becoming rapidly insensible or recovering. In other instances they extended to the whole of the voluntary muscles, and were of the most violent description, ceasing frequently for from two or three to fifteen or twenty minutes, and recurring again with increased severity. In a few instances the nervous irritability seemed as much increased as in hydrophobia; and some patients appeared to be in a state analogous to *somnambulism.* Although unconscious, and incapable of understanding or of answering questions, yet the countenance indicated the greatest terror,—the eyes rolled wildly about; and a few drops of water poured on the ground near him were sufficient to throw him into the most violent convulsions, and to elicit from him screams of agony. In most of these cases the convulsions ceased some time before death. The symptoms then became identical with those which characterize the ordinary course of the disease. In a few, however, the convulsions continued to the last; and in one or two death took place when the body was still contorted with them (BARCLAY).

The mortality from *insolatio* is equal to 42·734 *per cent.* (BARCLAY); or 43·3 *per cent.* (BUTLER); one half the cases (SWIFT); 560 per 1,000—the result of the following extension of Dr. Morehead's statistics:—

Observer.	Cases treated.	Deaths.
Sir Charles Napier, 1843, June 15, Nussurpoor,	44	43
Dr. Joshua Jones, 1864, Andersonville,	53	53
Mr. Hill (collected cases),	504	259
Dr. Taylor, Gazeepore,	115	16
Mr. Longmore, Barrackpore,	16	7
Mr. Lofthouse, 14th Light Dragoons,	80	10
Dr. Simpson, 71st Regiment,	25	6
Mr. Ewing, 95th Regiment,	60	17
Field Hospital, Hansi,	29	10
	926	521

In the cases which terminate favourably a gradual remission of the urgent symptoms takes place; but the irregularity of the heart's action and oppressed breathing may persist during the next day; and if the patient has been exposed to the influence of malaria, paroxysmal febrile phenomena may supervene.

The patient cannot be considered free from danger till the skin becomes cool and moist (SIMPSON, BARCLAY); indeed, a relapse of all the worst phenomena may occur even after free perspiration and sleep has been procured. Dr. Crawford relates such a case:—An orderly being left in charge of the patient during the night, with instructions to keep a cold lotion to his head, and to call the surgeon in the event of any change occurring, no matter how slight; the surgeon, visiting the hospital at one in the morning, when summoned to see another patient then taken ill, found the orderly asleep, and, to his horror, his patient moribund,—the face swollen, of a dark livid colour, the eyes protruding from their sockets, with stertorous breathing and spasmodic twitchings of the muscles of the chest and arms. The patient died shortly afterwards.

Morbid Anatomy.—In Mr. Longmore's cases, in which a vitiated atmosphere, from the want of ventilation, was associated with the extreme heat, the appearances after death were those usually found in death by asphyxia—namely, excessive engorgement of the lungs, amounting to complete obstruction of the pulmonary circulation. Some parts of the lungs had all the appearance of true interstitial apoplexy. Cerebral congestion was less marked in character and less constant in amount, and it seemed to be secondary to the failure of the due performance of pulmonary functions, resulting, perhaps (as Mr. Longmore suggests), from loss of tone in the vessels, and from the enfeebled action of the heart, consequent upon the imperfectly oxygenated blood it was receiving. The congestion of the head was generally expressed by engorgement of the vessels of the *pia mater* and *choroid plexus*, and by numerous *blood-puncta* in the substance of the cerebrum, as shown on section. The appearance of the brain indicated generally sanguineous determination without serous effusion; and when serous effusion had taken place, it was generally into the cavities of the lateral ventricles, and sometimes into the subarachnoid space. Other observers record engorgement of cerebral meninges, but no congestion of brain-substance (H. C. WOOD). Others again record marked cerebral congestion (FLINT); or the vessels of the dura mater full of dark liquid blood, easily pushed along by the handle of the scalpel. Others record the brain softened and breaking down by its own weight, or by the slightest pressure, with myriads of minute red points (torn vessels?) on its broken surface. The choroid flexus of a dark purple hue. No effusion into the ventricles. Uncoagulated blood on the under surface of the brain. Liver full of liquid blood, which poured from it on pressure like water from a saturated sponge (LEVICK). Veins of the membranes of the brain greatly congested. Brain moist, with large blood spots (BAUMLER).

The blood is always fluid (MOREHEAD). Its re-action has been recorded as faintly alkaline (LEVICK), and also as acid (H. C. WOOD). Levick, Gerhard, and H. C. Wood hold, that the most obvious, constant, and essential lesion in sunstroke is an *altered* state of the blood. That alteration indicates a loss of its life as shown by the change in its physical properties, especially in the inability of the fibrin to coagulate, the shrunken condition of the corpuscles, their escape from the blood-vessels, forming myriads of petechial spots, and rapid putrefaction, beginning before actual death has taken place.

Various opinions are entertained with regard to the mode of action of the various agents which combine to produce this singular affection of the nervous system. The phenomena during life and the *post-mortem* appearances are in accordance with death from coma, slowly induced, or from syncope.

The manner in which heat acts in the production of such asphyxia as is seen in cases of *sunstroke* has been variously interpreted by writers on the subject; and observations are very much to be desired as to the exact range of temperature of the body-heat in such cases. It is known, however, to be excessive, as already shown at p. 207; and the increased temperature of the blood, which results from prolonged exposure to great heat, must have a deleterious influence upon the constitution during the metamorphosis of tissue going on under such circumstances. Intense heat applied to the whole body tends to produce death by *syncope*, as in concussion of the brain (ALISON). Heat acting on the peripheral distribution of the nerves, and accumulating in the system, as it seems to do in *sunstroke*, produces such an effect on the heart, the lungs, and the brain, as to produce the phenomena of *syncope* and *coma*.

The pre-existing cutaneous derangement in all the cases, the total inaction of the skin, its dryness and intense heat, betoken an accumulation of heat in the blood which cannot fail to influence the delicate textures of the brain and lungs. The "embarrassed and heavy breathing;" the "sense of weight over the sternum" (LONGMORE); the "hurried and heavy breathing" (BARCLAY); the "catching at the chest;" the "constricted feeling, as if of approaching suffocation, caused by wind at a temperature of 112° Fahr." (M'GRIGOR), indicate that physiological state of "*anxietas*" which prompts to such acts of inspiration as are seen on the approach of *syncope*, or of *apnœa*, from depression of the nervous influence of the *medulla oblongata.*

Pollution of the blood, from the prolonged continuance of function under such circumstances as impair the normal action of the skin, the lungs, and the kidney, produced by the atmospheric conditions already described, and the phenomena of the disease in its severe form, denote the culmination of functional efforts to get rid of the rapidly accumulating elements of disintegration which must have resulted—as indicated by progressive emaciation, augmentation of animal heat, and total suppression of the cutaneous function.

Cases of sunstroke occurring "in quarters" seem, under such circumstances, to die from *coma*, inducing *apnœa;* and the most common complication is undoubtedly pulmonary congestion from oppression of the *medulla oblongata*, evidences of which are found on *post-mortem* examination in the majority of fatal cases, as originally pointed out by Dr. Marcus Hill (*Indian Annals*, vol. iii., October, 1855); and afterwards by Mr. Longmore (l. c., vol. vi., July, 1859, p. 396). In those cases of death by *coma*, the most striking point in the *post-mortem* appearances is the enormous congestion of the lungs; in which Dr. Parkes remarks, that although he has dissected men in a very large number of diseases, both in India and in England, he has never seen anything like the enormous congestion observed in two or three cases of this kind (*Practical Hygiene*, p. 345).

Pulmonary engorgement, however, is not always present. On the march and in the field the functional phenomena are chiefly of the cerebral, spinal, and sympathetic systems, as indicated by a painful state of nervous irritability from over-stimulation of intense heat. The long exposure of the eyes to the glare of the sun in camp may account for the more constant occurrence of their congested state in cases of *sunstroke* in the field, compared with such cases occurring in quarters.

The pallor of the face and of the surface generally seems also to be more an attribute of the disease in the field than in quarters, the cases being probably more anæmic in the field and more plethoric in quarters (BARCLAY). Practically, it has been observed, as Dr. Barclay points out, that there are at least four different ways in which death may occur in cases of *sunstroke*, which are necessary to be borne in mind with regard to the line of treatment necessary to follow.

(1.) The affections of the nervous system alone, more particularly those which occur during active exertion "in the sun," when the intense heat acts on the surface with the greatest power, producing at last a condition similar to severe concussion, and more or less instantaneous death by *syncope* (ALISON, CRAWFORD, BARCLAY, MOREHEAD). (2.) Death may be prolonged, when pulmonary complication may occur from destruction more or less complete, of the pulmonary circulation, and death by *asphyxia* ensues. Or, (3.) There may occur cerebral congestion, and death by *coma*. These states may, and generally do, co-exist together; and symptoms of either may predominate. (4.) Recovering from the immediate effects of these conditions, the patient may die two or three days afterwards, a febrile attack succeeding, with serous effusion within the cranium.

Two distinct pathological conditions are set forth by Dr. Levick, and described as "sunstroke," namely,—

(1.) Cases in which there is simple loss of nerve-force, caused by over-exertion during exposure to a high heat. Such cases may happen in the open air, under the vertical rays of the sun, or in the close and heated atmosphere of furnace-rooms, laundries, or of crowded barracks in India. The symptoms of such cases are, a feeble and moderately frequent pulse, moist skin, head generally hotter than the trunk, little or no change in the pupil—a tendency to syncope on the slighest exertion. Such cases generally recover.

(2.) Cases of a severer form, as a result of exposure to the sun's rays, or to a high temperature of the atmosphere, either within or out of doors. The attack is sudden, like a stroke, or with such slight premonitory symptoms as giddiness, confused blending of colours, and sharp pain in the head. The patient then falls unconscious, with stertorous breathing, restlessness and convulsions. The skin is pungently hot, body-temperature from 104° Fahr. to 110° Fahr., and the pulse so quick that it cannot be counted. Death may be immediate, or within six hours of seizure, finding its victims generally among the young, the robust, and the unacclimated, (*Pennsylvania Hosp. Reports*, vol. i.)

Causes.—Heat, and atmospheric conditions of the nature already indicated, seem to concur with the following predisposing circumstances to induce cases of *sunstroke:*—(1.) Plethora and unacclimation; (2.) Debilitating causes of every kind, particularly such as lower the tone of the nervous system or increase its irritability—*e. g.*, excessive fatigue and prolonged exposure in extreme temperature, prolonged marches, bad ventilation in tropical temperatures; (3.) A febrile state, from whatever cause. As a general rule, Dr. Barclay found that plethoric men incur greater danger from exposure than others; (4.) Intemperate habits; (5.) Exposure to an atmosphere highly charged with electricity (CRAWFORD, BARCLAY); (6.) During the season of prevalence of sunstroke the temperature would seem to have ranged from 96° to 120° Fahr. in the shade—extremes of atmospheric heat chiefly observed on the Coromandel Coast, Central India, the North-West Provinces, Scinde, and the Punjaub (MOREHEAD, l. c., p. 615).

The effects of the constant stimulation of excessive heat in producing nervous irritability and pervigilium cannot be doubted (MARCH, BARCLAY), and Indian medical officers can bear witness, from personal experience, to the extreme misery resulting from such excessive stimulation, in combination with the conditions already mentioned.

The investigations and experiments of Obernier show that the symptoms of *sunstroke* or *insolatio*, do not as was formerly supposed, depend on hyperæmia of the brain. The characteristic symptoms consist in *paralysis* of all the functions of the brain, occurring either suddenly or gradually. In the latter case the paralysis is preceded by excitement, delirium, and other symptoms of cerebral irritation. In our country, latitude and climate, the action of the sun's rays is not alone sufficient to induce such severe attacks as are seen in the tropics, but if great fatigue is superadded to a very hot day; and if, at the same time, the action of the skin is in abeyance, sweat being very little, then a severe attack may be the result. Radiation of body-heat is reduced to a minimum, while the production of body-heat is increased by active muscular exertion, there is overheating of the body and increase of body temperature to a degree incompatible with the continuance of life. Fulness of the veins of the meninges is no indication of a hyperæmic brain—under such conditions of fulness the brain tissue is generally anæmic.

Treatment.—Keeping in view the nature of this disease, and the various modes in which death may approach, the line of treatment may be indicated as follows, on the authority of Dr. Barclay:—

With regard to the class of cases in which death tends to occur suddenly from *syncope*, there is little opportunity afforded for treatment; but the measures indicated are—the *cold douche*, keeping the surface wet and exposed to a current of air, or assiduously fanned, exclusion of light as far as possible, the immediate employment of stimulants, external and internal, by the rectum as well as by the mouth. *Depletory measures of any kind are not to be thought of.* In the less rapidly decisive cases prompt treatment is of the greatest use; while delay is fraught with the greatest danger. The patient must be immediately stript of his outer clothing; and, being placed in a semi-recumbent position, the cold douche is to be applied, from a height of three or four feet, over his head and along his spine and chest, his extremities being at the same time sponged over with cold water. Relaxation of the pupil is the first symptom that shows the good effect of the treatment, which may require to be repeated several times, on account of returning insensibility; but if there is any evidence of failure of the pulse, this treatment must be discontinued, and the application of cold to the head is then all that can be borne. Stimulants administered by the rectum are recommended by Dr. Levick, to counteract the tendency to prostration. The hair is to be cut short as soon as possible, and a blister applied to the nape of the neck, the surface having previously been well sponged over with the *acetum lyttæ.* When the first violence of the attack is subdued, increasing confidence in the ultimate result may be indulged in so soon as vesication takes place; and in cases where insensibility recurs, after an interval of ten or twelve hours, it may be removed by the application of a second blister to the vertex; and which may be again repeated, there being no doubt as to the good effect it produces. A blister may also sometimes be applied along the spine in the worst cases. Stimulation by the use of the electro-galvanic current, with the moist sponges applied along the sides of the neck, chest, and epigastrium, ought also to be employed. *Sinapisms* ought generally to be applied to the extremities, and to the chest or sides.

As soon as possible after the employment of the douche, a strong purgative enema ought to be given, those of a stimulant nature being preferred. But as the enema may have to be repeated several times before any effect is produced on the bowels, it may be advisable to let the first enema be of a simple purgative character; and afterwards let it be followed up by turpentine enemata.

If cerebral congestion is indicated by the state of the eyes already described, a few leeches to the temples may relieve the congestion (LONGMORE, BARCLAY); but the prevailing opinion among medical officers of experience in the treatment of this disease is against the employment of blood-letting by venesection, even in severe cases. In all the cases which have been recorded, in which it has been employed, it seems to have been generally hurtful, and to have hastened the fatal termination.

In cases where the breathing is much oppressed, and the bronchial tubes loaded with mucus, the patient should be turned occasionally over on his face and side.

In the convulsive from of the disease, where the greatest irritability of the nervous system prevails, the douche is found to be inadmissible, from the agony which it occasions; and in such cases Dr. Barclay has found great benefit from the inhalation of chloroform. After a few inspirations the convulsions for the most part ceased, and sleep was very easily induced; but in one or two instances, after a considerable interval of consciousness, febrile symptoms increased in severity, coma supervened (probably effusion having occurred within the cranium), and was followed by death. But the cases in which chloroform can be used are comparatively few; and very great care is necessary in its employment, so that the inhalation may be suspended at once, as soon as any effect is produced upon the pulse.

In the most severe forms of the disease the principle of management is to reduce as quickly as possible the blood-heat. This is best effected by rubbing the body over with ice, as large as can be handled, as recommended by Dr. Levick, and as has been proved to be successful by Baümler and Dr. Woodman at the London Hospital. Tepid body baths, with cold affusion to the head and back of the neck, or general cold affusion may be employed; also injections of iced water. Keeping pieces of ice in the axilla until there is returning consciousness, which may not be for several hours, and, therefore, great caution is required in such use of ice, so as not to induce gangrene of the skin.

The sequelæ of sunstroke are generally persistent headache, fixed or shifting pain in the back, choreic movements of the fore-arm and hand, convulsive disorders, mental weakness. These symptoms are suggestive of hæmatoma of the dura mater, as a probable lesion in such cases, and indicate rest, attention to the functions of the skin, and change of air. When the pain is fixed and severe, long-continued counter-irritation at the nape of the neck, and a course of *iodide* of potassium may benefit the patient; but many cases are quite unrelieved by treatment, as the records of the invaliding hospital at Netley show every year (DR. MACLEAN).

Measures for the Prevention of Sunstroke.—(1.) All weak and sickly men should be weeded out and left behind, if a march is to be undertaken during hot weather in India; (2.) The costume should be suitable for the early morning hours before sunrise, as well as for the scorching period which follows, when the men are for the most part in their tents. It should consist of materials of slow conducting power, of a colour by which heat is not readily absorbed, and should be as loose and light as possible. A flannel shirt should be worn, to prevent the men being exposed to sudden chills, and a flannel belt round the loins may be worn with advantage, except in the hottest weather. The shirt-collar should either be open, or made so wide as to prevent all risk of its pressing injuriously on the veins of the neck. Above all, some other form of knapsack should be devised than that hitherto in use. The authorities, strongly urged by Dr. Parkes, have adopted a valise equipment, which is a great improvement, and which does away with the use of cross belts so injurious to the functions of the organs within the chest. The march ought not to be commenced too early in the morning. The troops should be on the *new* ground about an hour after sunrise, and the pace should never exceed three and a half miles an hour. There should be a halt for seven or eight minutes every hour, or oftener if the men are exhausted, and a longer halt half way, when each man should have a cup of coffee and a biscuit. They ought also to have their ration before starting in the morning. An ample supply of water should be provided for the men by "*bheesties*" being attached to each company, and always compelled to march with it. No man should be allowed to fall out without being accompanied by a non-commissioned officer, whose duty it should be to bring him to a medical officer at once, if sick, and if not, to bring him up to the column at the halt. All men so falling out should be brought up to hospital tents for examination immediately on the arrival of the regiment in camp. The men should carry nothing on the march but their rifles and ammunition—the quantity of the latter keing kept as small as is consistent with safety. They should be allowed to march "easy" and loosely clad, more particularly in passing through jungles or ravines. No halt should ever take place on such ground when better may be had within a moderate distance. When the sun is up, halts should be so timed that shelter may be obtained by open *topes* of trees. Camps should be formed on as high and open ground as possible. The sites for camps in India, marked out by the "official" pillars, are generally the most objectionable that could have been selected (BARCLAY, INDIAN SANITARY COMMISSIONERS, and others). As much space should be allowed between the tents as the ground will admit of. Tents should be pitched as speedily as possible, camels and elephants being provided for their transport—carts never. "*Kus-kus*" mats should be kept constantly wet.

Troops in the field during the continuance of the hot winds in India should have the best description of tents that can be got for hospital purposes, and be provided with the best known means for keeping them cool. An abundant supply of water in camp is of the utmost importance, care being taken that the bags containing it are in good repair, hung up within easy reach of the men, and kept always filled with water. Sentries should be under cover during the heat of the day; and the men should be instructed, whenever they go out in the extreme heat of the sun, to put a wetted towel or thick handkerchief over the head, under the cap, and around the back of the neck and face. The men should be encouraged to take exercise during the cool of the evening; or, at all events, to leave their tents, so as to permit of their thorough ventilation; and wherever it is practicable, bathing should not be omitted.

Rations of spirits ought certainly to be discontinued in India. It may be a question as to what should be substituted instead; but all authorities are agreed that the system of "spirit rations" tends to convert young soldiers into drunkards.

The sleeplessness which sometimes is a premonitory symptom of the disease has been relieved by opium, *provided there is no evidence of commencing insensibility,* and followed by a purge, if necessary (M. Wrench, late 12th R. Lancers).

CHRONIC HYDROCEPHALUS.

Latin Eq., *Hydrocephalus longus;* French Eq., *Hydrocéphale chronique;* German Eq., *Chronischer Hydrocephalus;* Italian Eq., *Idrocefalo cronico.*

Definition.—*Effusion of fluid in the sub-arachnoid space, so that the arachnoid becomes a sac filled with serum, or generally distending the ventricles of the brain, and differing from cerbre-spinal fluid in containing more albumen* (Hoppe); *occurring chiefly among children, and when occurring later in life, generally dating back to childhood. The tissue of the brain in contact with the fluid, especially the commissural parts, are apt to be broken down by œdema into a thin white pulp—(hydrocephalic or white softening).*

Pathology.—Chronic hydrocephalus was very little known till Dr. Whytt published his *Observations on Dropsy of the Brain* in 1768; but since that period, Dr. Fothergill, Dr. Watson, Dr. Cheyne, and a large number of other writers, have contributed to illustrate its nature.

There are a few cases in which effusion of serum into the ventricles, or into the cavity of the arachnoid, is unaccompanied by any morbid appearance of the brain or of its membranes whatever; and thus there are many instances in which hydrocephalus is not demonstrably inflammatory. More commonly, however, some lesion of the brain or its membrane does exist. Thus the substance of the brain is often marked with more bloody points than usual; the *septum lucidum,* the *fornix,* and other parts forming the walls of the ventricles, are often found in a state of softening—sometimes so soft that Gölis gives a case in which water could be expressed from it as from a sponge. The membranes also are sometimes congested, or opaque and thickened, with spots of lymph, evidently the effect of a low inflammation.

The quantity of fluid effused varies from a few tea-spoonfuls to seven or eight ounces, and of this the greater part is generally contained in the lateral ventricles, which from this cause are often so enlarged and distended that the finger placed on the brain, immediately over the ventricle, is sensible of a distinct fluctuation, while the anterior portion of the fornix is often so raised as to cause a free communication with the third ventricle, and perhaps with the fourth—at least, the effused fluid is found likewise distending those cavities. The quantity of fluid effused between the membranes is also often very great, sometimes filling the whole cavity of the arachnoid as well as the ventricles. Dr. Abercrombie has found serum effused even between the cranium and dura mater, and so also have other observers—a circumstance hardly known in any other disease. The choroid plexus or ventricular membrane, although in general pale and healthy, yet sometimes has the intercellular tissue so infiltrated that it appears studded with small cysts.

The first thing that strikes us on examining those patients who suffer from the chronic form of the disease is the enormous *size* of the head. The adult head averages about twenty-two inches in circumference. Dr. Bacon gives the case of a child whose head at three months had attained the enormous size of twenty-nine inches in circumference, (*Med.-Chir. Trans.*, vol. iii.) The head of Cardinal, a celebrated hydrocephalic man about London, long in St. Thomas's Hospital, and who afterwards died at Guy's, measured thirty-three inches and a half. There are instances, however, in which the cranium has been found unusually small, and of a conical shape, the sutures being closed before birth; and in these cases the children are still-born, or die shortly after delivery. When the disease comes on at later periods of life, and after the sutures are closed, the size of the skull is natural, the cavities within the brain distended, and its substance wasted and anæmic.

The *form* of the hydrocephalic head is also sometimes very irregular, one side being much larger than the other, while the bases of the orbits are for the most part convex instead of concave, thrusting the eye unnaturally forwards. On cutting through the skull the bones are found to be remarkably thin and transparent. The sutures, although generally closed towards the base of the skull, are commonly separated from each other by a wide extent of membrane at their superior portions. If, however, the patient should survive for several years, the membranous portion becomes ossified by a number of points forming "ossa Wormiana," and the sutures are thus partially closed. In some very few instances the sutures not only close, but the bones of the skull have a morbid thickness.

The membranes of the brain are generally thickened, and the fluid found effused either into the cavity of the arachnoid, into a cyst, or into the ventricles of the brain. When the fluid is contained within the cavity of the arachnoid, the brain is sometimes so compressed that there are instances in which hardly a vestige of that organ remains. A singular and rare variety of this affection occurs when the arachnoid sometimes protrudes through the *fontanelle* or open suture, and the *dura mater* and integuments yielding, a pyramidal bag, with its apex downwards, forms externally, which hangs low down the back like a jelly-bag.

When the effused fluid is contained in the ventricles, those cavities are found exceedingly dilated. The convolutions have no depressions, but appear unfolded. The *corpus callosum* is much raised, the *septum lucidum* is torn, or the grey commissure destroyed, and the white commissure elongated to the extent of an inch, so that the ventricles communicate. The parts at the base of the brain also, as the *corpora striata* and *thalami optici*, have scarcely any existence. In fact, the brain seems expanded into a large sac, in which the medullary and cortical substances are so confounded as to be undistinguishable. In Dr. Bacon's case the brain and membranes, even the dura mater, had ruptured, and a probe passed easily through the ethmoid bone into the nose, by whose orifices a considerable dribbling of the fluid took place during life. Gölis met with a case in which the water was contained in a cyst the size of a goose's egg, situated between the hemispheres of the brain of a child aged six years, and who died, the cyst being entire.

The quantity of fluid contained in the cranium in cases of chronic hydrocephalus varies from a few ounces to a few pounds. In the case of Cardinal it was found to exceed ten pints (nine pints in the cavity of the arachnoid, and one pint in the ventricles). Other cases have been, however, recorded in which the quantity has amounted to twenty pints.

Authors have greatly differed as to the nature of this disease. Some, considering it a mere increase of fluid from functional activity, have named it *dropsy of the brain*, most often congenital, and others have as constantly referred it to an inflammatory origin; but they have generally concurred in describing an acute and chronic form of the disease. The symptoms during life are due to the mechanical action of a variable amount of fluid, causing enlargement of the head.

In the chronic form of hydrocephalus certain parts of the base of the skull are liable to deviations, which are quite as important to be recognized as are the numerous extensive changes in the vault of the cranium. These changes in the base have been especially pointed out by Mr Prescott Hewett in the first volume of St. George's Hospital Reports. They occur especially in the lateral parts of the anterior and of the middle fossæ, especially the orbitar plates of the former. These orbitar plates are driven downwards, and either present a plain surface, oblique from before, backwards—or they may be perpendicular, or even convex, bulging into the orbit so as to reduce the orbit to a mere chink. The orbitar arch is more or less done away with; the frontal and orbitar portions of the bone may even present one continuous line convex in its whole length.

This deviation is usually associated with dropsy of the ventricles. If the orbitar plates are natural in shape and direction, then the effusion into the ventricles has either occurred at a period when the bones were not easily acted on by the pressure of fluid, or that the accumulation of fluid is on the surface of the brain—sub-arachnoidean and not ventricular. In the former—*the arachnoidean dropsy*—the fluid is limited to the upper and lateral parts of the surface of the brain; and however great may be the quantity of fluid, it cannot press on the bones at the base of the skull.

The deviation in the orbitar plates characteristic of *ventricular dropsy* is recognized during life by the state of the eye-balls. In such cases the eyes are more or less driven out of their sockets, and have a marked direction downwards; a great part of the pupil is hidden beneath the lower lid, and the white of the eye is much more uncovered than usual.

The deviations of the middle fossæ of the skull exist in the lateral portions, where, by the bulging out of the bones, the base in some cases undergoes extensive and strange alterations, leading to singular deformities about the face.

The deviations are of the following kinds:—The fore part of the squamous portions of the temporal bones, as well as the great wings of the sphenoid, may be driven outwards and downwards, and bulging into the zygomatic fossæ, these bones lie on a level with the alveolar margin of the superior maxillaries. The zygomatic arches, closely fitted to the expanded bones, are themselves flattened, especially the malar bones, to twice their natural width. The orbitar plates of the sphenoid project into the orbits, and are quite convex. In some instances, extending from the orbits in front of the spinous process of the occipital bone behind, large pouches bulge out on either side, as if the cheeks were blown out. These pouches are partly osseous and partly membraneous. The fluid in these pouches was ascertained in a case during life to communicate with that in the skull (Creutzwiezer, in *Rust's Mag.*, 1835, p. 463), and the whole contained within the cranial cavity.

The blood circulation and nutrition of the fundus of the eye also undergo great changes, and which may to some extent be explained by the compression of the cerebral substance from accumulation of fluid. As the fluid collects and the pressure increases, there occur:—(1.) Greater vascularity of the papillæ and choroid, with dilatation of the veins; (2.) An increase in the number of the vessels of the choroid; (3.) Partial or total serous infiltration of the papilæ; (4.) Atrophy of the choroid and its vessels; (5.) Atrophy of the optic nerve which may be complete.

Symptoms.—There are two forms of chronic hydrocephalus, the internal and the external, or hydrocephalus in which the membranes protrude. In either case, when this disease is fully formed, whether it be congenital or subsequent to birth, the child is generally of the most feeble intellect, irascible, often epileptic, and of extreme muscular debility, so that, if not palsied, he is hardly able to walk. Dr. Baillie met with an instance of chronic hydrocephalus in a man aged fifty-six, and whose ventricles contained six ounces of serous fluid. His chief symptoms were pain in the head, and a loss of Memory so great that he could recollect only five words, which he continually reiterated to express all his wants. Cardinal, whose case has been mentioned, had more Memory, and he prided himself, says Dr. Elliotson, in being able to say "The Belief," but he usually stumbled when he got to "Pontius Pilate." This man was epileptic, of very feeble intellect, and so irascible as to be always quarrelling with the patients, and would have been extremely difficult to manage except for his muscular debility. His case was one principally of *arachnoidean dropsy:* and seven pints of fluid were found in the cavity of the arachnoid. The perpendicular portion of the frontal bone was widely expanded; and its orbitar portion had become horizontal, while the arch of the orbit was complete. Heberden, however, mentions a case in which eight ounces of water were found in the ventricles of the brain, and yet no symptoms of hydrocephalus existed during life.

Causes.—The remote causes of hydrocephalus are often extremely obscure; but exposure to cold or heat, errors in diet, falls or blows on the head, the retrocession of a cutaneous eruption, or the extension of an inflammation of the ear, are among the most common. Disordered functions of the liver or alimentary canal is also a frequent cause, and so is dentition, or the presence of worms; and the circumstance of a child being seized in consequence of its feet having by accident been put into a bath of boiling water will show that any other extreme irritation will equally produce it. Many morbid poisons also will occasion it, as that of *scarlet fever, pertussis*, or *measles*; of *constitutional* diseases, *tuberculosis* is the most common exciting cause.[1]

Prognosis.—At any period the prognosis is most unfavourable, and Dr. Cheyne estimates the loss from confirmed hydrocephalus at six to one, and perhaps this is near the truth. The immediate danger in the cases of chronic hydrocephalus is not great, but few patients survive the age of puberty; Cardinal, however, lived to the age of thirty-two. Aurival speaks of another instance which reached forty-five; and Gall of a third who survived till fifty-four.

Treatment.—Professor Gölis of Vienna, recommends, after much experience, calomel in quarter or half-grain doses twice a day; also at the same time inunction of an eighth or a fourth of an ounce of mild mercurial ointment into the shaven scalp once in twenty-four hours. The head is to be kept constantly covered with flannel to prevent any check to perspiration. After a lapse of six or eight weeks, diuretics in the form of *acetate of potash*, or *tincture of squills*, or both, are to be given with the mercury. Counter-irritation by issues in the neck are also advocated by him to be kept open for months.

Compression of the head by bandaging, or by well applied strips of adhesive plaster are also to be tried over the whole cranium. Puncture of the skull, if the fluid is *arachnoidean*, may also prove of service,—not so if the fluid is *ventricular*. It is done with a small trocar and canula at the coronal suture, about an inch and a half from the anterior fontanelle, taking care to avoid the longitudinal sinus.

The fluid must be slowly evacuated and compression of the skull carefully maintained for many weeks, while the fluid is allowed to drain away. The operation has been successful in very young children.

Hygienic measures ought to be of the first importance in the rearing of delicate children, so as to prevent if possible the development of those conditions which lead to effusion of fluid within the cranium.

HYPERTROPHY OF THE BRAIN.

LATIN EQ., *Hypertrophia cerebri*; FRENCH EQ., *Hypertropie du cerveau*; GERMAN EQ., *Hypertrophie des Gehirns*; ITALIAN EQ., *Ipertrofia del cervello*.

Definition.—*Occasionally a congenital lesion in which there is a proliferation of the neuroglia, or connective tissue which unites the nerve elements, or* *developed after birth; is mainly limited to childhood, and is usually accompanied with cerebral irritation and paralysis.*

Pathology.—There is no increase of nerve-tissue elements but simply a growth (probably from frequent recurring hyperæmia) of the neuroglia. The lesion is occasionally congenital, and common in dwarfs; but more frequently it develops after birth, and is mainly limited to childhood. It commonly accompanies rickets, with deficient evolution of the thymus gland and hypertrophy of the lymphatics (ROKITANSKY).

Morbid Anatomy.—The brain, almost exclusively the cerebrum, appears larger and heavier than normal; so that when the calvarium is removed, the brain substance bulges out so much that it is impossible to replace it again. The membranes are thin and bloodless, with no trace of liquid in the subarachnoid space. The cerebral convolutions are flattened and pressed together, so that the sulci between them are obliterated. The ventricles are contracted, the brain tissue bloodless, dry, and elastic. If the lesion is developed early the skull is distended, as in congenital hydrocephalus; otherwise if the sutures are closed before the lesion commences the cranial wall is strained by absorption, and the inner table loses its smoothness and becomes rough. If the disease increases rapidly at an early age, the sutures may again open up, and the bones may be pressed apart (NIEMEYER).

Symptoms of irritation and paralysis are the result of the disturbance to the intra cranial circulation which results from the increase to the contents of the skull.

These symptoms, however, are not well expressed, so long as the sutures remain unclosed, and the skull can distend in proportion to the cerebral enlargement. When there is no enlargement of the head, the most common symptoms are attacks of epileptiform convulsions, especially when the arterial anæmia, usual with such cases, is temporarily increased.

A probable diagnosis only can be made where the skull is enlarged and hydrocephalus can be excluded.

The cause is always chronic, and death results generally during a severe attack of convulsion, or by complication with hæmorrhages and inflammatory exudations.

ATROPHY OF THE BRAIN.

LATIN EQ., *Atrophia cerebri*; FRENCH EQ., *Atrophie*; GERMAN EQ., *Atrophie des Gehirns*; ITALIAN EQ., *Atrofia del cervello.*

Definition.—*Diminution of brain substance, without induration or softening.*

Pathology.—The disease is usually congenital, or the consequence of some severe hydrocephalic disease, or of old age, or of long-standing exhausting diseases, especially in children, serum being effused in the space between the brain and its coverings, in order to supply the deficiency of bulk. Sometimes also one side may be more atrophied than another; the membranes appearing to be greatly shrivelled after the fluids escape; the convolutions are thin, and the sulci wide. The sufferers are generally idiotic, and possess but little use of their limbs. Andral gives a singular case, in which the patient, a girl, though an idiot, was able to do little errands in the neighbouring villages, and lived to an early adult age, yet, when examined after death, she was found to have no trace of cerebellum.

True atrophy of the brain consists in a diminution of the size or number of the brain's elements, without a previous destruction of them, or shrinking of cicatricial-like tissue (NIEMEYER). Two forms are to be distinguished—

(1.) Incomplete development, or congenital deficiency.

(2.) Retrogression, or disappearance of brain elements.

In the first form the development may be so incomplete that either there is perfect idiocy or life cannot be maintained.

There occurs also during fœtal life and the first year of childhood an arrest of growth on one side—the opposite side continuing to grow. Such cases may live and attain a certain degree of mental development.

After the development of the brain is complete, atrophy may set in primarily as *senile marasmus,* or it may be a result of exhausting and wasting diseases.

Local disease in the brain is also a cause of secondary atrophy, such as the lesions of *apoplexy, softening,* or *encephalitis.*

Idiocy with paralysis is usually associated with cerebral atrophy. Continued pressure on the brain, from tumors and from hydrocephalic effusions, also lead to atrophy. Also injury of peripheral nerves leads to atrophy of the central parts, whence these take their origin.

Morbid Anatomy.—The left side of the brain is most frequently atrophied by congenital deficiency.

The space created by the atrophy is usually filled by fluid, collected partly in the ventricles and mostly in the meninges. The skull is often unsymmetrical and thickened over the atrophied hemisphere.

Symptoms.—Weakness of intellect or decided idiocy are the usual accompaniments of atrophy.

The organs of special sense, especially the eye, are very obtuse, and the peripheral sensory nerves over the paralyzed half of the body is diminished. Paralysis and atrophy of the body on the side opposite the atrophied hemisphere are usual symptoms of the lesion. Paralysis is generally complete and combined with contractions of tendons. The bones are atrophied as well as other parts. Epilepsy is usual, and although the disease is not of itself fatal, intercurrent lesions usually very readily cause death.

WHITE SOFTENING OF THE BRAIN—*Syn.,* ATROPHIC SOFTENING.

LATIN EQ., *Cerebrum fluidum albens*—Idem valet, *Mollities atrophica;* FRENCH EQ., *Ramollissement blanc;* GERMAN EQ., *Weisse Gehirnerweichung;* ITALIAN EQ., *Rammollimento bianco*—Syn., *Atrofico.*

Definition.—*Softening, which is the result of imperfect nutrition, owing to* *deficient supply of blood, is in most instances dependent on mechanical obstruction, or degeneration of the cerebral arteries.*

Pathology.—There is undoubtedly a softening which results from obliteration of the cerebral arteries, consequent on coagulation of the blood in them, or on obstruction from clots of fibrin (CARSWELL, HASSE, KIRKES, SANKEY). Mr. Paget has also shown that such softening is more probably the result of a fatty degeneration of the brain-tissue, as a result of the deprivation of its nutrient material.

Thus softening of the cerebral substance, although characterized by some distinctive phenomena during life, cannot be regarded as an affection *sui generis,* apart from inflammation as now understood (see vol. i.), of which it is simply the result.

The two hemispheres of the brain suffer from ramollissement with nearly equal frequency; and ramollissement of the cerebellum is much more rare than of the brain. But it is not determined whether the grey or the white matter is the more liable to inflammation. Bennett has been led to consider the white as the more frequently softened; while Gluge is of opinion that it is more frequently the grey; but it must be remembered that the grey matter is naturally specifically lighter than the white, and probably also from its structure more amenable to the softening process.

The subjects of white softening are usually of advanced age, suffering from renal or cardiac disease, and in whom the arterial coats of the vessels within the brain are diseased. Or, some of these minute vessels may be oblitered by arrest of blood-clot within them, the result of embolism from diseased cardiac valves.

TUMORS OF THE BRAIN AND ITS MEMBRANES.

Definition.—*New growths, parasites, and aneurisms, implicating the central substance, the membranes of the brain, or both.*

Pathology.—Different forms of tumor occurring in the brain have been described under the following names:—

(a.) *Simple cerebral tumor* (scleroma), or indurated portions of the brain; (b.) *Gliomata;* (c.) *Adonoid, sarcomata,* or fleshy tumors; (d.) *Strumous tumor,* tubercular deposits (*tyroma*); (e.) *Gelatiniform* tumors (*myxomata, colloma*); (f.) *Adipose* and lardaceous tumors (*lipoma*); (g.) *Cholesteatma,* or pearl-like tumors; (h.) *Encysted tumors;* (1.) *Cysticerci and echinococci;* (2.) *Blood-cyst* (*hæmatoma*); (i.) *Fungoid tumor;* (k.) *Melanotic cyst;* (l.) *Cancers;* (m.) *Syphilomata;* (n.) *Aneurisms* (CRAIGIE, ROKITANSKY, VIRCHOW, NIEMEYER).

The origin and progress of these tumors are very obscure.

Morbid Anatomy.—(a.) *The simple cerebral tumor* takes the form of an induration, or *scleroma,* of particular regions of the brain. There is an absence of red colour, and the part acquires the consistence of the white of egg boiled hard. M. Dance gives the case of a patient who received a blow on the head about seven months before his death. He afterwards suffered from epistaxis and severe and frequent paroxysms of headache. At length he fell down in walking from the bath, and died convulsed in about a quarter of an hour. On inspecting the brain the convolutions were flattened; there was very little blood, and no serous fluid in the encephalon; but all the substance of the brain resembled white of egg boiled hard. Its weight and density were considerable, and it yielded and recovered its form like an elastic body. There was no trace of a red vessel; but the cortical substance was paler, and the medullary substance whiter than usual (*Répertoire Général d'Anatomie et de Physiologie*, 1828).

Isolated portions of brain in this condition have all the characters of a cerebral tumor, and have been described as such by Meckel, Abercrombie, Greding, Bouillaud, and others. It is more correct to regard them, however, as portions of brain indurated to an unusual degree, and changed in structure. On section the part has generally a pale yellow or orange-grey tint, and surrounded by some crebral softening. The ventricles contain fluid; and if the indurated mass approaches the convolutions, the membranes become opaque, thickened, and adherent. In some cases the part gets surrounded by a vascular cyst, and then it seems encysted; and in some instances the substance seems fibrous (CRAIGIE).

(b.) *Gliomata* result from proliferation of the *neuroglia*, or connective tissue of the brain; and as it grows up into a tumor, the surrounding or implicated nervous elements become destroyed. Microscopically they consist of roundish nuclei, distributed throughout a finely reticulated basement substance. They do not appear to form circumscribed tumors, but gradually to pass into healthy brain tissue; and are never seen to pass from the brain to the membranes of the brain, and generally originate amongst the medullary substance of the cerebrum. They may attain the size of one's fist, and are liable to hæmorrhagic exudations and fatty degeneration. In consistence they are between medullary cancer and healthy brain. On section they are seen to vary in colour from a whitish yellow to a bright-grey red, and abound in cut vessels. Such tumors have often been taken for cancers; and they usually occur under the fortieth year of life (NIEMEYER).

(c.) *Adenoid*, *sarcomata*, or *fleshy tumors* resemble a mass of flesh or an enlarged lymphatic gland, of a light pink or pale flesh colour, and of considerable firmness, and sometimes attaining the size of a large apple. They are usually attached to, and seem to proceed from the meninges, although they are for the most part imbedded in the cerebrum. Sometimes they contain cavities filled with fluid. Microscopically they are seen to consist of spindle-shaped cells, arranged in fibrous-like striæ. They differ from cancer in being sharply bounded, and often enclosed in a vascular membrane, from which they can be turned out. They are liable to calcareous degeneration (CRAIGIE, NIEMEYER, VIRCHOW).

(d.) *Strumous tumors, as tubercles of the brain*, or *tyroma*, are the most frequent form of cerebral tumors. They are composed of matter of a white or pale yellow colour, firm, like soft cheese, but less tough, granular, and friable.

The growth occurs in various forms :—

(1.) As one to four, five, or six homogeneous masses of considerable size, from that of a pea to a walnut, always without blood-vessels or trace of organic structure.

(2.) Numerous (30, 40, to 200) minute spherical masses disseminated through the substance of the brain (Reil, Chomel).

They are located most frequently in the *cerebellum* or *cerebrum;* more rarely in the *central ganglia.* They are generally surrounded by loose connective tissue, and on section may disclose softening at the centre, to the extent of a vomica containing tubercular pus; or the centre is marked by dark discoloration. Sometimes the main portion of the tumor passes gradually into the brain structure by a slightly translucent exudation, consisting of young tubercle elements (see page 10, vol. ii). Such exudation indicates that growth is still going on, that scrofula is in the act of tubercle formation (Craigie, Niemeyer).

(e.) *Gelatiniform tumors, colloids,* or *myxomata,* are growths of a peculiar soft jelly-like translucent material, like thin glue. Microscopically, they are composed of variously formed cells, imbedded in a mucous hyaline substance. They are found most frequently in the medullary substance of the cerebrum. They are liable to undergo blood extravasation. Dr. Craigie relates of such a tumor, that he found it extending over the base of the brain from the *optic chiasma* backwards to the *protuberance,* and on each side over the hemispheres, and in some parts as thick as half an inch, with an irregular outline. It had produced softening of the brain, over which it lay, and the base of the cranium, on which it rested, was absorbed, rough, and carious.

The earliest indications of the lesion were epileptic seizures and loss of memory.

(f.) *Adipose, lardaceous tumors,* or *lipoma,* are usually small, nodulated, and solid, attached to the dura mater; or they are cysts enclosing hairs, fat, or oily fluid, with excrescences from the inner wall of the cyst.

(g.) *Cholesteatoma, pearl-like* or *margaroid tumors,* consist of white glistening globular masses like pearls, each mass varying in magnitude from the size of vetches to pease. The colour is of a dead silvery, pearly, or waxy grey, and the aggregation of those form tumors varying in size from a nut or walnut to a small pippin apple. The mass is generally irregular and elongated. They are situated mostly on the base of the brain, cruri, or lower surface of the cerebellum, in the subarachnoid areolar tissue, between the *arachnoid* and the *pia mater.* They sometimes start from the cranial bones or meninges. They show no trace of organic structure, and chemically consist of cholestrine, with concrete layers of epidermic cells, enclosed in a delicate membrane (Cruveilhier, Craigie, Niemeyer).

(h.) *Encysted tumors* are chiefly of two kinds,—the one kind owing a parasitic source (see "Parasites," vol. i., 115). The parasites contained in them are always immature, and transition stages of tape-worm parasites occur, such as the *cysticerci* or *echinococci*—examples of each of which are preserved in the museum of the Army Medical department at Netley.

The *cysticerci* are usually found in large numbers, and generally in the grey substance. Occasionally they have commenced to undergo degeneration (calcareous), but the hooks can generally be recognized.

Ehinococci or *hydatid tumors,* watery bags, have been long known as existing in the brain. They form large vesicles, enclosed by their delicate germinal membrane, already fully described at p. 170, vol. i.

The blood cyst or hæmatoma has also been described, (see p. 199, vol. ii.)

(l.) *Cancers*, in the form of medullary fungus hæmatodes, or scirrhus, are similar in the brain to their structure elsewhere, as described at p. 903, vol. i. They sometimes start from the brain itself, from the *dura mater*, or the cranial bones; or, commencing in the external soft parts of the skull and neighbouring cavities, especially the orbit, they infiltrate by the vessels and nerve-sheaths, and so press into the skull. Although it is rare for cancers commencing inside to penetrate the membranes and skull bones, yet such growths frequently make their way outwards, and protrude the orbit, when they at once commence to soften and ulcerate. Such tumors often commence symmetrically (CRAIGIE, NIEMEYER).

(m.) *Syphilomata* occur in the brain as in other parts, as *gummata* or nodes, or as diffuse infiltrations. They have been already described at p. 889, vol. i.

(n.) *Aneurisms* of the cerebral arteries form tumors generally at the base of the brain, in connection with the blood-vessels there, especially the *basilar arteries*, those of the *corpus callosum*, and the *Sylvian fissure*, and the communicating artery of the *circle of Willis*. Usually they are about the size of a pea or a hazel nut, but sometimes are larger before they rupture.

Symptoms of Tumors are not characteristic as distinguished from *softening*, *abscess*, or local lesions, already described; and all produce certain common changes in the contiguous cerebral substance. All of them tend to some extent to derange the capillary circulation of the brain and its membranes, and to that extent they tend to produce cerebral symptoms and cerebral irritation. The greater the vascularity and congestion of the tumor, the greater will be the infiltration of the whole brain by serum. In connection with the membranes, excitement and congestion of the pia mater and grey substance of the hemispheres are attended with peculiar psychical phenomena, and often by the symptom of effusion into the ventricles.

Chronic headache and epiliptiform attacks are the most common accompaniments of cerebral tumors; and when the vascular excitement of tumor and brain is at the greatest, then loss of memory and of intellect, convulsions, or palsy are the results.

If the growth of the tumor is slow, and its size small, little influence may be exercised on the general functions of the brain; but according to its locality will certain special indications occur.

If the tumor affect the integrity of the *anterior* lobes of the brain, more or less loss of speech (*aphasia*) will be the result, depending either on disorder or abolition of memory, or on that of the muscular motions of the organs of speech (BOUILLAUD).

If the tumor affects the anterior part of the *central ganglia* (*corpus striatum*), the motions of the legs may be disordered or impaired; when the posterior region of these central parts (*optic thalamus*) are injured, the motions of the arms are likely to be impaired (SERRES, FOVILLE PINEL, GRANDCHAUMP).

But there is an utter absence of constant symptoms pathognomonic of tumors of the brain; and the diagnosis must be made (*a.*) from the history of the case; (*b.*) the physiological phenomena indicating the location of lesion at a point where tumors are known to be common, and other lesions rare; (*c.*) from peculiarities in the course of the disease.

The history of the case may show an exciting cause for brain disease—e.g., *parasites, syphilis, diseases of the heart* (hypertrophy or of the valves), *or of the arteries* (degeneration), *cancers, scrofula, injury to head, caries of petrous bone.* If it is considered that a tumor is the lesion or brain disease, its nature may also be recognized from some of these causes of brain disease.

The symptoms may be of brain disease or lesion, but cannot be regarded as indicating a tumor; and tumors may reach a large size without inducing symptoms, provided their growth is (1.) *slow;* (2.) not in the vicinity of the central ganglia; (3.) not in the vicinity of vessels, so as to interfere with the flow of blood to and from the brain; (4.) not vascular in themselves, so as to swell at times from overfulness of blood, or suddenly decrease from containing less blood; (5.) not of such a kind as will compress capillaries, so as to cause such a change in the brain as will lead to loss of function of the part and its connection where the tumor is located.

Severe, persistent, and sometimes intermittent headache is often for a long time the only symptom indicative of brain disease—presumably tumor—if the headache is usually intense and severe. The continuousness of the headache may be intermittent in the case of vascular tumors, when fulness or emptiness of vessels may cause exacerbations of pain.

Constant pain referred to the back of the head may point to a stretched tentorium as the probable site of a tumor in the posterior cerebral fossa.

Giddiness and vomiting accompanying headache also point to brain-lesion, and confirm the probability of tumor.

Local symptoms consist of *hyperæsthesia, neuralgia, formication, twitching, partial anæsthesia, partial paralysis*—all of which may commence before headache. Such symptoms are referable to disturbances of circulation in the vicinity of the lesion, and are not pathognomonic, because they are associated with so many other lesions; but if these phenomena are limited to *cranial nerves,* they are suspiciously indicative of brain disease—probably tumor.

Lastly, cerebral tumors sometimes cause blindness; and when blindness is due to cerebral tumor, the ophthalmoscope furnishes important information, as shown in the changes of the optic disc.

The observations of the late illustrious and amiable Von Graefe furnishes the following results:—

(1.) Simple swelling of the optic papillæ, with great tortuosity of the *vena centralis,* indicates obstructed venous circulation, most frequently seen with tumors encroaching on the orbit from within.

(2.) Slight inflammatory swelling of the optic papillæ, with less distinct venous hyperæmia and inflammation of retina, occurs, with inflammation of optic nerve and its membranes, extending to the retina.

(3.) Atrophy of optic nerve from a tumor, or from meningitis.

If the symptoms have come on so slowly and gradually as that the commencement cannot be fixed, the fact points to tumor rather than to any other lesion; unless where the tumors are vascular, when the symptoms may appear suddenly in consequence of vascular changes within the cranium.

Epileptiform convulsions occur more frquently from cerebral tumors than from any other cerebral lesion, and especially when the tumors are located in the cerebrum, and near the cortical substance.

When the tumor perforates, it is usually through the parietal or temporal bones; less often through the occipital, when it may sometimes be seen to move with respiration.

The general functions and organic processes of the body are sluggish,—pulse slow, and respirations rare; bowels constipated and urine scanty. The body may increase in bulk, or marasmus may be excessive, with bed-sores, and dropsy of the feet and ankles.

Prognosis—Always unfavourable. Death by *coma* is the most usual termination, either directly or by intercurrent disease.

Treatment.—Under these circumstances medicinal treatment can be of no avail; but the patient may be protected as much as possible from hyperæmia of the brain aggravating the morbid state. His nutrition and mode of life must be regulated, and also the functions of the bowels. Apoplectic or local inflammatory attacks may be met by local blood-letting and cold compresses. Hypodermic injections of morphia are also to be used in suitable cases.

If syphilis exists, or is suspected, anti-syphilitic remedies are at once to be adopted (Craigie and Niemeyer, from whose writings these statements are compiled).

Section IV.—Discoveries regarding the Structure and Function of the Spinal Cord, Illustrative of the Pathology of its Diseases.

The doctrines regarding the nature of the diseases associated with the structure and functions of this portion of the nervous system are in a state of transition. The inquiries into the structure of the cord, the arrangement of its minute component parts, and their connections with each other—with the brain on the one hand, and with the different parts of the body on the other—are only yet being investigated with all the care and appliances of modern research, and to some extent elucidated with success. Difficulties of the most formidable kind surround the anatomical, physiological, and pathological relations of the structures, functions, and diseases of the spinal cord and nerves. Much labour is required, in the first instance, to expose the cord in the dead body, and to examine its morbid states; while delicate manipulations and unwearied research, by the most experienced observers during the last half century, have served alike to show how mysterious and difficult is the subject in all its bearings, and how important and interesting the results. In this arduous task the names of anatomists, physiologists, and the busiest of physicians of this country have, in all such recent investigations, borne a distinguished and pre-eminent part.

In 1811 Sir Charles Bell took the initiative in these researches, and surprised the scientific world by his beautiful and interesting discovery regarding the distinct functions of the *anterior* and *posterior* roots of the spinal nerves.

The epoch of another era is marked by the interesting indication of the existence of the property of "*reflex action*," foreshadowed by Unzer and Prochaska, but which the ingenious and important investigations of the late Dr. Marshall Hall so largely contributed to develop. The names of John Reid, Grainger, Swan, Solly, R. B. Todd, and Bowman, stand prominently forward in this line of inquiry.

A third era is characterized by careful records of the history of cases, by the most inquisitive microscopic research into the diseased parts after the death of the patient, and by physiological experiments in living animals, to show the connections and arrangements of the more minute component parts of the spinal cord. (See results of a series of microscopic studies of the *medulla oblongata*, by Dr. John Dean, vol. xiv., "*Contributions to Knowledge*," by the Smithsonian Institution, U. S., America.) In this field of research the labourers are not few; and our own country is especially distinguished by the observations of Mr. Lockhart Clarke, the late Dr. R. B. Todd, and Mr. Bowman, Drs. Beale, Handfield Jones, Edward Meryon, J. W. Ogle, Roberts, and Bastian. Abroad, the persevering industry of Kölliker, Valentin, Stilling, Remak, Engel, Van der Kolk, Wagner, Brown-Séquard, and De Bois Reymond, and many others, have thrown much light on most of the important questions regarding the structure and functions of the spinal cord.

But "let not the spark be lost in the flame it has served to kindle." The beautiful discovery of Sir Charles Bell, while it astonished the scientific world at the time, soon expanded in magnitude and importance. From it, as from a mighty tree, the boughs that have dropped from its parent stem have borne to the earth those living blossoms which, germinating in their turn, are now daily expanding their branches into every land where the Science of Medicine is advancing.

It may be useful, in studying the nature of the diseases associated with the structure and functions of the spinal cord and nerves, to have a distinct conception of the more important general points which seem to have been established relative to its anatomy and physiology, and how far these are illustrated and supported by observations on its morbid anatomy. The following is a condensed statement of the general results established by the prolonged labours of Professor Schroeder Van der Kolk, of Utrecht, a more extended detail of which may be seen in *The Medico-Chirurgical Review* for January, 1857; and which are consistent in many respects with the observations of Valentin and Stilling:—(1.) The spinal cord (including the *medulla oblongata*) is the instrument through which the power of motion is generated and expressed, and the co-ordination of movements effected, and through which sensation is transmitted to the brain, and to the grey cerebro-spinal matter of the cerebro-spinal centres. (2.) Complete division of the spinal cord abolishes sensation and voluntary motion in all those parts of the body supplied with spinal nerves from below the seat of injury. Any lesion of the nerve-substance which results from a disease-process may do this if it destroys completely the nerve-matter at the seat of lesion. According to the region in which such a lesion may be situated, so are the different forms characteristic of this loss of power. The nearer the brain and *medulla oblongata* the more immediately fatal to life. If, at the junction of the cord with the *medulla oblongata*, such an injury were to happen, immediate death would ensue, as may be seen when an animal is "*pithed*." Generally speaking, from the head downwards the parts of the body are supplied, *seriatim*, by the nerves coming off from the spinal cord, so that according as the injury to the cord is situated lower and lower down, so does the paralysis affect less and less of the body from below upwards to the seat of disease. Thus in the cervical region, below the origin of the phrenic nerve, when the lesion is throughout a complete segment of the cord, and above the origin of the superior intercostal nerves, breathing is performed only by the diaphragm and abdominal muscles, while the intercostal muscles cease to act, and the ribs cease to rise and fall. In this condition the patient may live a few days, seldom a week, and never a month (WATSON). If the lesion of the segment occurs below the cervical region, in the upper dorsal portion, for example, the breathing is not affected, or but slightly, while the digestive functions become impaired, and paralysis of the trunk and lower limbs is complete. Such a condition is technically called *paraplegia*. It implies palsy and loss of feeling in the lower limbs, hips, loins, and trunk, according as the injury is higher or lower in the dorsal or lumbar region of the cord. A person in this condition may live a long time, depending greatly on the seat of injury; the higher up, generally the sooner fatal. (3.) The nerves which issue from the cord by two roots (motor and sensific) unite and form compound or mixed nerves, whose filaments or strands pass from their origins to their destinations isolated from each other; and everywhere throughout the body the sensific ramifications of the mixed nerve pass to the surface of the part which is moved by the muscles receiving their motor fibres from the same compound nerve; so that, while the former supply sensation to the part, the latter convey the stimulus to excite the act of motion (VAN DER KOLK). (4.) The anterior (*motor*) and posterior (*sensific*) roots of the compound or mixed spinal nerves are determined to have certain relations with the grey corpuscular elements of the spinal cord, accumulated throughout its central part in such a manner that a transverse section of the cord shows anterior and posterior *cornua* of this corpuscular grey substance. The corpuscular or multipolar cells, constituting the grey matter, are arranged in several distinct vertical columns, extending throughout the whole length of the cord, the anterior column being the principal. The most considerable of these cells constitute the columns of the anterior horns; next, those by the posterior commissure; then those between the anterior and posterior horns; and, lastly, those in the posterior horns, which rank the smallest in size, and it is even doubted by some whether they are really nerve-cells. These columns of corpuscular nerve-cells are larger and richer in nerve-cells at the cervical and lumbar enlargements; and the proportion of cells increases still more at all those points where the roots of the nerves penetrate into the cord to its grey substance. Thus, clusters of cells, occurring more or less apart, are placed above each other longitudinally (CLARKE, VAN DER KOLK). These so-called nerve-cells are extremely simple in structure. They consist of a more or less rounded mass of matter (cells?), having two or more caudate prolongations, with a circular centre or nucleus and nucleolus containing granular molecules. (5.) The anterior roots (*motor nerves*) spring from the cord itself, and take their origin out of the ganglionic cells of the anterior horn, each cluster of which forms a ganglionic plexus. (6.) The anterior medullary fibres of the cord are the channels through which the influence of the will from the brain is conveyed to these corpuscular or ganglionic plexuses, whence these motor nerves take their origin. (7.) The posterior roots (*sensific nerves*) have been traced towards groups of ganglionic cells, but have not been shown to communicate with them. (8.) These posterior roots have been traced to subdivide into two portions, which may be called sets of radicles or rootlets. At the posterior part of the cord one set of these posterior nerve-roots ascends immediately in the white substance, and appears to proceed directly towards and into the brain, thus constituting the channel of sensation; the other portion of the posterior nerve-roots transversely penetrates the white substance of the cord towards the posterior horn of the grey matter, through which it passes. Its fibres there mingle, in part, with certain nerve-fibres which are observed to encircle in a transverse direction the posterior horn of the grey substance of the cord; and, in part, they lose themselves amongst the ganglionic cells of the centre of the grey matter, between the anterior and the posterior horns. (9.) These latter rootlet nerve-fibres, of the great posterior roots, thus constitute the apparatus of reflex action, directing their stimulus through the group of ganglion-cells, with which they appear to be connected, into the ganglion-cells of the anterior horn, from whose plexus of cells the filaments of the motor roots arise. The posterior nerve-roots include, therefore, two descriptions of nerve-fibres—namely, those for sensation proper and those for reflex action; and hence the greater thickness (more than double) of the posterior roots compared with the anterior. (10.) These different cell-groups or plexuses of corpuscular cells appear to be united throughout the cord by longitudinal connecting-fibres, so that co-ordination of movement is effected. (11.) The roots of the motor nerves thus receive the excitement or stimulus to action from the group of ganglionic cells in which they originate. It is communicated to them, either through the will anteriorly, and from above downwards, or by the sensific nerves by their reflex filaments posteriorly, and from the peripheral parts of the body with which the sensiferous nerve-filaments are connected. An individual group of corpuscular cells (whence the motor roots arise) thus becomes susceptible to a *psychical* as well as to a *physical* stimulus. (12.) All reflex action takes place by a definite channel; and its operation seems to be regulated by communicating fibres, which bring the different plexuses of nerve-cells into communication with each other. Thus co-ordination and combination of movements are explained; and so also is the diffusion of action over remote regions, especially in great irritation of the cord, as in attacks of epilepsy and tetanus, or while the system is under the influence of the strychnine poison or hydrophobia. (13.) The grey matter of the cord seems chiefly to avail for *motion;* the posterior part being chiefly subservient to *reflex function*, and to the *co-ordination of motion;* while *sensation* is transmitted upwards exclusively by the posterior and lateral medullary columns to the encephalon, and has probably its proper centre in the *medulla oblongata.* Here, also, is probably localized the centre from which the more universal reflex movements and convulsions take their origin. Experience has convinced Professor Van der Kolk that the attention of the physician ought to be directed to the condition of the *medulla oblongata* in cases of epilepsy. He has frequently succeeded, where the disease has not been of long duration, in procuring a recovery through derivative applications to the nape of the neck; while the pathological changes resulting from protracted epilepsy are not unfrequently manifested by induration of the *medulla oblongata.* The morbid state of the spinal cord, which I accurately determined to exist in *four* cases of tetanus, is also consistent with statements regarding its minute anatomy and the relation of its parts. Each of the four cases exhibited one character in common, and pointed out the spinal cord as the seat of lesion in that formidable malady. The lesion referred to was not manifest to the naked eye, but was determined to exist with certainty by an examination of the specific gravity of the cord-substance. For this purpose the cord was separated from its nerves, and divided into parts of a uniform size, and the specific gravity of each determined. "Each of the four cases showed that the general specific gravity of the spinal cord throughout is increased in cases of tetanus, the average specific gravity of the healthy cord being 1·036. They showed, also, that a change is *suddenly* indicated about the region of the cord in immediate communication with the wounded part, and that in one case of idiopathic tetanus the change was uniform throughout. In the first case I examined, where the wound was on the occiput, the uppermost three inches of the cord were of the highest specific gravity, and the difference became *suddenly*, and not *gradually*, manifest at the fourth inch. In the third case a very marked difference was apparent when the cervical region was compared with the rest of the cord; and the difference was suddenly marked where the roots of the cervical and first dorsal nerves left the cord to form the brachial plexus. The wound in this instance was on the fingers. "In the last case the difference was suddenly manifested in the lowermost part of the cord, corresponding to the region where the nerves were in communication with the lower limbs, which were the seat of the injury" (*Glasgow Med. Jour.*, No. IV., Jan., 1854). Mr. Lockhart Clarke has since examined the cord in tetanus cases microscopically, and has found peculiar lesions of a most minute kind scattered throughout its substance (*Med.-Chir. Reports*, August, 1865); and more investigation shows a great increase in the growth of cell elements in the implicated portions connected by nerves with the site of injury. (14.) The anterior commissure of the cord is distinguished from the posterior by the decussation of the fibres. After their intersection, when traced downwards, these fibres are observed to be deflected so as to run in part along the margin of the anterior fissure, interlacing themselves within the white substance; and in part to enter the inner edges of the anterior grey horns, where they mingle with the encircling fibres already noticed, which spread themselves thence in the medullary columns of nerve-cells and join the longitudinal fibres. Their function is probably to maintain the motion of the right and left sides of the body. The fibres of the posterior commissure have a parallel course, without any intersection. (15.) Some observations on the secondary affections of the cord by Dr. Turck seem to show that when disease destroys a certain portion of the nervous centre, the strands, filaments, or cords of nerve-substance which proceed from that centre, or arrive there, subsequently degenerate, having ceased to receive or convey an impulse. This degeneration takes place in the same direction in which these strands or filaments convey impressions; thus in the centripetal fasciculi (posterior columns) the secondary affection occurs always in the centripetal direction; while in the centrifugal fasciculi (anterior columns) it shows itself in the centrifugal direction; and in the mixed fasciculi (lateral columns) it shows in both directions. Thus it happens that secondary affections of filaments or strands of fibres in the spinal cord may result from a lesion in one of the hemispheres of the brain; generally in about five weeks after the primary injury.

This observation may be regarded as a rule; and to some extent it may also explain cases of so-called "*paralysis musculaire progressive*" which have been described by Meryon, Cruveilhier, Aran, Valentiner, and Roberts; although the nervous lesion has only been demonstrated in some of the cases.

SECTION V.—DETAILED DESCRIPTION OF THE DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

INFLAMMATION.

Definition.—*Inflammation of the substance of the cord, or of its membrane, or of both.*

Pathology.—Two varieties of the disease are to be recognized more easily after death than during life, namely:—(*a.*) *Spinal meningitis;* and (*b.*) *Myelitis.* Of these in their order; and first—

(*a.*) SPINAL MENINGITIS.

LATIN EQ., *Meningitis spinalis;* FRENCH EQ., *Méningite spinale*; GERMAN EQ., *Entzündung der Häute*—Syn., *Meningitis spinalis;* ITALIAN EQ., *Meningitide spinale.*

Definition.—*Inflammation of the membranes of the spinal cord.*

Pathology.—The inflammatory states of the membranes of the cord, and the morbid effects they produce, are the same as those of the membranes of the brain. It probably never occurs as an independent disease.

The rachidian *dura mater* may be inflamed either at its free or at its adherent surface. On examining the spinal canal, after caries of the vertebræ, the areolar tissue uniting the dura mater to the walls of this cavity is often found greatly loaded with venous blood, and in some instances is broken down, so that the dura mater is entirely detached—a state most probably due to inflammation. This inflammation may terminate by resolution, or it may advance, and serum be effused between the osseous structure and the dura mater. In this site the effusion has no communication with the cavity of the cranium, because the dura mater of the cord, while it is but loosely attached by areolar tissue to the vertebræ, is very firmly attached round the margin of the *foramen magnum,* and especially to the basilar portion of the occipital bone.

The *dura mater* of the cord also appears liable to the ulcerative process and to gangrene. In a case given by Ollivier (vol. ii. p. 569) of a druggist who died on the twentieth day, after suffering from lumbar pains, with rigidity of the trunk and lower extremities, together with tetanic spasms, there was found, on cutting through the muscles of the lumbar region, half an ounce of pus, or more, which was traced to the cavity of the arachnoid, the rachidian *dura mater* having ulcerated and ruptured.

The spinal *arachnoid* and *pia mater* are liable to inflammations similar to the corresponding membranes of the brain.

Diffuse inflammation of all the folds of the *arachnoid of the cord* has often been observed; those membranes being red and injected for a greater or less extent; but it is almost solely observed in connection with inflammation of the *dura* and *pia mater* of the cord. Chronic inflammation of the arachnoid, resulting in partial thickening and ossification, may occur as a primary disease, *i.e.*, without any perceptible cause.

Effusion of serum, both into the cavity of the *spinal arachnoid* and between the *dura mater* and *pia mater* of the cord, is not uncommon. Such effusion communicates freely with the cavity of the cranium, so that fluid may pass easily from the one to the other.

Suppurative inflammation of the spinal membranes also occasionally takes place in the cavity of the *arachnoid.* Frequently, if the inflammation is acute, it is associated with disease of the *cerebellum,* or of the intracranial membranes; and in the chronic form it rarely exists, except in connection with caries of the vertebræ.

Effusion of fluid within the cavity of the spinal cord is analogous to fluid in the ventricles of the brain. It leads to compression of the cord, and paralysis as a symptom of such compression. *Hydrorachis,* as it is termed, is usually a congenital state, associated with a defective development of the vertebral arches. The paraplegia it induces generally extends to the sphincters and the bladder, producing incontinence of fæces and urine. The condition is indicated by a tumor at the lower end of the canal, over the region of the sacrum; and the cavity of such tumors communicates with the cavity of the vertebral canal; and the paralyzing influence is occasioned by the pressure of the fluid on the spinal cord. Congestion of the membranes may occur as a stage preliminary to all of these conditions.

Morbid Anatomy.—Inflammation of the *dura mater* of the cord occurs generally in more or less circumscribed patches. Injection, infiltration, and softening, are the usual results, which may go on to purulent formation or to permanent thickening of the membrane, which becomes adherent to the bone; or the pus may make its exit through the membrane when *diffuse meningitis of the cord* may supervene.

Inflammation of the *pia mater* of the cord is, on the other hand, generally extensive. It becomes injected and turgid with infiltration, so that in the subarachnoid space there is purulent effusion, flocculent exudation, or deposits. The spinal marrow is pale and bloodless.

Symptoms.—The symptoms of *rachidian arachnitis*, or *meningitis* of the cord, are often obscure at the commencement; but once formed, the disease is characterized by pains in the back, with affection of the muscles, and retention of urine. Paralysis does not occur, except by pressure produced by exudation of fluid, or by extension of inflammation and disorganization of the cord itself (MERYON). Ollivier, quoted by Meryon, gives a case of spontaneous *spinal meningitis*, in which the symptoms closely resembled those of structural lesion of the cord itself. The patient, a man aged twenty-four, was admitted into the Hôtel Dieu with obscure symptoms. On the fifth day of the disease he had paraplegia, with hyperæsthesia of the lower extremities. Three days afterwards the arms became partially paralyzed, semiflexed, and stiff; the right pupil was more dilated than the left; the association of ideas was slow. Two days subsequently he died. The cellular tissue which surrounds the dura mater of the spinal canal was filled with vessels injected with blood. The spinal cord was enveloped in a layer of gelatinous yellow matter, between the arachnoid and pia mater. It was most abundant over the lumbar enlargement, and extended as high as the third cervical vertebra. It was thickest where it covered the posterior columns of the cord (*Traite de la Moelle Epinière,* tome ii., p. 551, 2me, edition).

One marked symptom of congestion of the membranes, referred to in a case related by Dr. Meryon, is the difficulty experienced in walking on first arising after a night's rest. I have observed this to be a constant indication of spinal congestion; and it may be temporarily induced by *strychnine* or *nux vomica.*

The affection of the muscles varies from simple stiffness of the part to *opisthotonos.* This latter symptom is often limited to the neck or trunk, without the limbs participating, as in a case given by Rayer, in which the trunk and neck were drawn backwards, while the patient walked freely till the time of his death. In the case of a waggoner thrown off his cart, and pitched on his neck and shoulders, the neck was stiff, the jaw was locked, the body convulsed, and the patient delirious. It was not till the twelfth day, however, that the lower extremities became affected and palsied, when the patient sunk into a typhoid state and died. A large quantity of pus was likewise found in the spinal arachnoid cavity. Neither the pulse, nor the tongue, nor temperature, is much affected at the commencement of *spinal meningitis,* but towards its close the pulse becomes rapid and feeble, the tongue brown and dry, and the teeth fuliginous. The patient is now said to be "*typhoid,*" and he dies delirious or comatose. Retention of urine generally persists from the beginning to the termination of the disease. Constipation often exists to a great degree at first, but afterwards the bowels act regularly, or even suffer from diarrhœa.

Severe irritation of parts supplied by spinal nerves, followed by paralysis, indicate acute inflammation of the *pia mater* of the cord. Fever follows a chill, and severe pain in the back and the extremities is complained of, aggravated on motion and on pressure over the spine. Oposthotonis and contraction of the limbs may ensue, which intermit, and are greatly aggravated by movements of the spinal column. Pain in the extremities is often a very prominent symptom at the outset, and may be mistaken for rheumatism; but the most important sequence of symptoms is paralysis commencing in the lower limbs, extending to the bladder, rectum, and finally the upper extremities. The paralysis is at first incomplete, but afterwards increases, and anæsthesia succeeds.

Diagnosis.—The symptoms which distinguish *spinal arachnitis* from inflammation of the substance of the cord are *pain* and *contraction* or *convulsions* of the limbs. In pure *myelitis* there is seldom any severe or constant pain, while the limbs are generally palsied, and their sensations benumbed or lost. It is distinguished from *rheumatic lumbago* or *psoas abscess* by the affection of the limbs and of the bladder, and the gradual advance of paralysis from below upwards.

Prognosis.—Many authorities consider *spinal arachnitis* to be incurable; but numerous cases marked by the characteristic symptoms in a mild form do recover. On the other hand, more severe cases progress from bad to worse; and as paralysis extends to the medulla oblongata, death follows, or from catarrh of the bladder.

Treatment.—Spinal *arachnitis,* seldom depending on a morbid poison, is perhaps in all cases best treated by bleeding and mild purgatives. General bleeding is sometimes necessary; but local bleeding, either by cupping or leeches, along the vertebral column, is most useful, and cannot be omitted with safety. The medical treatment consists in moderate purging by the neutral salts, as the *sulphate of soda* or the *sulphate of magnesia;* for, as these act on the bladder as well as on the bowels, they are probably the best remedies. But whatever purgative may be selected, it will be proper to combine it with the *tincture of hyoscyamus,* or other mild opiate, to procure the patient some relief from his sufferings. Ergot of rye (*secale cornutum*) has been very much used in France; and Dr. Meryon speaks favourably of its effects, combined with *iodide of potassium,* in a case which manifested no complication of spinal effusion (*Medical Times and Gazette,* Aug. 31, 1863). The warm bath is an excellent adjuvant in the earlier stages of the disease; whilst in the latter stages *blisters, setons, moxæ,* or the *ointment of the tartrate of antimony,* are more beneficial, or at least are deserving of a trial. The paralyzing effusion may disappear during their use, combined with the action of diuretics. The *external* application of *belladonna* and *chloroform* will be found of essential service in diminishing the violent pain which accompanies meningitis of the cord (MERYON). Cold, in the form of bladders filled with ice, applied along the spinal cord, may be of service where congestion prevails.

Abstinence from all animal diet should be imperiously prescribed throughout the whole course of the disease.

In cases of *hydrorachis*, where a tumor is associated with *spina bifida*, iodine injections have been proposed (VELPEAU, DEBOUT, BRAINARD, GROSS, MERYON). In operating, the puncture should be very small, by a small flat curved needle, directed subcutaneously into the sac. A drain of the fluid may then be allowed to escape through a canula, and the injection used must be very weak at first, the object being to excite a slow process of inflammation in the cyst. One-eighth of a grain of *iodine*, and a quarter of a grain of the *iodide of potassium* in solution, is the quantity prescribed for injection by Dr. Gross. Dr. Brainard uses a solution composed of five-eighths of a grain of *iodine* and half a grain of *iodide of potassium* to one drachm of water. The puncture is to be closed with a twisted suture, and coated over with collodion. An anodyne should be administered, and the child kept lying on its face. Mr. W. M. Coates, of Salisbury, has been also successful by a similar method of treatment. If the life of the child be saved, paraplegia is still apt to remain, and perhaps involuntary defecation and micturition (Meryon, in *Brit. Med. Journal*, July 11, 1863, p. 28; also *Practical and Pathological Researches on the various Forms of Paralysis*, p. 25).

(*b*.) MYELITIS.

LATIN EQ., *Myelitis;* FRENCH EQ., *Myélite;* GERMAN EQ., *Entzündung des Marks*—Syn., *Myelitis;* ITALIAN EQ., *Mielitide.*

Definition.—*Inflammation of the substance of the spinal cord.*

Pathology.—As the spinal cord is a continuation of the brain, and similarly composed of medullary and cineritious matter, it is reasonable to expect that its diseases will be similar. Such is observed to be the case.

Inflammation of the cord may be diffuse. It is characterized, *post-mortem*, by a few more bloody points than usual, or by a slight red or rose-colour suffusion throughout its substance. There is reason to believe, writes Dr. Abercrombie, that inflammation of the substance of the cord, like the corresponding affection of the brain, may terminate fatally, either—(1.) In the *acute inflammatory stage;* (2.) By *ramollissement;* (3.) By *undefined supuration;* or (4.) By *abscess.*

The most common affection, however, is ramollissement or serous inflammation, in which the substance of the cord is greatly broken down and softened, so as to be sometimes reduced to a mere pulp; or so diffluent as to give the sensation of fluctuation under the finger. This disorganization may embrace the whole thickness of the cord, or sometimes only one of its columns, so that it is of variable extent. It is constant, however, and the centre or *grey* substance of the cord is more softened than that of the circumference or white substance. The ramollissement may exist in the cervical, dorsal, or lumbar portions; but it is most common in the lumbar, and after that in the cervical portions, or in those parts which contain the greatest quantity of *grey* substance, and the greatest number of blood-vessels. The part affected is generally swollen—a circumstance more striking than in similar diseases of the brain, because the spinal canal is large in proportion to its contents, compared with the cranium. The softened part is also generally ash-coloured or white. Some pathologists have regarded ramollissement of the cord as a particular alteration of the nervous system, resembling the effects of a contusion of soft parts, and the result of shock. It often occurs, however, when no shock has been received, and has not the least resemblance to a contusion of soft parts.

Induration of the spinal substance is another result of *myelitis*, and probably depends upon a form of inflammation in which fibrinous exudation becomes consolidated. Portal states he has found the cord of a cartilaginous hardness, while the membranes were red and inflamed; and Abercrombie gives a similar case.

The substance of the cord may likewise become infiltrated with pus, or it may be collected into an abscess. The fact of infiltration is perhaps questionable; but there can be no doubt of an abscess having occasionally formed in the substance of the cord. Velpeau gives a case, quoted by Dr. Abercrombie, in which an abscess was formed in the right column of the cervical portion of the cord, three inches long and two lines broad, while a smaller one existed in the left column (*Revue Méd.*, vol. ii., p. 217).

Morbid Anatomy.—When myelitis is confined to circumscribed spots it usually commences in the grey substance, and affects the whole thickness of the cord; or, commencing as "*central softening*," it extends widely through the grey substance. The affected part is swollen, and one section is of a red pulpy appearance, rising above the level of the section. By blood extravasation and changes, the various coloured softenings are brought about, such as red, white, brown, or yellow.

Symptoms.—The symptoms of *myelitis* are in general expressed by the parts below the lesion. In a few cases, however, the effects of the accidents are reflected from below upwards. In general, both upper or both lower limbs are affected; but in a few instances only one limb. The earliest symptoms are recognized in the fingers and toes, in the feeling of numbness, with a sensation of coldness extending up the limb. Shortly afterwards the patient complains of pain in the back, corresponding to the seat of greatest intensity of the inflammation. This is not constant; but when we make pressure with the finger over the spinous processes of the affected part, it may be augmented or only then felt. These symptoms are succeeded by impaired motion, and often likewise by diminished sensation of one or more limbs, followed by paraplegia or other form of palsy. If only one side of the cord be affected, the paralysis which results is confined to one side of the body. When the anterior columns chiefly are the seat of the inflammation, the paralysis which follows is that of muscular motion, but of sensation if the lesion exist in the posterior columns; and if a careful analysis be made of the several cases in which the grey substance of the cord has been implicated, it will be found that the function of reflex action has been deranged (MERYON). In the early stage, when congestion prevails, there is exaltation of tactile sense and of muscular contraction

Another marked symptom may be often distinguished—namely, a difficulty experienced in walking on first rising after a night's rest—a feature more or less constant in cases of spinal congestion. The palsied limbs may be either relaxed or permanently contracted: thus, the hand may be bent on the upper arm, or a leg be flexed upon the thigh, or the affected limb may be attacked with convulsive twitchings, or may beat incessantly. As the disease advances, the bladder becomes affected, and the patient is incapable of retaining his urine, from the sphincters being palsied. The action of the bowels is slow in the first instance; but towards the close of the disease the patient may be purged, and the stools pass involuntarily. If the disease be the result of an accident, the pulse is at first rapid and full; but if the disease be spontaneous, the pulse is generally natural, until the powers of life are broken down by the continuance of the affection. As death approaches, the nates and the prominent parts of the pelvic region, on which the body rests, ulcerate extensively, so that deep sloughs form; and although the patient, from anæsthesia, suffers no pain, he nevertheless ultimately sinks exhausted.

In *myelitis*, and in injuries of the spine from wounds and contusions, some differences in the symptoms have been observed, according to the seat of the injury. The disorganization of the substance of the cord entails a condition of paralysis more or less extensive, according to the seat and the extent of the inflammation. Every part of the body which receives its nerves from the spinal cord below the upper level of the structural disorganization is paralyzed; consequently, when destructive *myelitis* extends throughout the cord to the *fifth* pair of cervical nerves, the upper extremities are paralyzed, and all those parts which receive their nerve-power from a lower level of the cord are paralyzed too. If, again, the spinal cord be lacerated or divided above the origin of the phrenic nerves, or above the third cervical vertebra, death is the immediate consequence, the nervous influence being no longer transmitted with sufficient completeness to the diaphragm and other muscles of respiration. Petit gives two remarkable instances of this. The only son of a working man went into the shop of a neighbour, who in play raised the child from the ground by putting one hand under his chin and the other at the back of his head. The child, only six or seven years old, struggled, dislocated his head, and died immediately. There are a few cases, however, in which disease of these parts has not been immediately fatal. Thus, the *odontoid process* has been destroyed by caries, or the second cervical vertebra has been dislocated, and yet the patient has continued to live for some months, or even some years. A remarkable case of a diminished area of the occipital foramen, whence resulted great pressure on the cord, is related by Mr. Holberton in *The Medico-Chirurgical Transactions*, vol. xxiv., p. 108. The patient lived more than two years, the most remarkable symptom being an extremely slow pulse. In these chronic cases the formation of the disease is slow, so that the cord becomes accustomed to the gradually increasing pressure, and the respiration consequently still continues to be carried on principally, though feebly, by the muscles of the neck and shoulders, the diaphragm and intercostal muscles being more or less palsied.

When the injury, however, is below the origin of the phrenic nerves, or at the level of the fifth and sixth cervical vertabræ, the inspiration is free, but the expiration is laborious, for the intercostal and abdominal muscles are paralyzed, and incapable of assisting in that process. The patient can yawn, for that is an act accompanied by inspiration; but he cannot sneeze, for that is an act accompanied by expiration. At this point, also, the upper extremities are still palsied, both as relates to motion and to sensation. When the palsy of motion and of sensation is complete, the patient, during the short remaining period of his life, presents the extraordinary phenomena of a living head, with its sensibility and muscular powers unimpaired, attached to a trunk and extremities of whose existence he is only conscious by the sense of sight (Brodie).

The circulation of the blood is affected, and the action of the iris of both eyes, through the medium of the sympathetic nerves (Meryon). Another very common symptom connected with injuries of the upper portion of the cord is *priapism*, which may show itself about the second or third day after the accident, and generally subsides after the first fortnight. It sometimes occurs even when all sensation in the part itself is destroyed, so that the patient is not sensible of the introduction of the catheter.

If the injury be in the situation of the sixth and seventh cervical vertebræ, the palsy of motion and of sensation of the upper extremities is frequently imperfect, while it is complete in the trunk and lower extremities.

When the spinal cord has been injured in the part corresponding to the first dorsal vertebra, the upper extremities may still suffer from an incomplete palsy either of motion or of sensation, or both. When, however, the seat of the lesion is in a line with the second dorsal vertebra, the sensation and motion of the upper extremities remain unimpaired, but the respiration is still difficult, from the palsy of the intercostal and abdominal muscles. If the paralyzing influence do not extend through the entire thickness of the cord, then the lower extremities may preserve their sensation and motion, although the arms hang powerless, owing to the disease having dissected out, as it were, the groups of ganglionic cells which determine the action of certain sets of muscles, whilst the conductors of the Will for the movements of the legs pass by unscathed (Meryon). An illustrative case, occurring in the practice of M. Broussais, is recorded by Ollivier. The patient was a medical student, aged twenty-one, who had, as a result of acute myelitis, complete paralysis of the upper limbs, while the legs, as well as the bladder and rectum, retained their healthy power. He died on the eighth day after the attack. There was some increased vascularity of parts of the encephalon, considerable congestion of the sinuses of the cord with fluid blood, and much sanguineous effusion between the dura mater and the vertebral arches opposite the brachial enlargement of the cord, as well as a considerable quantity of red serum between the pia mater and arachnoid at the lower part. Four minute cartilaginous laminæ were found about the centre of the dorso-lumbar enlargement; and the opposite surfaces of the arachnoid were adherent at several points over the brachial enlargement, while part of the cord, especially the grey substance, was remarkably soft for about two inches. The remaining part of the cord below was somewhat softened.

When the disease occurs in the dorsal region between the two enlargements of the cord, the respiratory muscles, which are under the influence of the dorsal spinal nerves, obeying the laws of irritability, are frequently agitated by violent spasms, and the breathing is accomplished by short and painful efforts. If the disease extend to either enlargement, the arms or legs may participate in the spasmodic movements. But, as the work of disorganization goes on, anæsthesia of the surface and paralysis of the muscles, above alluded to, follow in the train of symptoms;—abdominal respiration, disturbed circulation, embarrassed digestion, difficult defecation, inefficient micturition, and all the consequences of these respective functional disturbances ensue.

The symptoms, when the injury is in the lumbar region, are not dissimilar to those of the dorsal region, except that the respiration is unaffected. When the lumbar region is the seat of the disease, the sound introduced into the bladder is more frequently covered with incrustations, and the patient also more commonly suffers from ulceration of the nates; but these symptoms, perhaps, result only in consequence of the patient surviving for a longer period than when the superior portions of the cord are affected. When the *myelitis* is limited to the lumbar enlargement of the spinal marrow, the convulsive movements occur at an early period of the disease, and cease *pari passu* with the disorganization of the cord. For a time the electro-muscular contractility is retained; but eventually it is almost always lost. The urine generally becomes alkaline, from retention by spasmodic contraction of the sphincters of the rectum and bladder; and *priapism* not unfrequently results as a reflex action from a distended bladder—a state which soon gives place to a negative condition, ushered in by reflex spasms of the legs during defecation and micturition (MERYON, l. c., p. 35).

In chronic affections of the cord the palsied limbs usually waste, and become atrophied.

In cases in which a limb has suffered from palsy, both of sensation and of motion, some singular phenomena of reflex action still remain. When a stimulus has been applied to the palsied limb, it occasions involuntary contraction of the muscles of that limb. Thus, when a feather is passed lightly over the hollow of the foot, as in tickling, convulsions occur in the limb, although the patient is quite unconscious that anything is touching his foot. These movements are quite independent of Volition, and vary in extent and force inversely with the degree of voluntary power possessed by the affected limb, being most forcible when the loss of voluntary power is most complete, and diminishes gradually in extent and force as that power is increased. In some instances, by irritating one leg, movements were caused not only in that leg, but also in the other leg: and similar phenomena have been observed by Sir G. Blane and others in decapitated animals, showing that (consistent with the anatomical observations already referred to) a portion of the cord may furnish a supply of nervous energy after disease has interrupted its connection with the brain.

In all cases where the lesion of the cord is of such a nature as to intercept the transmission of the influence of the Will from the brain, convulsive movements are apt to occur in the legs, and to continue for a long time even after the arms have become completely paralyzed. These phenomena, says Dr. Meryon, are doubtless owing to the excitement of disease reflected from the spinal marrow to the motor nerves of the lower extremities. Similar involuntary movements may be produced artificially by tickling the soles of the feet, whose nervous connection with the brain is cut off by the destruction of a portion of the cord. The influence of the stimulus is transmitted to the spine by the incident nerves, and is reflected back by the motor nerves, thereby producing spasmodic contractions of the limb, (Budd, in *Med.-Chir. Trans.*, vol. xxii.)

Diagnosis.—Diseases of the spinal cord and diseases of the brain are often followed by nearly similar symptoms; and, consequently, the one may be confounded with the other. But the history of the case, whether it has or has not been preceded by a fit of apoplexy or of epilepsy, will often enable us to determine the particular seat of the disorder. The antecedents and concomitant circumstances of every case must be carefully inquired into, and judged of upon their own merits. Myelitis is distinguished from lumbago, psoas abscess, and hip disease, by the absence of pain, and by the existence of palsy.

The characteristic symptoms of paralysis, as induced by destructive myelitis, are as follow (MERYON, l. c., p. 35):—(1.) Pain over that portion of the back which corresponds to the seat of inflammation. (2.) Lesions of sensation, giving rise to feelings of formication, creeping, prickling, tingling, heat, or cold, to numbness or complete anæsthesia. (3.) A gradual and progressive diminution of muscular power, distinguishing it from the paralysis which the French have denominated "*ataxie locomotrice progressive.*" (4.) An equable degree of paralysis in all the muscles which are implicated; for as in health the nerve-force is distributed to whole groups of muscles in an equal degree, so likewise is it annulled when the nervous centre is disorganized. (5.) Convulsive and reflex movements of the paralyzed muscles. (6.) Spasm or paralysis of the rectum and bladder. (7.) Alkaline urine. And, finally, (8.) The loss of electro-muscular contractility.

Prognosis.—There seems no reason to doubt that, as many perfectly recover from superficial inflammatory lesions of the brain after fever, so also many slight inflammatory affections of the substance of the cord may subside, and the patient do well. Many cases, indeed, even when the bladder is slightly affected, recover. If, however, the disease be of more than a few weeks' continuance, the prognosis is always grave. Still, some few cases recover, the palsied limb becoming withered. But more commonly the disease runs on, and the patient at length dies after a long illness. When paralysis has once supervened, there is great reason to fear that the inflamed portion of the cord has passed into a state of disorganization, and that the disease is incurable; but the prognosis also eventually depends in some degree on the precise seat of the lesion. If it be in the cervical region, the immediate danger is greater than when the lesion is in the dorsal region; in this latter, again, the prognosis is more unfavourable than when lower portions of the spinal marrow are affected; and when the patient retains the command over the motions of the rectum and bladder, and the acid character of the urine is persistent, the case is still more hopeful (MERYON).

Causes.—The more common causes of disease of this portion of the nervous system are accidental violence, as blows or falls. Affections of the cord, however, sometimes occur idiopathically, and the constitutional causes producing it are exceedingly undetermined. They have been referred to a suppression of the menses in the female, and to the suppression of a hæmorrhoidal flux in the male, while others attribute them to sitting in damp or wet clothes, to onanism, or to venereal excesses, and prolonged exertion in the erect posture without active movement, inordinate muscular exertion, the action of cold, and the development of tubercle.

No age is exempt from *myelitis*, but it occurs more frequently from ten years old and upwards. It is most common, however, in adult age, and more frequently attacks the male than the female sex.

Treatment.—In classing ramollissement of the cord with inflammation, it might appear necessarily to infer that the treatment should be strictly antiphlogistic. It is questionable, however, whether this mode of treatment is advantageous; and it may be laid down as a general rule that bleeding ought not to be had recourse to after palsy has occurred. It is then plainly improper; for, the nervous influence being intercepted, the powers of the lower part of the body are so reduced, that gangrene may rapidly supervene—a tendency which loss of blood greatly increases. Previous to that symptom it may be admissible; and it may be stated that so long as the affected muscles are convulsed, rigid, and irritable, the use of antiphlogistics and counter-irritants is indicated; but when the means calculated to subdue excitation have failed to arrest the further progress of the disease, and paralysis supervenes, stimulants are the only remedies which have the power of restoring to functional activity those nerve-cells and conducting fibres which are not irretrievably destroyed (MERYON). The chances of saving the patient by other antiphlogistic remedies mainly rest on acting on the alimentary canal so as to produce three or four motions in the twenty-four hours, and thus create such a derivation as in some degree to relieve the parts. The greater number of patients that recover are restored by these means. The particular purgative is not perhaps important; but as the neutral salts act not only on the intestines, but also on the bladder, that class of remedies is generally preferred. At the same time that the bowels are kept free, the patient should be allowed a liberal supply of wine, from six to eight ounces daily, and should have animal food at least once a day.

With respect to local counter-irritants, as blisters, moxas, or setons, little favourable can be said, unless they are employed previous to paralysis, as the tendency to gangrene renders their application of doubtful utility. When had recourse to, however, it will be found better to apply them above the seat of the disease than immediately over it, the greater vitality of the superior parts giving more assurance of the disposition of the wounds to heal. Of all stimulant remedies, *electricity* and *strychnine* are the most potent and the best; and *secale cornutum* has been recommended as a remedy possessing the same power as *strychnine* (BARBIER, PAYEN, MERYON). When there is no great pressure beyond that which simple congestion produces, nor actual disorganization of the spinal cord, the remedial power of *secale cornutum* is said to be very great. It seems especially to resuscitate the muscular contractility of the rectum and bladder, and pelvic viscera generally (GUERSANT, TROUSSEAU, BROWN-SÉQUARD, MERYON). The *ergot of rye* is best given in the form of *ethereal tincture, in doses of from ten to twenty drops twice or three times a day.* It does not relieve the reflex convulsions which are sometimes alleviated by *prussic acid, digitalis,* or *belladonna* (MERYON, l. c., p. 40), *chlorodyne,* or *chloro-morphine.*

After the local pain in the back has been subdued by the regular and repeated application of two or three leeches to the painful part, followed by a large warm poultice over the whole length of the spine, and a beladona plaster of equal length to follow it, or an occasional blister on each side of the spine, together with mild, warm purgatives, if necessary, Dr. Meryon has found no remedy so effectual as *strychnia, in the dose of one-twentieth of a grain, repeated more or less frequently* (*twice or three times a day*) *according to the evidence of its action.* It may be combined advantageously with *ipecacuanha* in cases where the intestinal mucus seems deficient.

The absence of pain and of spasmodic muscular contraction necessitates great caution in determining the precise moment when the spinal cord is likely to be benefited by the energetic excitement of *strychnia.* The internal administration of this remedy ought, therefore, to be always preceded by its external use, together with other stimulants in the form of embrocations over the spine, when the stage of excitation has been subdued.

Electricity, after the activity of inflammation has been subdued, is a therapeutic agent of great value; and the continuous current of galvanic electricity seems to be just as efficacious as the induction or intermittent current. But whether galvanism or electro-magnetism be employed, no high degree of tension is required for the restoration of muscular power; on the contrary, Dr. Meryon justly believes that the favourable course of many a case has been retarded by the employment of strong currents. Dr. Althaus, also, is in favour of weak currents.

In cases with a history of syphilis, and where there may be some reason to believe that hardening or induration of the cord or its membranes has taken place, the *iodide of potassium* may relieve the early phenomena, and by the aid of *setons,* for reasons already given (vol. i., p. 894), the progress of the disease may be held in abeyance so long as the discharge is maintained from the *seton.*

When disorganization of the spinal cord has become an accomplished fact, the disease is incurable; but yet the exigencies of the patient, as Dr. Meryon justly observes, are not the less pressing on the careful attention of the physician, and in nothing more so than in the protection which is called for against bed-sores, which will sometimes occur in spite of the greatest care (MERYON, *op. cit.,* p. 40).

SPINAL HÆMORRHAGE—*Syn.*, SPINAL APOPLEXY.

LATIN EQ., *Hæmorrhagia spinalis*—Idem valet, *Apoplexia spinalis;* FRENCH EQ., *Hémorrhagie*—Syn., *Apoplexie de la moelle;* GERMAN EQ., *Hæmorrhigie;* ITALIAN EQ., *Emorragia spinale.*

Definition.—*Hæmorrhage of the spinal marrow or of its coverings.*

Pathology.—It occurs usually as small ecchymoses, accompanying excessive hyperæmia, usually traceable to injuries of the spinal meninges. In some cases such hæmorrhages are the result of chronic degeneration of the cord. Extravasations of blood from meningeal hæmorrhage of the cord are found chiefly in the lower part of the spinal canal, and often fill the subarachnoid space; and the changes undergone, alike by the cord substance and the blood clot, are similar to those described as going on in the encephalon, under similar circumstances.

Symptoms.—Effusions between the meninges give rise to severe irritation, pains in the back, and spasms, especially in the parts supplied by the nerves going off below the seat of injury, and ending in paralysis. Perfect anæsthesia accompanies large extravasations and complete paralysis; so that it depends upon the position of the hæmorrhage—high up or low down—whether the paralysis will be sooner or later fatal.

If the symptoms of interrupted nerve-conduction from the brain are sudden—anæsthesia and loss of voluntary motion in the lower half of the body, combined with paralysis of the bladder—then it is most probable that the cause is rather hæmorrhage than inflammatory softening; and in other respects, what has been written regarding *spinal meningitis* and *myelitis* can only be repeated here.

SECTION VI.—DISEASES OF THE NERVES.

PARALYSIS.

LATIN EQ., *Paralysis;* FRENCH EQ., *Paralysie;* GERMAN EQ., *Paralyse*—Syn., *Lähmung;* ITALIAN EQ., *Paralisi.*

Definition.—Palsy *or* paralysis *are terms commonly restricted to those affections where voluntary motion is lost, in which the motor fibres are no longer acted upon by volition*—acinesis—(from α privative and κινησις, motion), *while the term* anæsthesia *implies a* palsy *of the nerves of sensation. Such various forms of palsy, or paralysis, are rather symptoms of a lesion than specific diseases.*

Pathology.—Palsy of a part is a very constant symptom of structural disease of the brain or of the spinal cord, but it occasionally occurs from a diseased state of a nerve-trunk itself. Palsy may affect a whole limb, or merely a part of one, and it is also limited to the muscles of certain regions. Palsy of a finger, a hand, an arm, or a leg, is an example of the first; palsy of the facial muscles of expression, from disease connected with the *portio dura* of the seventh pair, or facial nerve, is an example of the second.

The interesting clinical lectures of the late Dr. R. B. Todd describe the following conditions as giving rise to paralysis of motion:—(1.) Lesion of a nerve in some part of its course destroys its power of transmitting that force which is expressed by a contraction of the muscle into which the nerve is distributed. (2.) A lesion of some part of those centres of the nervous system whence the nerve takes its origin, or with which it may be connected directly or indirectly. And, as a co-relative statement, it may be written that whatever interferes materially with the conducting power of nerve-fibre, or the generating power of nerve-vesicle, will constitute a paralyzing lesion. Poisoning of the nervous matter will operate in this way. *Chloroform, ether, opium*, the poison of *lead* and of *mercury*, applied directly to the nerve-fibre of a living animal, suspends its power of transmitting the nervous force so long as the influence of the poison lasts. Poisons formed or retained in the living body operate in the same way, such as the retained *urinary biliary* principles, as in Bright's disease, the poisons of *rheumatism, gout*, and probably also *syphilis* in some of its more severe tertiary effects. Whatever, in short, impairs the natural structure of the nerve-matter, such as *inflammation, atrophy, condensation, softening* (spinal, as in the form of *tabes dorsalis*), *solution of continuity*, either by simply cutting the trunk of a nerve, or by the deliquescence of the nerve-fibres, as a result of disease, such as *white softening*, a *sanguineous* (*spinal apoplexy*) or *serous effusion, pressure on a nerve or a nervous centre*, are causes which will produce more or less complete paralysis. Of this there is abundant proof—*e. g.*, the inclusion of a nerve in a ligature, compression of a nerve by a tumor, a depressed piece of bone in fracture of the skull, or an apoplectic clot on the exterior of the brain.

Four different conditions of the muscles are to be observed in cases of paralysis, namely,—(1.) A condition little different from that of health, but less firm, less excitable by the galvanic stimulus, when the paralyzing lesion is not of an irritative kind. (2.) Complete relaxation of the muscles, characterized by softness, imperfect nourishment, and rapid wasting—so rapid, that in a few days the size of the limb experiences a marked diminution. Such muscles scarcely, if at all, respond to the galvanic stimulus. (3.) Contraction of the muscles, with rigidity and wasting (the flexors being always more rigid than the extensors)—a condition which is due to a chronic shortening of the muscles themselves, and generally associated with a form of muscular atrophy. (4.) Nutrition not impaired, constant firmness and rigidity, incomplete paralysis, increased susceptibility to galvanic stimulus.

The practical inferences to be drawn from these conditions are of great value in treatment. Thus, early rigidity indicates local bleeding or counter-irritation, while complete relaxation is against antiphlogistic treatment.

The different forms of paralysis of common occurrence are due—(1.) To disease of the brain or spinal cord, in which form the muscles may be rigid or relaxed, the disease of the brain being the result of *apoplexy, minute hæmorrhages, softening, renal disease, induration*—the result of *syphil-*

itic poison,—the *epileptic* or *choreic* state; (2.) To pressure upon or injury to a nerve; (3.) To hysteria; (4.) To the influence of poisons, such as *lead*, *arsenic*, *mercury*, and some kinds of food-grains in a diseased state, as from *lathyrus sativus*.

Typical forms of paralysis comprehend,—

(1.) *Paralysis of the Insane*, or *General Paralysis.*
(2.) *Hemiplegia.*
(3.) *Paraplegia.*
(4.) *Locomotor Ataxy.*
(5.) *Wasting Palsy*, or *Progressive Forms of Paralysis.*
(6.) *Infantile Paralysis.*
(7.) *Local Paralysis*—e. g., (*a.*) *Facial Palsy*, and (*b.*) *Scrivener's Palsy.*
(8.) *Paralysis from Blood-poisons.*
(9.) *Paralysis from Lead-poisoning*, or other poisons in food or drink, or specific disease, such as *Diphtheritic Paralysis.*
(10.) *Paralysis from Lathyrus Sativus.*

HEMIPLEGIA.

LATIN EQ., *Hemiplegia;* FRENCH EQ., *Hémiplégie;* GERMAN EQ., *Hemiplegie*—Syn., *Halbseitige lähmung;* ITALIAN EQ., *Emiplegia.*

Definition.—*A form of paralysis affecting one lateral half of the body.*

Pathology.—It is one of the commonest forms of paralysis, and one to which the name of "*paralytic stroke*" is commonly applied. Either half of the body may be affected; and the parts which are actually involved are generally the upper and lower extremities, the muscles of mastication, and the muscles of the tongue on one side; the respiratory process is not interfered with. The paralysis may be either complete or incomplete as regards motor power. Consciousness may or may not be perfectly retained; and whether it is so or not, the patient, when seized, falls to the ground, because the power of maintaining his equilibrium is destroyed by the failure of the antagonizing muscles of one-half of the body. The affected arm and leg lie as if lifeless on the side, all power of motion in them being destroyed. Stimulation, however, of the extremities of the sentient nerves, by slight titillation with the fingers, sometimes gives rise to active movements. The combined effect of such stimulation and the resulting movements is to cause considerable pain. These excited motions, to which the name of "*reflex actions*" has been given, occur almost exclusively in the lower extremities. Other involuntary movements of the paralyzed limbs occur simultaneously with the action of yawning, or result from emotions of surprise, joy, pleasure, grief, laughter, crying.

When the *fifth* nerve is implicated in the hemiplegia, the proper masticatory movements are unequal on the two sides, in consequence of paralysis of the temporal, masseter, and pterygoid muscles of the affected side; but the buccinator escapes. There is a want of force in the masseter muscles of the paralyzed side; and there is therefore apt to be lateral displacement or obliquity of the inferior maxilla, either when at rest or during mastication. The mesial line between the lower incisors is thus also apt not to correspond to that in the upper jaw.

Sometimes the *third* nerve may be paralyzed (indicating lesion of the *crus cerebri*), in which case the upper eyelid drops, and there is inability to raise it, combined with outward squint (*lateral diversion*), and dilated pupil.

The protrusion of the tongue is also characteristic in hemiplegia. It is pushed out towards the side affected, and on being retracted it is drawn towards the healthy side. Imperfect articulation exists in *hemiplegia*, and results from the palsy of the *ninth* and *fifth nerve;* and where the power of speech is wholly lost, or utterance is limited to monosyllables, the sign is not favourable, but denotes, with other symptoms, extensive lesion of the brain, superficial as well as deep. When deglutition is impaired, serious and extensive lesion of the brain, connected with the *vagus* or *glosso-pharyngeal nerve*, is denoted.

The lesions which give rise to hemiplegia are of the following kinds: —(1.) *Hemiplegia* typical of diseased brain depends on a softening clot, abscess, tumors, or exudation, involving or compressing some considerable portion of the centre of volition, such as the *corpus striatum* or *optic thalamus*, or in the immediate vicinity of those parts. Unless pressure be produced, or the fibres otherwise interfered with, paralysis does not result. The centre of volition " reaches from the *corpora striata* in the brain down the entire length of the anterior horns of grey matter of the spinal cord, and includes the *locus niger* in the *crus cerebri*, and much of the vesicular matter of the *mesocephalon* and of the *medulla oblongata*." Disease of any part of this range of structure is capable of producing paralysis; and the palsy is on the side of the body opposite the lesion. (2.) The intracranial portion of this range exercises the greatest and most extended influence in the production of voluntary movements, and the most extended and complete paralysis takes place from disease of the *intracranial portion*. (3.) In cases of cerebral disease it must be observed and remembered that the intracranial portion of the centre of volition for the left side of the body is situated on the right side, and that for the right side is situated on the left side of the cranium, while the *intraspinal* portions maintain relatively their respective sides. These two *inracranial* portions are connected by the oblique fibres coming from the anterior pyramidal column of the *medulla oblongata*, which (crossing from right to left) decussate with similar fibres proceeding from left to right. (4.) Exudations which are the result of inflammatory or other diseased states of the membranes of the brain, which, as they increase and cause pressure on the surface, transmit the effects of pressure downwards to the *corpus striatum* and *optic thalamus*, and thus cause paralysis. (5.) Morbid states which affect or destroy fibres of deeper-seated parts, such as the *crura cerebri*, or of the *cerebellum* in its crura (because a connection exists between the hemispheres of the cerebellum and the fibres of the pyramids in the *pons Varolii*), cause paralysis. (6.) The slow accession of paralysis following symptoms of irritation indicates a gradual morbid change, such as from exudations slowly taking place. (7.) An important feature in paralysis is due to the condition of the muscles, as to whether they are rigid or relaxed. (8.) Rigidity, whether supervening or occurring simultaneously with the paralysis, indicates irritative disease within the cranium. (9.) In cases where the rigid condition of the muscles does not come on till after a long period of paralysis, and after the muscles are perhaps wasted from atrophy, such a condition indicates loss of substance in the brain, and that the cicatrix is undergoing contraction. (10.) *Hemiplegia* typical of spinal disease, where the palsy is on the same side of the body as the disease, is caused by a lesion involving a lateral half of the spinal cord *below* the decussation of the pyramids. (11.) In *hemiplegia* typical of epilepsy, the lesion is transient, the palsy in general remaining only a few hours, or at most a few days, after the epileptic attack. It is termed *Epileptic Hemiplegia.* (12.) The *hemiplegia* associated with chorea occurs during acute attacks of that disease, and is termed *Choreic Hemiplegia.* (13.) The *hemiplegia* associated with hysteria is also of transient endurance. (14.) There is a form of *hemiplegia* where the morbid phenomena seems to spread from the periphery to the central parts. (15.) It may be associated also with disease of the surface, e. g., *arachnitis.* (See Wilks, *Guy's Reports,* vol. xii.)

In all these forms of hemiplegia the paralysis is a paralysis of motion more or less complete. In general, however, sensation is also more or less impaired; but it is less easy of recognition. In estimating the condition of the sentient functions, the same method is to be adopted which Weber devised in comparing the sensibility of the surface of the skin in different parts of the body. It consists in ascertaining how near the sharp points of a pair of compasses may be approximated, and yet be distinctly felt as two points by the patient. (See page 137 *ante.*)

The special lesions of the brain causing hemiplegia are—(1.) Obstruction of a principal cerebral artery by a plug of fibrin detached from an excrescence on one of the aortic or other valves of the heart,—the result of a former endocarditis (Kirkes and Virchow). (2.) A coagulum formed in an artery, resulting from some altered nutrition of its wall, and connected in general with a rheumatic or other morbid state of the blood. (3.) A softened state of the brain, such as the condition known as white softening, which follows the retardation and diminution of cerebral circulation by diseased arteries, or by the complete stoppage of an artery by a plug. (4.) Apoplexy, induration, or tumors—*e. g.*, tubercle or cancer in the parts indicated above.

Treatment.—The object to be aimed at in the early treatment of hemiplegia is to keep down the frequency and force of the heart's action. For this purpose strict maintenance of the horizontal position is necessary; and when Consciousness exists, let the Mind be kept tranquil by every means. Remove any local impediment to the easy flow of blood, and let the head be slightly raised, sufficient to prevent gravitation favouring the escape of blood from the ruptured vessels, but not so as to create any impediment to the flow, and so embarrass the action of the heart. Let the bowels be cleared out, so that no irritation from them may operate injuriously on the brain. In so doing, enemata ought to be employed; and failing these, castor oil or calomel, with compound jalap powder, may effect an efficient evacuation.

With regard to blood-letting, there are three objects to be attained,—(1.) To diminish an undue amount of blood to the head; (2.) To check hæmorrhage, or to prevent it; and (3.) To quiet the action of the heart (Todd).

The circumstances under which its use is *inadmissible* are thus defined by Dr. Todd:—If the patient be cold and collapsed; if the heart's action be feeble and intermittent; if there be an anæmic state; if the patient be of advanced age; if there is evidence of extensive disease of the arterial system or of the heart; or, lastly, if it can be ascertained that already a large amount of hæmorrhage has taken place into the brain;—these, singly or conjointly, are reasons why bleeding *ought* NOT *to be resorted to.* If none of these objections exist, it is to be considered whether any of the indications noticed require to be fulfilled, and whether they can be fulfilled by a local or general blood-letting. Modern investigations show that the brain is not generally in a hyperæmic state; so that it is chiefly to check or to prevent hæmorrhage that bleeding is to be resorted to in such cases. The sudden or rapid abstraction of a moderate quantity of blood, either from the arm or temple, or by skilful cupping, may check hæmorrhage, but the quantity taken should be small; and so, likewise, the quantity drawn ought to be moderate if it is desired merely to lessen the frequency and force of the heart's action. "Generally," writes this distinguished physician, "I have come to the conclusion that, in cases of white softening, you are less likely to err by omitting than by adopting the practice." The rigidity of the muscles, which comes on very early, and which indicates an inflammatory process going on round the clot, is to be combated by urinary and alvine evacuants, and by counter-irritation. It is not desirable to interfere in the *later* forms of muscular rigidity. With regard to the use of expedients for promoting the restoration of the paralyzed limbs to their normal conditions, Dr. Todd writes that he knows of nothing which is of more decided benefit than a regulated system of exercise—active when the patient is capable of it, passive when he is not.

Any congestion of the spinal cord apt to supervene on cerebral hemiplegia must be diminished. The patient should not lie on his back, but, if possible, flat on his belly, the arms and legs being encased in flannel, and placed at a lower level than the spine. A hot douche may also be applied to the spine, the water falling from a height of at least *four* or *five* feet, through a tube *three-quarters* of an inch or *one inch* in diameter; and its temperature should be between 98° and 100° Fahr. The applications should be continued two or three minutes, and continued daily for some time.

Dry cupping over various parts of the spine may also be of service, and so also may blisters, moxas, cauteries (BBOWN-SÉQUARD).

The most useful internal remedies are those which tend to lessen congestion in the cord, namely, *belladonna* and *ergot of rye.* The dose of *ergot* in powder may be at first *three grains twice a day,* and gradually *the dose may be increased till it reaches six grains twice a day.*

Belladonna may be applied to the spine in the form of a plaster *four inches wide and six or seven inches long;* and if no amelioration of the symptoms follows in a few weeks, the *extract of belladonna* may be given in doses of *a quarter or a third of a grain twice daily;* and if after six or eight weeks of this treatment no improvement is observed, Dr. Brown-Séquard recommends that *iodide of potassium* in doses of *five* or *six grains twice a day* may be given in addition to the *belladonna.* If meningitis is believed to exist along with chronic myelitis, then the *iodide of potassium* should be given from the very commencement, along with the *belladonna*, or with *secale cornutum*, as recommended by Dr. Meryon. It has been given in doses of x to xviii grains daily for two months with success, in a case where loss of feeling over the whole of one side was complete for two years (Briquet). Spermatorrhœa may be relieved by pressure applied over the region of the prostate (Thomas and Meryon).

To prevent atrophy of paralyzed limbs, the application of galvanic currents and the use of the flesh brush are recommended; and when œdema of the limbs exists, a warm bath to the part every night is of service. The bowels must be kept open; and if anodynes are required, *opium* should be avoided, and *hyoscyamus, conium,* or *Indian hemp* should be used instead. Dr. Brown-Séquard recommends *hyoscyamus* *Iodide of potassium* ought to be taken before food in the morning, and an hour before dinner, so as to avoid its presumed decomposition by the gastric juice.

The nutrition of the spinal cord ought to be improved by the daily use of the cold douche; and sea bathing may be of service. If symptoms of irritation do not exist, *belladonna* is not to be given: it is only in cases of congestion that it may be useful in diminishing paralysis. And the same rule applies to *ergot of rye. Strychnine* increases the amount of blood in the spinal cord. It may be employed in paralysis only when there is no sign of irritation, and ought to be avoided when there are signs of congestion or irritation (Brown-Séquard).

PARAPLEGIA.

Latin Eq., *Paraplegia;* French Eq., *Paraplégie;* German Eq., *Paraplegie;* Italian Eq., *Paraplegia.*

Definition.—*A form of paralysis affecting the lower half of the body, in which both legs, and perhaps also some of the muscles of the bladder and rectum, are paralyzed.*

Pathology.—Of this kind of paralysis there are at least two forms, which differ as to their mode of origin—namely, (A.) *Reflex Paraplegia;* (B.) *Paraplegia due to myelitis in some one of its numerous forms.*

(A.) *Reflex paraplegia* or *reflex paralysis* are terms first proposed and used by Brown-Séquard, to whom we owe so much of our accurate knowledge and methods of investigating this subject. It has also been proposed to name it *paralysis from peripheral irritation* (Jaccoud).

One theory of this form of paralysis is, that reflex contraction of the blood-vessels of the medulla takes place, and that paralysis results from blood deprivation—*paralysis from reflex ischœmia, i. e.* from *reflex vascular contraction* producing *ischœmia.*

But whatever the correct interpretation of the physiological phenomena may be, it is certain that physicians and surgeons at different times have met with and recorded cases of paralysis which the amount of disease present in the nervous centre or its covering after death would not account for, which blood-poisoning would not account for, but which were found to be uniformly associated with injuries or disease of parts or organs remote, and not directly contiguous to the spinal marrow. Such cases were assumed to be cases of "reflex paralysis," and they have now been long recognized and described by various observers. Mr. Stanley, in 1833, published a paper in *The Medico-Chirurgical Transactions* "On Irritation of the Spinal Cord and its Nerves in connection with Disease of the Kidneys." He there records cases of paraplegia in which no morbid appearances were detected in the cerebro-spinal axis, but where inflammation of the bladder or kidneys, or gonorrhœa, had existed for some time. Similar cases of "Urinary Paraplegia" have been recorded by Romberg, Graves, Rayer, and several other writers. Mr. Spencer Wells recorded in *The Medical Times and Gazette* cases of this kind, in 1857, having made them the basis of an excellent clinical lecture at the Grosvenor School of Medicine. Dr. Brown-Séquard, in a series of four lectures, published in 1861, relates the grounds of diagnosis and the principles of treatment of these various forms of paraplegia. Lastly, Dr. Meryon describes seven forms of paralysis as due to reflex action, namely—(1.) *Emotional paralysis.* A case of this kind is described by Dr. Meryon, in his work already referred to, p. 172. Dr. Wiblin, of Southampton, in *The Lancet* of August 11, 1860, records a case of so-called emotional paralysis. The subject of that case died in October, 1864, and I had an opportunity of removing the brain and cord in connection, which I sent to Mr. Lockhart Clarke for his inspection. From the *post-mortem* appearances, I regarded the case as one of *chronic meningitis,* associated with disease of the blood-vessels. In this view Mr. Lockhart Clarke concurred, and mentioned that he found cysts also in different parts of the brain—two or three small ones in the white substance round the *corpus dentatum ceribelli.* One of these contained a fluid that was perfectly milky, and appeared under the microscope in the form of granular globules of oil, about twice the size of pus-globules, with a multitude of oily molecules. Several cysts were found in the *cerebrum;* and the *corpus quadratum* of the right side was hollowed out into a large cyst. Such lesions remove this case from the class of so-called "*Emotional paralysis.*"

(2.) Pregnancy is apt to induce such *reflex paraplegia,* or sometimes *hemiplegia* or *amaurosis* (CHURCHILL).

(3.) *Neurolytic paralysis,* in which, from no adequate cause, the functions of the cord seem suspended for a time, associated generally with exposure to cold and wet.

(4.) Paralysis from the *irritation of worms* in the intestinal canal (DAVAINE, MERYON).

(5.) Paralysis from the *irritation of teething* in children.

(6.) *Urinary paralysis.*

(7.) Paralysis from *uterine disease,* as from *dysmenorrhœa,* or *metritis.*

(8.) *Mechanical injury* of one part may give rise to reflex paralysis in another part; as in cases of gunshot wounds recorded by Dr. S. W. Mitchell, Morehouse, and Keen of the United States Army (War Department, Surgeon-General's Office, *Circular,* No. 6).

As examples of this form of paralysis, they record the following cases:

—(1.) A wound involving the muscles of the right thigh is followed by reflex paralysis of the right arm and left leg; (2.) A wound of the right thigh, is followed by paralysis of right arm; and several others; but in all, however great the loss of motion or sensation at first, the power of movement and sensation began to return early, and continued to improve till the part had nearly recovered all its normal powers; but in nearly all, some amount of paralysis continues permanent, and the part remains weak.

The views entertained by these several writers regarding the existence, especially, of a "Reflex Paraplegia" have been called in question by many able pathologists, and chiefly by Drs. Gull, Nasse, and Valentiner; but the facts on which the belief in "Reflex Paraplegia" rests, and which show that "a paralysis of the lower limbs may be caused by some alteration in the *periphery*, or the trunk of various sensitive nerves," may be shortly stated as follows:—(1.) A very rapid cure of the paraplegia follows the removal or cure of the primary disease which involved the peripheric sensitive nerves. Such rapid cures do not result in cases of paraplegia when the spinal cord or its membranes are primarily diseased. (2.) There are certain characteristics of *reflex paralysis* which tend to show how distinct it is from the cases of paralysis depending on organic alteration of the nervous centres. These characteristics are,—(*a.*) An outside excitation connected with some morbidly sensitive surface or nerve, and which exists for some time before the paralysis comes on—e. g., *stricture of the urethra, gonorrhœa, disease of the kidney, prolapse of the womb*, and the like; (*b.*) Variations in the degree of this excitation are followed by variations in the paralysis; (*c.*) The cure of the paralysis follows the subsidence of the primary disease.

Cases of "reflex paralysis" are also well known to occur in all the upper parts of the body—*e. g.*, paralysis of the optic nerve is sometimes due to injury of the frontal nerve; paralysis of the auditory nerve is sometimes due to neuralgia of the face; local palsy, as of the eye, the neck, the trunk, the bladder, or the rectum, is sometimes due to teething, worms, or other sources of excitement of the sensitive nerves, terminating in the mucous membranes or the skin; and the cure of amaurosis after the expulsion of worms is by no means uncommon.

Cases of paraplegia following diseases of the womb, and cured after the cure of this disease, are recorded by Romberg, Hunt, Wolf, Brown-Séquard, and others. Cases of paraplegia due to a disease of the urethra are on record by Graves, Wells, and others. Cases of paralysis consequent on inflammation of the bladder—gonorrhœal cystitis—have been related by Rayer and Brown-Séquard. Cases of reflex paralysis are also due to diseases of the prostate or kidney, to enteritis, dysentery, diphtheria, and affections of the lungs and pleura. The paralysis which attends teething has been known to increase and decrease as the molars are cut. Irritation of the nerves of the skin, generally following cold and wet, has been known to induce reflex paraplegia (*neurolytic paralysis* of Handfield Jones); and so also has disease of the knee-joint. In all these instances the paralysis follows the primary disease which is the cause of the local "peripheric excitation." Increase or decrease of this excitation and of the attendant paralysis, according as the cause persists or if suppressed, is among the recognized phenomena of such cases.

There are at least two ways by which the reflex action from the outside irritation may induce paraplegia, namely—First, *the reflex contraction of blood-vessels.* Brown-Séquard has the great merit of having actually demonstrated this. We know that the blood-vessels can contract with energy on the application of a stimulus or excitant, sometimes to the extent of a real and prolonged spasm; and we know that the nervous system is amply supplied with blood-vessels. In *three* different places—namely, (1.) *In the spinal cord;* (2.) *In the motor nerves;* (3.) *In the muscles*—such a contraction of blood-vessels is found to cause "paraplegia." In the vessels of the pia mater of the spinal cord, Dr. Brown-Séquard has seen how the vessels contract when a ligature was applied on the *hilus* of the kidney, or when a similar operation was performed on the blood-vessels and nerves of the *supra-renal* capsules.

Generally, in those cases, the contraction is much more evident on the side of the cord corresponding to the side of the irritated nerve. Those demonstrations also coincide with other physiological evidence. More than half a century ago, Comhaire extirpated the kidney from living dogs, with the effect of instantly producing paralytic weakness of the hind leg of the side operated on. Brown-Séquard found the same result on extirpating either a kidney or a supra-renal capsule. It is, therefore, quite legitimate to conclude that irritations of these parts from disease may produce a paraplegia, by causing contractions of the blood-vessels of the cord similar to those which took place in these experiments.

There are other reasons which show that such paralysis is due to reflex action and probable contraction of blood-vessels—namely, the fact familiar to surgeons, that chills, tremors, and even convulsions, are often connected with catheterism. Another kind of proof is that of a negative kind, which shows that "the spinal cord may have its functions impaired, and even lost, and that suddenly, without any anatomical lesion." This is proven by the *post-mortem* examination of cases dying after symptoms of reflex paraplegia. Such cases are related by Stanley, Rayer, Cruveilhier, Chomel, Drs. Stokes and Graves, of Dublin, and Dr. Gull.

The present position of our knowledge of the structure and functions of the spinal cord hardly entitles us to assert, without careful microscopic examination, whether alteration of the cord does or does not exist in cases of *reflex paralysis.* Of all these cases, only in that of Dr. Gull was any microscopic examination made; and it is not impossible, but it is very probable, that a functional lesion, established even in a reflex way, may go on to organic mischief in the end.

Paralysis due to *myelitis* having been already noticed in connection with that disease, it may be useful to contrast here, in a tabular form, the leading phenomena of the two diseases, as given by Dr. Brown-Séquard in his admirable lectures *On Paralysis of the Lower Extremities;* and which, to some extent, are subscribed to by Dr. Edward Meryon.

PARAPLEGIA DUE TO

(A.) URINARY REFLEX IRRITATION.	(B.) MYELITIS.
1. *Preceded* by an affection of bladder, kidneys, or prostate.	1. Usually no disease of the urinary organs, except as a consequence of the paralysis.
2. Usually lower limbs alone paralyzed.	2. Usually other parts paralyzed besides the lower limbs.
3. No gradual extension of the paralysis upwards.	3. Most frequently a gradual extension of the paralysis upwards.
4. The paralysis is usually incomplete—an extreme debility or weakness of the limbs rather than paralysis.	4. Very frequently the paralysis is complete.
5. Some muscles more paralyzed than others.	5. The degree of paralysis is the same in the various muscles of the lower limbs.
6. Reflex power neither much increased nor completely lost.	6. Reflex power often lost, or sometimes much increased.
7. Bladder and rectum rarely paralyzed, or at least only slightly so; *sphincter ani* weak.	7. Bladder and rectum usually paralyzed completely, or nearly so.
8. Spasms in paralyzed muscles extremely rare.	8. Always spasms, or at least twitchings.
9. Very rarely pains in the spine, either spontaneously or on the application of pressure, percussion, or a hot moist sponge, or ice.	9. Always some degree of pain, existing spontaneously or caused by external excitations.
10. No feeling of pain or constriction round the abdomen or the chest.	10. Usually a feeling as if a cord were tied tightly round the body at the upper limit of the paralysis.
11. No formication, pricking, nor disagreeable sensations of cold or heat.	11. Always formications, or pricking, or both, and very often sensations of heat or cold.
12. Anæsthesia rare; the tactile sensibility being but slightly if at all impaired, but the muscular sense may be almost lost.	12. Anæsthesia very frequent, and always at least numbness.
13. Usually obstinate gastric derangement.	13. Gastric digestion good, unless the myelitis has extended high up in the cord.
14. Variations in the degree of the paralysis corresponding with changes in the primary disease.	14. Ameliorations very rare, and not following changes in the condition of the urinary organs.
15. Urine is usually acid, unless the urinary organs are diseased.	15. Urine almost always alkaline.
16. Cure of the paralysis frequently and rapidly obtained, or taking place spontaneously, after a notable amelioration or cure of the urinary affection.	16. Frequently a slow and gradual progress towards a fatal issue; very rarely a complete cure.
17. *Usually* muscles do not become atrophied, and temperature is little lowered.	17. Atrophy of the muscles of the paralyzed parts.

It is necessary, therefore, to look for some primary disease to account for *reflex paraplegia*, believing at the same time that such primary disease may ultimately establish a myelitis, while a diagnosis of *reflex paraplegia* is mainly based on a contrast of the phenomena which attend the various forms of paraplegia due to other causes.

In addition to the characters in the table, there are some symptoms peculiar and almost pathognomonic of forms of paralysis due to other local lesions—*e. g.*, meningitis of the spinal cord induces rigid spasms of the muscles of the back; intense pain on motion of lower limbs or of the spine; spontaneous acute pains that radiate from the spine to the lower extremities (similitude to rheumatism); frequency of cramps; and pressure on the cord by a tumor or diseased bone. They may and often do produce a myelitis. Pressure causes a feeling of tightness and pseudo-neuralgic pains. A tumor in the grey matter of the cord is attended with anæsthesia from the very first, which may reach a higher degree than the power of motion; and reflex power below the lesion becomes then extremely exalted. In hæmorrhage into the cord there is pain, then sudden paralysis, and often convulsions.

Treatment.—In cases of *reflex paralysis* means must be taken—1st. To diminish the external irritation which causes the paralysis; 2d. To improve the nutrition of the spinal cord; 3d. To prevent the ill effects of rest on the paralyzed nerves and muscles.

In cases of disease of the urethra or prostate, *an injection of a solution of one grain of the extract of belladonna, in twenty drops of laudanum, is to be made into the urethra, and the injection should be retained for half an hour, or even an hour, after which some emollient decoction should be used, such as lintseed tea, to wash out the passage.* Every two or three days this operation should be repeated. If the bladder be diseased, Dr. Brown-Séquard advises a solution of *one grain of the extract of belladonna,* in twenty drops of laudanum, to be used as an injection, after a complete emission of urine. Injections of carbolic acid are also of use in counteracting decomposition of urine and epithelium in the bladder. If the prostate is enlarged, a suppository ought to be put at times into the rectum. One of the best suppositories for this purpose is composed of the following ingredients, namely:—*White sugar, white soap, and gum-arabic in powder, of each three grains; opium, in powder, a grain and a half; or belladonna extract, one grain; or both combined. These being mixed together, the mass is to be formed into a conical shape, and being dipped in melted wax, is ready to be introduced when required into the rectum* (SIMPSON).

When the irritation causing reflex paraplegia starts from the vagina or uterus, a pill of half a grain of the extract of belladonna, with a grain of opium, surrounded by a piece of cotton wool, is to be introduced into the vagina, and made to surround the neck of the womb. By means of a thread it may be withdrawn so soon as the pain ceases or diminishes (TROUSSEAU, BROWN-SÉQUARD).

Belladonna ought not to be used constantly. *Opium,* combined with *strychnia,* is of greater use in reflex paralysis; and of all remedies, *strychnia* is best suited for promoting the second indication, which points to increasing the nutrition of the spinal cord. The dose combined with opium must be a small one—namely, *one-fortieth* to *one-thirtieth of a grain daily;* and when used alone, its dose may be *one-twentieth of a grain;* and when employed together with *belladonna,* its dose must be still larger.

In cases where no congestion nor inflammation of the spinal cord exists, *strychnia* ought to be persistently employed; but its use ought to be suspended at once whenever it produces spasms, or even numbness of the feet in getting out of bed in the morning (BROWN-SÉQUARD). *Sulphur baths* are also productive of great benefit.

The third indication—namely, to prevent the ill effects of rest on the paralyzed nerves and muscles—is best met by the employment of galvanism and shampooing of the paralyzed limbs. Two or three applications, of ten minutes each, in a week, are sufficient; but of all things it is necessary that the primary disease (causing by its persistent existence the reflex paralysis) should be cured or mitigated.

LOCOMOTOR ATAXY.

Latin Eq., *Ataxia motus;* French Eq., *Ataxie locomotrice;* German Eq., *Ataxie locomtrice;* Italian Eq., *Atassia locomotrice.*

Definition.—*A peculiar form of paralysis, characterized by unsteady and disorderly muscular movements, but with muscular power entire, and more or less progressive loss of the faculty of co-ordinating power (volunatry and instinctive). There is sometimes temporary diplopia, with unequal contraction of the pupils. The course of the disease is slowly progressive; and the anatomical lesion is generally a degeneration of the posterior columns and shorn of the spinal cord and posterior roots of the spinal nerves, sometimes with peripheral structure-change in the cranial nerves; and chiefly the second, third, and sixth pairs, in cases where the sight is affected.*

Pathology.—The disease is one of middle life; most common in the prime of life, between thirty-five and fifty years of age; it is very rare in youth or old age. It is also more frequent, in a very large proportion, in males than females. It has received various names, some suggestive of its progressive nature from bad to worse, such as *Progressive locomotor ataxy; Progressive motorial asynergia;** *Consumption of the spinal cord; Tabes dorsalis, grey degeneration of the posterior columns of the spinal cord* (Leyden), *cerebral paraplegia* of Dr. Gull.

Morbid Anatomy.—The essential anatomical lesions are found in the posterior columns of the spinal cord, the posterior roots of the spinal nerves, the peripheral extremities of the cranial nerves, and, exceptionally, in those of the extremities. The membranes of the cord are generally unaffected or merely congested—sometimes thickened posteriorly by exudations adherent to each other and to the posterior surface of the cord. The pia mater is constantly thickened, opaque, or cloudy, and more or less adherent to the posterior columns. The degeneration and atrophy of the posterior columns of the cord cause the cord to seem flat in its antero-posterior diameter. The posterior median fissure is effaced or marked only by a whitish line. The degeneration consists of an atrophy with disintegration of the nerve fibres (gelatiniform degeneration of Cruveilhier and Virchow), with proliferation of the connective tissue, giving to the columns a greyish-red transparent aspect. It always commences in the immediate vicinity of the posterior fissure, close under the *pia mater;* and thence it spreads towards the sides and the grey commissure. On section the diseased part always exhibits the shape of a wedge, with the base towards the *pia mater.* *Corpora amylacea* are found imbedded in the connective tissue. The blood-vessels which traverse the columns are loaded on their external coats, and surrounded to a variable depth with oil globules and granules of various sizes. The *dorso-lumbar* region of the cord is the most constant site of lesion, which may involve one or two inches, to nearly the whole extent; and may extend in depth to the surface of the lateral columns, and occasionally even along the edges of the anterior columns. Localized areas of some widely-spread degeneration have even affected the extremities of the posterior cornua and deeper parts of the grey substance (Lockhart Clarke, *Lancet,* June 10, 1865). The posterior nerve-roots, both within the cord and after leaving its substance, are similarly involved in the degeneration. They resemble thin vascular translucent connective-tissue cords rather than nerves—an appearance especially well marked in the posterior roots of the *cauda equina.* The general progress of the disease in the cord seems to indicate a central commencement advancing to the periphery; but in the cerebrum the phenomena indicate an opposite course—namely, affection of the peripheral distribution of the first, second, third, fourth, fifth, sixth, and seventh nerve, travelling to the centres. From the retina and optic nerve it has advanced as far as the *corpora geniculata,* and even to the *corpora quadrigemina.* The eighth pair seems the only cranial nerve that has not yet been implicated. The condition of the sympathetic is unknown.

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* From *α*, privative, and *συνεϱγος*, joint-work, co-operation (Bazire); and the Editor of the American edition of this book suggests *motorial* in place of *locomotor.*

In the later stages the degenerated parts of the medulla shrink away. The parts become hard and less transparent.

These anatomical lesions (so exactly limited to the posterior columns of the cord, and the roots issuing therefrom) by no means account for all the symptoms which are clinically recognized as distinctive of this disease. The nature of the peculiarly characteristic gait is sometimes referred for explanation to the loss of the "*muscular sense*" of Bell, or the loss of "sense of muscular activity" of Gerdy; or something equivalent. The inco-ordination is essentially a reflex disorder; and the lesions involve the co-ordinating centres (posterior column of spinal cord) of certain muscular acts, such as walking and standing. Some of the phenomena of the disease suggest involvement of the sympathetic—*e. g.*, the vesical phenomena, also the phenomena of irregular contractions of the pupils, and their becoming dilated during paroxysms of pain, or when the legs or arms were pricked or pinched, with local perspiration increased in some parts only of the skin, *e. g.*, forehead, palms of hands, and fingers* (Bazire).

The disease, then, is a peculiar one of the nervous system, commencing insidiously with evidence of disorder of some of the *cranial* nerves, perversion of sensibility in different parts of the body, ultimately giving place to inco-ordination of motion—always associated with degeneration of the posterior columns of the spinal cord and posterior nerve-roots, but without loss of muscular power or impairment of the intellect. The connecting links between the initial disorders of the cranial nerves (advancing centrically) and the lesions of the spinal cord (progressing peripherally) have not yet been made out.

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* Dilatation of the pupil is known to result from disease of the *third* cranial nerve or its roots; section or lesion of the sympathetic in the cervical portion; intravertebral section of the two upper dorsal nerves, *followed by irritation of the peripheral ends of the cut roots.*

In addition to these characteristic *post-mortem* lesions, there is evidence during life, both at the commencement and throughout the course of the disease, of localized congestions, expressed by rachalgia and spinal tenderness.

Symptoms.—The outset of the disease is insidious and slow. Pains, generally ascribed to rheumatism or neuralgia, but now known to be of spinal origin, first attract attention, particularly in damp weather. These pains may be fixed and aching, or darting, piercing, and transitory. Affections of the *second*, *third*, *fifth*, and *sixth* pairs of cranial nerves, sometimes with headache, giving rise to imperfect or double vision, strabismus, or contractions of the pupil, or dilatations, or deafness, are also insidious phenomena which may attract early notice. These several phenomena generally appear at different times and singly, although sometimes several together; and, after persisting for some time, may pass away. Weakness in some of the limbs may supervene, but no actual paralysis. Another distressing harbinger of the disease is incontinence of urine, associated with spermatorrhœa during the night, with a great proclivity to sexual congress, which is no mere impotent desire, but results in effective sexual intercourse. After a period, however, (of perhaps two years) the emissions on sexual intercourse become hasty, and a gradual loss of sexual desire and the power of erection supervenes. The bladder becomes irritable. Constipation is occasional when the pains are severe, and it always aggravates them. Transient tickling sensations prevail in different parts of the body, such as the lips, nose, cheeks, forehead, with occasional numbness of the feet and arms, and the peculiar feelings of so-called "pins and needles, as if they were asleep." After another long interval (say of two more years), undue fatigue after walking is experienced; the legs give way under the weight of the body, and there is a sensation as if the patient walked on a spring-board and could not keep his balance. These feelings may disappear and return; and eventually cutaneous hyperæsthesia or neuralgia of the skin (one side of the scrotum, testicle, buttock, or external part of thigh), always unilateral, supervenes.

Paralysis takes no part in the phenomena; but rather disturbed co-ordination of muscular movements, as originally pointed out by Duchenne.

Motorial inco-ordination, however, may not come on till after a period of several years (four or five) of suffering from such nervous symptoms. The pains then occur very irregularly, and last from a few hours to many days. At times they dart from limb to limb, or from one part of a limb to another; or, fixing on a small circumscribed spot, they give a boring, gnawing, or tearing sensation. The pains leave behind them a stiffness and soreness of the part. There may be atrophy of the optic disc, with contracted pupil; cutaneous anæsthesia of the soles of the feet, legs, forearms, and lips; and tactile sensibility seems slowly conducted to the sensory centre. There is decided diminution of cutaneous and muscular sensibility. "The floor is no longer distinctly felt by the feet; the foot seems to rest on wool, soft sand, or on a bladder filled with water. The rider no longer feels the resistance of the stirrup, and desires to shorten the stirrup-leathers. If the patient also does not *see* his movements, the power of co-ordination will be still more uncertain; if, while erect, he closes his eyes, he immediately begins to sway about and totter. If he closes his eyes in the horizontal position, he cannot tell the location of his limbs—he cannot tell whether the right foot or leg is crossed over the left, or the reverse" (ROMBERG). Reflex movements are not excited by tickling the soles of the feet. A painful feeling of constriction, as if by a tight band, is sometimes experienced round the body; and the sensation of "bearing down," in the perineum and rectum, with constipation, prevails. The bladder imperfectly empties itself, and cannot retain the urine after experiencing the desire to void it. The desire to pass it is frequent and imperative, especially during the night. Some degree of paralysis of the bladder is indicated by diminished force in the strain of urine—the jet is not well arched, and drops dribble away after the act is believed to be over. The urine sometimes passes involuntarily. Sexual desire is eventually abolished. The gait is characteristic. The steps are quick, short, and jerking. The leg and foot are well lifted from the ground, but they are thrown spasmodically and forcibly forward, the whole limb being extended. In bringing the foot down, the heel strikes the ground first. As the disease progresses, the limbs are thrown involuntarily to the right or left without purpose, and without the power of restraining in any way their irregular movements. In walking, the eyes are kept fixed on the legs; and a stick is used, over the handle of which a handkerchief may be placed to increase the surface of contact required by the numbness of the hand. The muscular force remains good, so that efforts to bend or extend a limb against the will can be resisted with strength. Involuntary jerkings of the limbs in bed prevent sleep. It is especially at starting that the patient has the greatest difficulty in maintaining equilibrium.

When muscular inco-ordination of the upper extremities supervenes, the fingers become numb, and objects are handled with increasing clumsiness. The clothes cannot be buttoned, nor small things removed from the pockets by the fingers. If the patient is set on his legs with his eyes closed and his feet close together, although he has muscular power to stand yet he cannot preserve his body from falling, or guide himself in taking a few steps in the dark or with his eyes shut. He has no idea of the position of his lower extremities except from sight.

The prolonged course of the disease is characterized by frequent natural pauses in its progress, sometimes lasting for a long time, before its onward progress is resumed—a point to be borne in mind in the treatment of the disease.

Paresis of the *oculo-motor* and *abducens* occasionally occur, as shown by *diplopia*, less frequently by *strabismus* and *ptosis* of the upper lid, and lastly by *amaurosis* and *psychical* disturbances. Those symptoms indicate that the disease is progressing to the cerebral centres. Atrophy of the optic nerve has been traced as far as the *corpora quadrigemina.*

Electro-muscular sensibility is generally lessened; and during the early stages of the disease there is neither paralysis of muscles nor wasting of their substance, nor defective *nutrition* of the limbs. The patient may be strong enough to bear and to carry considerable weight;

so that there is not muscular weakness, far less paralysis, in the ordinary sense of the term. The seeming paralysis is entirely due to the peculiar deficiency in the power of co-ordinating voluntary movements. The duration of the disease ranges from a few months to thirty years, with a mean duration of *seven* years in 119 cases whose progress has been observed (DR. CLYMER).

Diagnosis.—In the early stage of the disease the differential diagnosis is between intracranial lesion and the peripheric lesions of the cranial nerves, which characterize the commencement of progressive locomotor ataxy. Here the opthalmoscope becomes of use. In the amaurosis of intracranial disease there is always evidence of recent or past *neuritis* of the optic disc; but such is not the case in this disease (H. JACKSON). Care must also be taken not to confound the disease with progressive muscular atrophy. (See next page.)

Prognosis is decidedly unfavourable. The lesions may remain stationary for long periods, but recovery never takes place, and eventually the disease progresses onwards to death. Of forty-three fatal cases, the immediate cause of death in *six* were lesions of the brain or spinal marrow, with softening, hæmorrhage, and progressive muscular atrophy. Three died of acute inflammation of the urinary organs, and four from extensive sloughs over the sacrum. Thirty died during the course of intercurrent diseases, not directly connected with the motorial asynergia—namely, thirteen of pulmonary consumption, three from typhoid fever, four from pneumonia, others from pericarditis and dysentery.

Causes.—The only positive determining causes seem to be prolonged exposure to damp, cold, depression of the nervous system from insufficient diet; mental exhaustion, trouble, and anxiety; venereal excesses, especially onanism. If there be a hereditary history of various nervous diseases, *motorial asynergia* may be connected with those diseases, and ascribed to a common origin.

Treatment.—The intercurrent localized congestions in the region of the spinal cord point to topical blood-letting by cupping or leeches, cautiously employed, and perhaps frequently repeated; with persistent counter-irritation over the spine (by *blisters, moxæ, actual cautery*, or *ice*), particularly over those parts where there is tenderness or pain.

Phosphorus, in the form of *phosphates of metals* and *salts*, of *diluted phosphoric acid*, as a drink in the daily allowance of water, of *phosphate of soda* as an aperient, may be given with benefit.

Nitro-muriatic acid as a tonic, and *cod-liver oil* as a dietetic agent, may also be of use. Flannel should be worn next the skin, and chills from cold and damp carefully avoided by the use of a chamois leather close-fitting jacket over the flannel, and reaching from the height of the clavicles as low as the folds of the *nates* behind. The diet ought to be of the most nutritious materials which the patient can digest.

Cannabis Indica and *belladonna* give the greatest relief to the pains. *Nitrate of silver* is recommended by Wunderlich in doses gradually increasing to half a grain daily.

So long as there is active local hyperæmia, the use of electricity is hurtful. Faradization may be of use in restoring, to some extent, sensibility to the skin, where local cutaneous anæsthesia has prevailed, after which patients seem to walk better for a time; but when used at all, electricity ought only to be employed during the pauses in the course of the disease, and then with great caution, in the form of a constant current rather than the induced current, or Faradization. Niemeyer has had no success either with nitrate of silver or the constant current of electricity, but he recommends them to be tried.

Phosphuret of iron is recommended by Dr. Gueneau de Mussy, and *ergot* by Dr. Clymer. Dr. Althaus recommends *sulphur baths* as adjuvants, relieving pains and diminishing numbness.

PROGRESSIVE MUSCULAR ATROPHY.

LATIN EQ., *Atrophia musculorum ingravescens;* FRENCH EQ., *Atrophie musculaire progressive;* GERMAN EQ., *Progressive muskel-atrophie;* ITALIAN EQ., *Atrofia muscolare progressiva.*

Definition.—*A peculiar wasting of muscles, with atrophy of their substance, and lesion in the anterior roots of the nerves of the spinal cord, and paralysis.*

Pathology and Symptoms.—Idiopathic paraplegia is doubtful; yet it is certain that cases do occur, concerning which it is not easy to say, during the life of the patient, nor even after a *post-mortem* examination, what is the cause of the paralysis. For example, the cases of so-called "wasting palsy," the *paralysie musculaire atrophique* of Cruveilhier, are not yet made out to be a really specific disease independent of the state of the spinal cord. When spinal cords are examined in the way they have hitherto been examined, most of them are pronounced healthy. Of the numerous cases of "*wasting palsy*" collected together by Dr. Roberts, in thirteen only was the nervous system examined out of one hundred and five in all, and of these thirteen, four of them had disease of the cord. Obscure structural changes in the grey substance of the cord, or even only in the grey substance of the ganglia on the posterior roots of the nerves, may affect the nutrition of parts to which they are subservient, without interfering with the function of motion or sensation in the first instance.

A very interesting case of local atrophy of muscles, and local paralysis of the specially affected groups, is related in Beale's *Archives of Medicine* for October, 1861, which would no doubt have been set down as a case of "wasting palsy" *independent of disease of the cord,* if that organ had not been sent to Mr. Lockhart Clarke, and carefully examined by him. He found, and demonstrated, that lesions existed in it which occurred in small isolated spots, sufficient to account for the limitation of the disease to particular muscles.

If, therefore, we are ever to arrive at accurate and available results, both as regards physiology and pathology, our mode of examining the spinal cord must be more searching and exact them heretofore. There is no royal road to the removal of the spinal cord. Time, patience, and great caution are necessary to avoid injuring the soft parts, so that, when hardened perfectly, entire sections may be got for examination. under the microscope. If this plan were more systematically adopted, it would undoubtedly open up a new field of pathological and physiological study, furnishing results which no experiment in vivisection could possibly reach, if the history of the case was complete. Numerous cases of wasting of the muscles of one or more limbs, independent of any well-defined cause, have been from time to time observed, and are recorded in various publications; and we are indebted to Dr. William Roberts, of Manchester, for the first systematic treatise on the subject, in 1858. Such cases have hitherto been looked upon as *extraordinary* or *anomalous cases*, and were described as instances of "creeping palsy," "partial" or "local palsy," or "anomalous hemiplegia." In France it has been described by the name of "*atrophie musculaire progressive,*" or "*paralysie graduelle du movement par atrophie musculaire*" (DUCHENNE, ARAN, CRUVEILHIER), corresponding to the third form of progressive paralysis described by Meryon—namely, from "granular degeneration of the muscles, and where no disease or lesion is found in the nervous centres."

It seems to be still, however, an open question, whether progressive muscular atrophy is really a substantive disease of the muscular texture, or dependent on a structural change in the spinal cord. The College of Physicians have numbered it, and classed it among *diseases of the muscular system.* This is also the view adopted by Niemeyer, who believes that the long dispute as to the nature of this disease has been decided in favour of those who regard progressive muscular atrophy as a primary muscular affection; since almost all observers have agreed in regarding the continuance of excitability in the atrophied muscles, as long as they contain muscular elements, to be the pathognomonic sign of the disease. The nerves and muscles alike retain their excitability till the muscles perish.

But besides the examination of the spinal cord made by Dr. Roberts and Mr. Lockhart Clarke, there are those of Gull, Guy, Herard, Virchow and others, who demonstrate the nervous rather than the muscular seat of the disease. But some place the origin of the disease in the sympathetic system of nerves (SCHNEEVOOGH, JACCOUD, BÄRWINKEL, REMAK, TROUSSEAU); and Dr. Williams Roberts, finally, comes to the conclusion expressed in the following words:—

"The opinion seems to be steadily gaining ground that the nutrition of the muscles is placed under the control of a special set of organic nerves, having upward connections with the sympathetic ganglia and the cerebro-spinal axis, which are by no means identical with the central connections of the motor nerve fibres of the same muscles. Assuming the existence of such nutritive centres, all the clinical phenomena of wasting palsy, and the various findings of the *post-mortem* examinations admit of easy explanation, on the supposition that these centres, or some of their ganglionic connections are the primary seat of the disease, and the numerous associations and complications can scarcely be accounted for on any other hypothesis."

In 1851 Dr. E. Meryon gave an account of a gentleman's family in which three boys were the subjects of general muscular degeneration. His observations were original, and are published in the *thirty-fifth* volumn of the *Medico-Chirurgical Transactions*, p. 73. Several cases have been described since Dr. Roberts called attention to the subject; and the more closely and carefully the spinal cord has been examined after death by competent observers, the more surely has it been found diseased. In all the cases observed the muscles affected are those under the control of the Will; hence the course of the disease is easily followed, either by the changes produced in the external form of the parts, the absorption of masses of muscles, the displacement of bones, the abnormal position of joints from loss of their muscular supports, or by the failure of certain movements which contribute to outward expression or inward function, such as facial physiognomy, deglutition, vocalization, or respiration—all of which depend on the operation of stripped muscles under the influence of the Will. When the disease affects the muscles of the limbs the *disappearance* of the *muscles* causes very notable changes of conformation. The rounded contour gives place to a lean and withered aspect, the bony levers stand out in unaccustomed distinctness, so that the limb has the appearance of a skeleton clothed in skin (ROBERTS). As a general rule, when one limb is attacked, its fellow on the opposite side shares its fate; when the disease is unilateral, the right side is more likely to be its seat than the left; but the disease seems to be extremely capricious and uncertain in its line of attack, scarcely two examples being exactly alike in the combination of muscles implicated, or the relative degree in which they suffer. But when the malady affects the shoulder it scarcely ever fails to include the upper arm; so also the fore-arm and hand are generally associated in the disease. In the upper limbs the morbid action seems to radiate from two centres,—one in the hand, from which the fore-arm is invaded; and the other in the shoulder, from which are reached the muscles of the upper arm and those which brace the shoulder-blade to the ribs. When the hand and arm are destroyed, the evil does not then pass up the arm, but starts away to the shoulder, or to the opposite hand. In the same way, when the shoulder is first attacked, the disease does not descend along the upper and fore-arm to the hand, but, passing over the elbow, it begins afresh in the ball of the thumb, and from that focus spreads up the limb; so that the parts latest reached are those about the elbow, especially the masses that take their rise from the humeral condyles.

The invasion of wasting palsy is usually slow and insidious. It creeps on unawares; and the victim of its attack only becomes cognizant of the disease when he notices some marked failure in certain muscular powers. The tailor notices that he cannot hold his needle; the shoemaker wonders that he cannot thrust his awl; the mason's hammer has grown too heavy for his strength; the gentleman feels an awkwardness in handling his pen, in pulling out his pocket-handkerchief, or in putting on his hat (ROBERTS). On comparing the weakened member with its fellow, it is seen to be wasted, and the failure of power increases; the lifting power is reduced to nothing; the grasp is gone; and at last palsy becomes complete. In the majority of cases the disease commences in the upper extremities; and if the disease commences in the legs, it is probable that the atrophy will spread to the trunk. In more than *one-third* of the cases noticed by Dr. Roberts the hand was the member originally seized; and the exact spot nearly always the ball of the thumb; and the right hand more often than the left. Next to the hands, one or other shoulder is the favourite starting point. *Loss of power* is a chief phenomenon, and it corresponds to the grade of the atrophy of the muscle. It is only in extreme cases that any part is reduced to absolute immobility. *Muscular vibrations,* consisting of little convulsive twitchings or quiverings of individual muscular bundles, are also early phenomena. They do not impart any movement to the entire muscle, but parts of the muscle seem to spring beneath the skin in quick momentary tremors, undulating over the surface of the muscle. Tactile sensation generally retains its delicacy in the skin over the affected muscles; but in some cases anæsthesia has been noticed over the skin, with exaggerated facility of reflex movement, chiefly in the muscles subject to the quiverings already described. Electric contractility exists, but is less in the affected muscles, and the amount of diminution is in direct proportion to the degree of their atrophy (BENEDIKT, DUCHENNE, REMAK).

Pain is by far the most common of the nervous symptoms. It is present in about half the cases recorded. It is generally transient, and is usually marked at the commencement of the disease. In some cases the pain is sharp and continuous in the joints and along the muscles from the outset, continuing for several months, with pain in the dorsal spine, and in the extremities, and in the bones (REMAK, MEYER, ROBERTS). The advance of the malady has, in one case at least, been attended by an almost childish degree of helplessness, and a most pitiable state of mental irritability and hypochondriac depression (W. T. GAIRDNER, ADAMSON, BELL, GULL, DAY.)

Unusual sensitiveness to low temperature is occasionally a prominent and very annoying symptom; the temperature of the affected parts is always lowered.

As a rule, the general health does not seem impaired. Intelligence is clear, Judgment firm, and the Emotions under control till towards the end, and all the organic functions appear to be performed with regularity.

Morbid Anatomy.—The lesions are to be sought in the nervo-muscular system; and when death occurs in this and other forms of progressive paralysis, it is usually by some disorder of the respiratory apparatus, inducing *bronchitis, broncho-pneumonia, laryngitis,* or simply *apnœa,* from paralysis of respiratory muscles. Lesion of the muscles is constant, and nearly identical in all the cases. They are wasted away, some slightly, some profoundly, even to annihilation, their place being marked merely by origins and insertions, composed of more or less condensed connective tissue. Where muscular tissue is visible, it has lost the red hue of health, appearing of a pale red colour, like the flesh of a frog or fish. The degree of change into fat is various; in some no fat can be found, in others it is abundant, probably as an after-degeneration. Under the microscope the stripped elementary muscular fibres are seen to be completely destroyed, the sarcous element being diffused, and in many places converted into oil-globules and granular matter, whilst the sarcolemma, or tunic of the elementary fibre, was broken down and destroyed (Meryon, *Med.-Chir. Trans.,* vol. xxxv. p. 76). The striæ may be distinct, and between them grey or brilliantly shining molecules are to be seen; and where no striæ exist, long cylinders may be seen in place of the primitive fibres (GALLIET).

With regard to the nervous system, the *central organs*, the *spinal roots of the nerves*, and the *peripheral distribution*, have each engaged attention. In thirteen only of the 105 cases recorded by Roberts was the spinal cord examined; and in *four* instances it was found diseased. It is unnecessary to repeat again here how imperfect our information is on this point. Of the lesions recorded, a summary may be stated as follows:—(1.) While the spinal centre was considered sound, the anterior roots and peripheral distribution of the muscular nerves were extensively diseased. (2.) Inflammatory softening of the cord accompanied fatty degeneration and destruction of the anterior roots; and in one instance the peripheral muscular branches were destroyed. (3.) Amyloid degeneration of the cord, confined to the posterior median columns, has been observed in one instance, associated with granular degeneration of peripheral muscalar branches, but with preservation of the anterior roots. (4.) In the cases examined by Mr. Lockhart Clarke, in the regions of the spinal cord which supplied the wasted muscles, numerous patches of transparent granular degeneration were found, especially in the grey substance, and generally around or in the vicinity of blood-vessels. The nerve-cells were shrunken and atrophic, and they contained an unusual number of coarse pigment-granules, while *corpora amylacea* were abundant around the central canal of the cord.

Causes.—With regard to the cause of this form of palsy, it is difficult to say whether it begins primarily in the muscles, or in the nerves associated with the muscles, and secondarily affects the spinal marrow connected therewith, in the manner referred to by Türck and Graves, and to which the term "creeping palsy" is applied; or whether (as Lockhart Clarke's observations tend to show) the lesion of the spinal cord is the primary lesion. In all cases the question to determine is whether or not the atrophy of the muscle is subsequent to the paralysis. If so, there are good reasons for believing that the wasting of the muscular tissue is an immediate or direct effect of the lesions in the cord; and the paralysis is generally too rapid to be accounted for by mere inactivity of the paralyzed muscles. Dr. Meryon believes that only in the last form the disease commences in the muscles. Dr. Roberts, on the other hand, contends that the muscles are the parts essentially diseased, in the first instance, in the cases of "wasting palsy" which he has described. A blight (?) seems to wither the muscles, of the nature of a degeneration. He believes the disease to be of constitutional origin; and the evidence of this rests upon the facts that it is transmissible from parent to offspring, and that in its march it exhibits a bilateral symmetry.

The disease has been observed to follow cases of fever and sunstroke, severe falls, and blows on the back of the head or spine; and it is well to observe in all cases whether or not the brain has received any accidental shock; and it is probable that several distinct diseases have been described under the name of "wasting palsy" (W. T. GAIRDNER).

Prognosis.—The *progressive forms of paralysis* are the most intractable in the domain of physic, and the gravity of the prognosis depends upon the disease confining itself to the extremities and the muscles thereto relating, or extending to the trunk and face. The signs of extension to the face are a diminished mobility of the lips, a slur in the articulation of words, frequent sighing, and fibrillary muscular tremors on different parts of the chest, abdomen, or face. If the disease has been hereditary, there is every fear that it will be fatal; and so also if the lower limbs are first attacked.

The disease runs its course with great slowness. Some cases complete their history in six or eight months, others linger on through many years. Recovery, permanent arrest of the palsy, or death, are the various terminations of the disease. The mean duration of the cases ending in recovery has been *one year and two months;* of those ending in permanent arrest of the palsy, the mean duration of the cases has been *two years and three months;* of those terminating fatally, the mean duration was *five years and two months.* The longest duration of a case ending in recovery has been *two and a half years,* and the shortest period *eight months.* The longest case ending in arrest continued active for *seven years,* and the shortest for *four months.* Fatal cases have not been known to terminate under *twelve months,* while one lingered for *twenty-three years,* another for *eight years,* and some beyond *four years* (ROBERTS).

In those cases which terminate by permanent arrest the wasting of the muscle ceases, and the limb continues for an indefinite period in its maimed condition, neither amending nor deteriorating. The muscles atrophied are not regenerated, but remain *in statu quo;* and those which are not completely atrophied continue to exercise their feeble powers under the influence of the Will, but they never regain their former bulk or vigour. After lying torpid for years, the malady may awaken to new and more violent activity (ARAN, VIRCHOW); and temporary lulls in the progress of the atrophy are common enough, but they seldom last long (ROBERTS). The disease is invariably fatal when it invades the trunk.

Treatment of *wasting palsy* ought to be strictly a restorative one (see p. 289, vol. i.), believing that the disease is one of nutritional deterioration of the nervous centres. Preparations of *wine* and *of cod liver oil* with *localized Faradization,* are the most likely agents to improve the condition of the body (ALTHAUS). Local means will aid the hygienic —namely, *methodical exercise* and *douche baths,* or *cold mineral baths;* so also *thermal* or *sulphur baths,* and *galvanism. Frictions,* with stimulating liniments (such as camphor liniment) are also favourably spoken of (DUCHENNE, GROSS, MEYER, ROBERTS). "Faradization" consists in the employment of the electricity of the induced or secondary current in the helix round the magnet, originally discovered by Faraday. It ought to be practised at least three times a week, *for from five to ten minutes each time,* and continued at least a month before it is given up, if negative results are only obtained. Every muscle ought to be Faradized in a special manner, according as it has suffered more or less in its electric contractility and nutrition. The power of the current ought never to be strong. When the sensibility of the muscle returns, the intensity of the current may be diminished. Its application should never be protracted beyond ten or fifteen minutes at the most, one minute, on an average, being allowed to each muscle, or distributed over several at a time.

Volta-electric machines are made for medical purposes by Messrs. Stohrrer, of Dresden. Magneto-electric machines are to be got from most of our philosophical instrument makers. The apparatus for applying a continuous current, as modified by Dr. Althaus, is to be obtained of Messrs. Legendre & Morin, Paris, and Weiss of London.

If, on the other hand, it turns out, as the searching examinations of Mr. Lockhart Clarke would tend to show, that these forms of progressive paralysis or "wasting palsy" are always associated with disease of the spinal cord, the efforts of treatment should, in the first instance, be directed to that part of the cord corresponding to the connections of the nerves with the site of lesions—the line of treatment to be pursued being determined by the general symptoms. The application of the *constant* current of electricity—the *positive electrode* being placed in the region of the cervical sympathetic, and the *negative electrode* upon the cervical and upper dorsal regions of the spine, have been followed by long amelioration, if not by a positive cure in the hands of Remak and Benedikt (Clymer, in *New York Medical Journal*, 1866, vol. iii.)

INFANTILE PARALYSIS.

Latin Eq., *Paralysis infantalis;* French Eq., *Paralysie de l'enfance;* German Eq., *Paralysis infantilis;* Italian Eq., *Paralysi infantile.*

Definition.—*An essential palsy which is the more or less permanent result of an acute disease of the brain, spinal marrow, or peripheral nerves.*

Pathology.—The original lesion may have been an inflammation, or an effusion as the result of inflammation, which terminates in a local paralysis. It is believed to arise from disease of the cord, or from spinal congestion. It occurs almost exclusively among children during the period of dentition, and during the eruption of the permanent teeth, especially of the molars, and for a short time afterwards—from the sixth month to the third year of life. It has also seemed to result from a fall (Wilks), also, as a sequence to various exanthemata and exposure to cold.

Symptoms.—There may be signs of cerebral irritation, with more or less fever; but, as a rule, the paralysis comes on suddenly, or is noticed with surprise, having been preceded by no feature to attract notice. Sometimes also there may be convulsions, and when consciousness returns, a foot, a hand, a leg, or an arm, or the lower half of the body (paraplegia) is observed. Hemiplegia, involving the extremities of both sides, never occurs (Niemeyer). Hence the disease seems to be independent of *cerebral apoplexy* or *encephalitis.* The bladder and rectum also are never paralyzed.

There may be loss of power, but there is no diminution of sensibility at first, beyond slight numbness.

The duration of the disease varies extremely; sometimes the paralysis will disappear in a day or two, and cure will be complete; but in most cases the paralysis is stationary and permanent. In the temporary form the contractility of the muscles remains; but in the permanent form a few only of the muscles of the paralyzed limb retain electric contractility. In other muscles it is entirely gone (DUCHENNE). Hence such an experiment becomes a ground of prognosis.

After the disease has existed for some time, the limbs become soft, relaxed, and flexible, so that they may be placed in any position, and they eventually become atrophied or withered away; the skin becomes thin, fat is absorbed, the muscles waste away, and even the bones diminish. In the course of a year, at the growing age of childhood, the withered limb has lost in circumference and even in length compared with the sound one. Circulation, too, is diminished in its amount and activity; it assumes a livid hue, and hideous chilblains and ulcerations are easily formed. There is a marked depression of temperature in the paralyzed limb, and nutritive changes are greatly impaired. Deformity, by shortening of the limb, compared with the growing one, is soon apparent; and it may also become contracted.

The general health may remain unimpaired, and many sufferers in childhood attain even a great age; so that all that can be said in the way of prognosis is—" the disease does not endanger life." It often leads to incurable deformity. No hope of cure can be reasonably held out. Recovery occurs only in the temporary cases already referred to. A paralytic and withered limb for life is all that can be hoped for.

Diagnosis.—The disease has been mistaken for disease of joints, especially hip-joint disease, and for the stiffness of rheumatism, and even in some cases the children have been believed to be shamming paralysis, either from the power of imitation peculiar to the age, or from other circumstances.

Treatment.—If electric contractity remain, much may be hoped for; and so long as nerves and muscles have not altogether degenerated. The principles of treatment are the same as have been advocated in analogous diseases,—namely, restorative and not depressent. The general health must be improved. The systematic, active, and persistent application of the induced current of electricity is the best means of preserving the functions of the implicated muscles, and of arresting their atrophy and degeneration, provided all other hygienic arrangements are maintained which have already been insisted on as to food, clothing, and ventilation.

Mild purgation may be required; alteratives, especially small doses of *mercury*, continued for a long time, have been of service; and so also has *quinine, iodide of potassium,* and *iodide of iron.*

The muscles of the paralyzed parts should be put into action as much as possible, either by passive movement or by the action of galvanic currents. Friction and shampooing ought also to be persevered in. Three or four small circular wooden balls, about the size of a walnut, shut up in a box, with holes in its cover, so as to expose one-third part of the surface of the balls on a uniform level, makes the best shampooing apparatus I have ever seen. It was shown me by my colleague, Dr. Maclean, as the device of a patient who had it constructed for this purpose.

The limb must be kept warm by proper coverings of flannel or chamois leather. *Syrup of the phosphate of iron, quinine* and *strychnia*, as well as all preparations mentioned under the subject of anæmia are of use.

LOCAL PARALYSIS.

LATIN EQ., *Paralysis ex parte;* FRENCH EQ., *Paralysie locale;* GERMAN EQ., *Oertliche Lähmung;* ITALIAN EQ., *Paralisi locale.*

Definition.—*Paralysis limited to particular sets of muscles.*

Pathology.—Of this form there are several varieties, three of which require special consideration, namely (*a.*) *Facial paralysis;* (*b.*) *Scrivener's palsy;* (*c.*) *Glosso-laryngeal or pharangeal paralysis.* The two former only are named by the College of Physicians. Of these in their order; and first, of—

(*a.*) FACIAL PARALYSIS.

LATIN EQ., *Paralysis faciei;* FRENCH EQ., *Paralysie de la face;* GERMAN EQ., *Lähmung des Facialis;* ITALIAN EQ., *Paralisi faciale.*

Definition.—*Paralysis of the muscles of the face, usually confined to one side, but sometimes, though rarely, affecting both sides, and apt to be followed by contraction or tonic spasm of the muscles previously paralyzed* ("*Spasmodic Tic*" *of Marshall Hall*).

Pathology.—In previous editions of this text-book erroneous doctrines were stated regarding facial paralysis, in accepting and perpetuating the views of Dr. Todd, that *the fifth nerve is more or less involved in the paralyzing lesion*," and in mixing up the phenomena of *facial paralysis*, depending on *central* or *cerebral lesion*, with *facial paralysis* due simply to lesion of the trunk or branches of the *portio dura.* I am indebted to my friend Dr. Sanders, of Edinburgh, for kindly directing my attention to this error, and for references to his important paper in the *Lancet* of October 21st, 1865. From his papers on this subject, and the clinical lectures of the late Professor Trousseau on *facial paralysis*, the following account is given of this affection :—

Facial paralysis is due to one of two causes, namely—

(1.) To a lesion of the trunk simply, or of branches of the *portio dura* nerve at some part of its course, generally through the petrous bone, and independent of cerebral disease. This form of facial paralysis is sometimes also known as *Bell's paralysis*, or *peripheral facial hemiplegia*, or *mimic facial palsy.*

(2.) *Facial paralysis* may be due to a cerebral lesion—a cause which acts upon the nerve before it enters the internal *auditory meatus*—in which case it is usually accompanied by hemiplegia of the limbs on the same side. This form of facial paralysis is known as *cerebral* or *centric facial hemiplegia.*

(3.) A third form of *facial paralysis* is ascribed to reflex paralysis of the *seventh nerve,* consequent on paralysis or lesion of the *fifth pair,* generally an agency involving the peripheral ramifications over the face.

The first affection is that most frequently met with in practice; and although it is not a dangerous form of paralysis, it is one from which recovery is very slow, and in which prognosis, as to complete recovery of symmetry of the face, is uncertain. The nerve may be destroyed in the Fallopian canal by caries or other lesion of the petrous bone, or by fracture, or gun-shot wound.

The second form is the more rare, and is always a very grave disease, and not usually recovered from. In this form the nerve is most frequently pressed upon or atrophied by cerebral tumors; more rarely from exudations or thickenings of the *dura mater* or from *exostosis.* Both kinds of facial paralysis present many points of resemblance and of contrast, of great interest and importance in diagnosis; and the occurrence of paralysis from one of the causes mentioned by no means excludes the other, and therefore cases of both may exhibit extremely complex symptoms.

The phenomena of facial paralysis are confined to the "muscles of expression," including the *buccinator,* and do not involve the masticatory muscles (*masseter, temporal, pterygoid*) supplied by the fifth pair of nerves.

A description of the phenomena of *facial paralysis* must therefore have due regard to the course and distribution of the *seventh nerve.* The facial nerve, or *portio dura,* of the *seventh pair* is the motor nerve of the face. It emerges from the lateral column of the spinal cord, as that column passes under the *pons Varolli,* and enters the *internal auditory meatus.* At the bottom of this *meatus* it enters the *aqueduct of Fallopius,* and follows the windings of that canal to the lower surface of the skull —namely, to the *stylo-mastoid foramen*—by which it leaves the osseous canal of the temporal bone. It is then continued forwards through the substance of the parotid gland, and separates in the gland, behind the ramus of the lower maxilla, into two primary divisions—the *tempero-facial* and the *cervico-facial*—from which numerous branches spread out over the side of the head, the face, and upper part of the neck, forming what is known as the "*pes anserinus.*" Within the temporal bone it is connected with the auditory nerve by filaments of union, and where it swells into its gangliform enlargement it is joined by the large superficial petrosal branch from the Vedian nerve, and also by the small superficial petrosal nerve. Close to the *stylo-mastoid* foramen it gives off several small branches—namely, the posterior auricular, a branch from the *digastric muscle* and *stylo-hyoid,* and a twig to the *stylo-glossus.* In front of the mastoid process it divides into an *auricular* and an *occipital* portion, and is connected with the *great auricular nerve* of the cervical plexus. It also gives off branches to the *digastric* and *stylo-hyoid* muscles, joining the *glosso-pharyngeal* near the base of the skull, and the plexus of the *sympathetic nerve* on the external carotid artery. The main trunk of the facial then separates into two primary divisions, the larger of the two being the *tempero-facial,* which is directed forwards through the parotid gland. Its ramifications and connections with other nerves form a network over the side of the face, extending as high as the temple and as low as the mouth. Its branches are arranged in *temporal,* *malar*, and *infra-orbital* sets.* The smaller of the two primary divisions —the *cervico-facial*—is directed obliquely through the parotid gland towards the angle of the lower jaw, and gives branches to the face, below those of the *tempero-facial division*, and to the upper part of the neck. Its branches are named the *buccal*, *supra-maxillary*, and *infra-maxillary*. In the gland this division of the facial is joined by filaments of the *great auricular nerve* of the cervical plexus, and offsets from it penetrate the substance of the gland.†

As the course, distribution, and connection of this nerve are most important to be remembered in acquiring a clear understanding of the phenomena of its paralysis, whether due to cerebral or peripheral causes, they are thus minutely given from the best text-book of anatomy (Quain's, the edition edited by Drs. Sharpey, Thomson, and Clelland).

One very important point to remember is that the *portio dura* of the seventh pair is the only motor nerve of the *buccinator muscle* for all its actions, whether of expression or of mastication; and that the fifth pair supplies it, not with motor, but with sensory fibres (MAYO, VOLKMANN, LONGET). *The buccinator muscle is interrupted in all its functions, whether of expression or of mastication, whenever the portio dura is paralyzed; it is*

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* (*a.*) "The *temporal branches* ascend over the zygoma to the side of the head. Some end in the anterior muscle of the auricle and the integument of the temple, and communicate with the temporal branch of the upper maxillary nerve near the ear, as well as with (according to Meckel) the auriculo-temporal branch of the lower maxillary nerve. Other branches enter the *occipito frontalis*, the *orbicularis palpebrarum*, and the *corrugator supercilii muscles*, and join offsets from the *supra-orbital* branch of the opthalmic nerve."

(*b.*) The *malar branches* cross the malar bone to reach the outer side of the orbit, and supply the orbicular muscle. Some filaments are distributed to both the upper and lower eyelids: those in the upper eyelid join filaments from the lachrymal and supra-orbital nerves; and those in the lower lid are connected with filaments from the upper maxillary nerve. Filaments from this part of the facial nerve communicate with the malar branch of the upper maxillary nerve.

(*c.*) The *infra-orbital branches*, of larger size than the other branches, are almost horizontal in direction, and are distributed between the orbit and mouth. They supply the *buccinator* and *orbicular oris* muscles, *the elevators of the upper lip and angle of the mouth*, and likewise *the integument*. Numerous communications take place with *the fifth nerve*. Beneath the *elevator of the upper lip* these nerves are united in a plexus with the branches of the upper maxillary nerve; on the side of the nerve they communicate with the nasal, and at the inner angle of the orbit with the *infra-trochlear nerve*. The lower branches of this set are connected with those of the *cervico-facial* division. Near its commencement the *tempero-facial* division of the facial is connected with the *auriculo temporal nerve* of the fifth, by one or two branches of considerable size which turn round the external carotid artery; and it gives some filaments to the *tragus* of the outer ear (Quain's *Anatomy*, edited by Drs. Sharpey, Thomson, and Clelland, p. 613).

† (*a.*) The *buccal branches* are directed across the masseter muscle to the angle of the mouth; supplying the muscles (buccinators), they communicate with the *tempero-facial division*, and on the buccinator muscle join with the filaments of the *buccal branch* of the *lower maxillary nerve*.

(*b.*) The *supra-maxillary branch*, sometimes double, gives an offset over the side of the maxilla to the angle of the mouth, and is then directed inwards, beneath the depression of the angle of the mouth, to the muscles and integument between the lip and chin: it joins with the labial branch of the lower dental nerve.

(*c.*) The *infra-maxillary branches* perforate the deep cervical fascia, and placed beneath the platysma muscle, form arches across the side of the neck as low as the hyoid bone. Some branches join the superficial cervical nerve beneath the platysma, others enter that muscle, and a few perforate it, to end in the integument (Quain's *Anatomy*, by Drs. Sharpey, Thomson, and Clelland, p. 615).

unaffected, and all its actions are preserved, in motor paralysis of the FIFTH PAIR (Sanders, *Lancet*, October 28, 1865, p. 478).

The threefold functions of the *portio dura* must also be recognized. Considered as a musculo-motor nerve, it contains within its common trunk the following sets of fibres serving different functions:—

(1.) *Voluntary motor fibres*, by which the voluntary movements of the features are performed, and by which especially labial and buccal speech and mastication are accomplished.

(2.) *Emotional fibres*, by which the features express the passions more or less involuntarily.

(3.) *Reflex motor fibres*, which are involuntary, for the act of winking and for the movements of the nostrils in the acts of respiration.

These different sets of fibres are believed to derive their peculiar functions solely from the nature of their origin and place of central connection in the brain or *medulla oblongata.* In lesion, therefore, of the nerve trunk, in which all the fibres indiscriminately are equally liable to be affected, not only voluntary but emotional and also reflex motions will be suspended. But when the cause of the paralysis is cerebral, the origin or course of certain sets of fibres may alone be involved by the lesion, while others may entirely escape injury. The symptoms, therefore, of central paralysis will vary with the special seat of the central lesion. The voluntary and emotional actions (either or both), which have their origin in the cerebrum, will usually suffer; while the fibres for reflex action, which have their source in the *medulla oblongata*, may be expected to retain their power. The play of the features will be lost, and buccal and labial speech and mastication (so far as the buccinator muscle is concerned) impaired; but the natural position of semi-closure, and involuntary winking of the eyelids, will be preserved.

Looking, therefore, at the circuitous windings of the *portio dura*, its intricate distribution, and the various textures through which it passes, it can readily be understood how varied are its tendencies to be involved in disease, and even traumatic injury. Tumors, hæmorrhages, or other lesions, may involve the nerve within the cranium. While it traverses the circuitous windings of the *aqueduct of Fallopius* it may be injured and pressed upon by the results of necrosis or caries of the bone, or of suppuration or lesions of the fibrous sheath or periosteum; while lesions or tumors, involving the parotid gland, may injure the nerve and paralyze the parts it supplies.

Three forms of paralysis of the facial nerves ought to be discriminated in considering the diagnosis of central facial hemiplegia, namely,—*Voluntary Motor Paralysis*, *Emotional Paralysis*, and *Reflex Paralysis* (Sanders, *Lancet*, October 28, 1865, p. 479).

Symptoms.—The accession of facial paralysis (when peripheral) is usually sudden, and is generally discovered by the patient when he begins to eat. He feels something peculiar in the act of chewing, and has some difficulty in mastication. When the food gets between the paralyzed cheek and the teeth, the cheek is instinctively squeezed or pressed upon by the hand, in order to push the food between the teeth again. The difficulty of mastication only concerns the buccinator muscle, and not the other muscles of mastication. There is no pain. The patient is soon, and often abruptly, told by the first kind friend who happens to meet him, that his mouth is awry, and that it becomes considerably more so when he laughs. He then naturally wishes to see all this for himself; and on looking at his face in the looking-glass, he may verify the observation of his friend, and is then generally greatly frightened and alarmed by the discovery.

When the face remains at rest the paralyzed side looks slightly flatter, and more flaccid and pendulous, than the sound side. The eye of the paralyzed side is also more widely open than the eye on the sound side. When speaking, and still more when laughing, is attempted, the angle of the mouth on the paralyzed side remains perfectly motionless, but on the sound side it is immediately drawn upwards and outwards. The eyelids, the cheek, and half the lip of the paralyzed side remaining thus motionless when efforts are made to contract the muscles, a singular and characteristic expression is given to the face. The eyelids remaining motionless on the paralyzed side, the patient is unable to shut the eye, but the globe of the eye itself moves perfectly in any direction at will, which shows that the motor muscles of the eye are not affected, and that the paralysis affects exclusively the *orbicularis palpebrarum* muscle, and does not involve the *levator palpebræ superioris.* Sight is unimpaired. The tongue is protruded with ease and regularity; lingual articulation is sufficient; but articulation of certain words is difficult, on account of the paralysis of the cheek. Sometimes the arch formed by the pillars of the fauces is larger on the paralyzed side than on the sound one, by the uvula inclining to the sound side. Cutaneous sensibility is unimpaired; and the patient may be in the best of health, the ailment being purely local—namely, paralysis of the facial muscles of expression on one side. There is an absence of all electric excitability of the paralyzed muscles supplied by the seventh pair.

Causes.—One of the most common causes of *facial paralysis* is exposure to cold (*coup de vent*), especially to cold when sleeping in a draught or in the open air, or exposure to cold after being in a state of perspiration, or sitting in a railway or other carriage with the side of the face exposed to the wind. "Halla ascribes the increasing frequency of facial paralysis to the railways. People hurry to the station, arrive there warm, enter the carriage, and expose their face to the draught of the window. The result is palsy of the face" (NIEMEYER).

The influence of *rheumatism* is also conspicuous in many cases, the patient, being seized as suddenly as in *lumbago,* acknowledging a similar cause; residence in a damp place, or other exposure to cold and wet, being sufficient to bring on the attack.

The influence of mental emotion—a sudden fright or start—has been known to induce the paralysis.

Of traumatic lesion the facial paralysis of new-born infants is not uncommon. It is generally due to the compression of the trunk of the facial nerves by the use of the forceps. If such compression has been severe or excessive, the injury may be permanent; otherwise, it is soon recovered from.

Wounds of the *portio dura,* or fractures of the skull, involving lesion to the *aqueduct of Fallopius,* may also be followed by paralysis, and in all these instances the paralysis is sudden. When the facial nerve, during some part of its course, is interrupted in its functions by the secondary influence of advancing organic disease, the nerve becomes compressed gradually, and ultimately altered. In such cases the paralysis comes on slowly. In severe chronic inflammation of the internal ear, with destruction of the tympanum and ossicula, the petrous portion of the temporal bone becoming carious, facial paralysis is apt to supervene, and the lesions may be fatal.

The form of paralysis due to such causes is that known as *peripheral facial hemiplegia;* but the paralysis may acknowledge a *cerebral lesion* as its cause, such as cerebral hæmorrhages, lesions of the *pons Varolii,* implicating the fourth ventricle. In such cases the facial palsy is usually partial, affecting more especially the muscles of the mouth and cheek, leaving the closing movements of the eyelids unaffected. As a rule, also, the paralysis due to a cerebral lesion gives rise to a hemiplegia involving parts beyond the face, such as one or more limbs of the affected side. But there are also instances in which the paralysis due to a cerebral lesion (small cerebral hæmorrhages) has been exclusively limited to the face (GRAVES, DUPLAY). This is in keeping with other forms of hemiplegia (cerebral) in which the paralysis is localized; as when cerebral hæmorrhages or apoplectic-like seizures paralyze the tongue only, or an arm, or distort the features more or less, but generally combined with a hesitation in the movements of the leg, and of which the patient is unconscious (TROUSSEAU). The previous existence of peripheral paralysis in such cases may render superadded paralysis from a cerebral lesion very difficult to diagnose. When the cerebral lesion is limited to the *pons Varolii,* to slight injury to the fourth ventricle, the paralysis of the face which results has many of the characters of the peripheral or Bell's paralysis, even to the absence of electric insensibility of the paralyzed muscles (VULPIAN, quoted by TROUSSEAU, who in the course of a very long practice never saw a case of the kind himself).

The most distinguishing characteristic of paralysis due to a *cerebral lesion* is considered by Trousseau to be the *absence* of complete paralysis of the *orbicularis palpebrarum.* "However complete hemiplegia of cerebral origin may be, I have never seen *complete paralysis of the orbicularis palpebrarum—the eye can always be closed;*" while in Bell's paralysis, the palsy of the *orbicularis palpebrarum* is never absent, and the eye cannot be completely closed (TROUSSEAU). This coincides with the experience of Dr. Sanders. There is, however, a slight modification to be acknowledged here, namely, that "although the patient with cerebral hemiplegia can close both eyes simultaneously, he cannot voluntarily close the eye on the same side as the paralysis while the other remains open" (BAZIRE). But it appears to me that this would only show *some extent* of paralysis if the patient were known to have been able to close either eye at will previous to his attack—a feat which many, especially females, are unable to perform who are free from any paralysis. From the threefold functions of the *portio dura* already noticed, and the diverse origins of its several sets of fibres,* it will easily be understood how, in very limited and localized cerebral lesions, *all* the branches of the facial nerve may not be affected. The extent of apparent paralysis in cases of cerebral facial hemiplegia is not usually so great, and therefore not so alarming to the patient, as in cases of Bell's paralysis, where the trunk of the nerve only is affected. But the prognosis must be more unfavourable. In ordinary facial paralysis of *cerebral origin*, Dr. Sanders has always found that the muscles of expression, including the buccinator, were more or less affected, while the action of the masseters, temporals, and pterygoids was unimpaired. The usual facial distortion was exhibited to a greater or less degree according to the amount of the paralysis, but it rarely approached the completeness usually seen in peripheral paralysis from lesion of the nerve trunk. With the exception of the slightness of implication of the *orbicularis palpebrarum* muscle, the phenomena of cerebral hemiplegia of the face are entirely similar to those of facial paralysis produced by lesion of the seventh nerve itself, and, as in the latter, are confined to the muscles of expression, including the buccinator, and do not involve the masticatory muscles (masseter, temporal, pterygoid) supplied by the *motor fifth.*

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* The seventh pair of nerves appear on each side, at the posterior margin of the

Another point of diagnosis is to be recognized in the behaviour of the paralyzed muscles under electric irritation. In facial paralysis of cerebral origin, the muscles respond *normally* to electric irritation; but if the paralysis is due to a lesion of the *portio dura*, their contractility is not at all, or scarcely at all, roused by an electric current (DUCHENNE, SANDERS, VULPIAN, TROUSSEAU). *The symptoms of cerebral facial paralysis* vary as the central lesion is more or less distant from the origin of the seventh pair. There is therefore much more variety in the expression of the face (according to the varying extent of the paralysis) than in cases of Bell's paralysis. The peculiar aspect of the face in *cerebral facial hemiplegia* is due to the want of symmetry between the two halves. The contraction of the muscles on each side is not co-ordinate. The sound cheek is wrinkled and shortened. The labial commissure on the sound side is drawn outwards and upwards, and is on a higher level than on the opposite side; the angle of the mouth on the paralyzed side is lower than its fellow; and if the paralysis is extreme, the commissure on the paralyzed side remains partially open, so that the saliva constantly escapes. The flaccid cheek is the result of paralysis of the buccinator. The nostril on the paralyzed side is more closed than on the sound side; but "in the great majority of cases of cerebral hemiplegia of the face, the *orbicularis palpebraris* is not materially affected; the act of winking and of voluntary closure of the eyes continues on the paralyzed as on the sound side, with the small exception that the voluntary closure is usually weaker on the palsied side.

pons, between the middle and inferior peduncles of the cerebellum, and nearly in a line with the place of attachment of the fifth nerve.

The *portio dura*, or facial nerve placed a little nearer to the middle line than the *portio mollis*, may be traced to the *medulla oblongata*, between the *restiform* and *olivary fasciculi*, with both of which it is said to be connected. Some of its fibres are derived from the pons. Phillipeaux and Vulpian affirm that the fibres arise from the outer wall of the fourth ventricle, and that many of them decussate in its floor. Connected with the *portio dura*, and intermediate between it and the *portio mollis*, is a smaller white funiculus. The roots of this accessary or intermediate portion are connected deeply with the lateral column of the cord (Quain's *Anatomy*, by Drs. Sharpey, Thomson, and Clelland, p. 587).

"These phenomena furnish the best diagnostic marks between *centric* and *peripheral* paralysis of the face; the hanging cheek, with wide open, staring, unwinking eye, denotes lesion of the *portio dura;* the flaccid face, with the natural position and motion of the eyelids, is the sign of cerebral lesion, and indicates a more serious disease" (RECAMIER, TODD, SANDERS).

There is difficulty in the articulation of labial consonants and vowels. The tongue protrudes in the normal direction, and if its point seems to diverge from the median line to the paralyzed side, it only seems to do so in consequence of the commissure of the lip being drawn in the opposite direction—namely, AWAY FROM the paralyzed side. But sometimes the tongue, too, is paralyzed and really deviates, and then *it is drawn to* the sound side, when the branches of the *portio dura,* going to the *stylo glossus* and the *genio glossus,* are implicated.

Opinions differ greatly as to the implication of the tongue, the uvula, and the soft palate in cases of cerebral facial hemiplegia. Trousseau, Todd, Hasse, Longet, Romberg, Bidder, and Sanders, all refer to such cases. Dr. Sanders shows that the position of the uvula varies frequently, both in the natural and hemiplegic palate. The only reliable sign that the palate is involved in the lesion of the *portio dura* is the existence of a vertical relaxation, or lowering of the (paralyzed) corresponding half of the *velum palati,* with diminished height and curvature of the posterior palatine arch on the paralyzed side (SANDERS). Such cases indicate lesion before the geniculate ganglion in the Fallopian duct, and implicating the petrosal nerves.

In both forms of facial paralysis the action of spitting is difficult, so also is whistling, as the patient cannot close the mouth nor inflate the cheek. A coachman was very thankful to Sir Charles Bell for the successful extirpation of a tumor near his ear; but complained that ever since the operation he could not whistle to his horses. No doubt the *portio dura* was cut in the operation. Mastication is badly done, but only so far as the buccinator is concerned, because the food accumulates between the paralyzed cheek and the teeth. The hand is often seen to be held almost instinctively against the cheek to give it support, and to press the food to between the teeth for proper mastication, which is efficiently performed by the muscles of mastication (*temporals, masseters, pterygoids.*)

In some cases of facial paralysis it has been noticed that hearing on the affected side becomes much more acute; and the taste is sometimes perverted, both indicating lesion high up within the bony canal traversed by the *portio dura* (TROUSSEAU, ROMBERG, BROWN-SÉQUARD, LANDOUZY, BAZIRE).

A peculiar and characteristic result of facial paralysis is the contracture and tonic spasms, or convulsions, of the muscles of the face, which have been paralyzed—the "*spasmodic tic*" of Marshall Hall.

Simple contraction of the muscles is very common after Bell's paralysis, when it has lasted a long time and been extreme. It comes on after the affection appears to be cured, and after the symmetry of the face has been apparently restored. At first, and most usually, the contraction is partial, and is mixed up with some degree of paralysis, the contraction of the muscles not being effected at will. This contraction is common to all muscles which have been paralyzed and out of use for some time. The spasms are sometimes painful, and the contracted muscular masses are painful on pressure. Sometimes one muscle and sometimes another is thus affected. Sometimes it is the orbicular muscle of the eyelid, when the eye, instead of being more open, as it was when paralyzed, now looks smaller and is more closed. Such contraction is a most frequent termination of paralysis due to *rheumatism*. A rapid return of tonicity in a paralyzed muscle, the electro-muscular contractility of which had been completely abolished, is always a sign of approaching contraction (DUCHENNE).

Diagnosis of facial paralysis, differentially, is based on a knowledge of the circumstances under which the paralysis set in, the progress of its development and the attending phenomena, bearing in mind the anatomical and physiological history of the *portio dura* already given. Sometimes the paralysis affects both sides; and one important test is given by Marshall Hall by which it may be known whether the cause of double facial paralysis is seated in the brain or in the course of the nerves. If the lesion is central (cerebral) the conducting power of the nerve trunks is retained for an indefinite period, so that by galvanizing the trunk and the principal branches of the facial nerves, all the paralyzed muscles supplied by the nerves are thrown into contraction, as if the muscles themselves were being galvanized; whilst, when the paralyzing lesion is *in the course of the nerves*, the nerves very easily lose their conducting power. If reflex movements be seen in the paralyzed muscles, the cause of the paralysis is assuredly a lesion of the nerve centres.

Prognosis.—Bell's form of peripheral facial paralysis generally gets well in time; and all the more rapidly that it sets in suddenly, and the patient young. From four to ten months is the ordinary duration of the affection; but there are instances in which the paralysis yields in twenty-four, fifteen, or even twelve hours, but such cases are exceptional (TROUSSEAU). The prognosis in *central facial hemiplegia* is less hopeful; so also in *double facial hemiplegia*; because the cause indicates—from the symmetrical nature of the exciting lesion (generally periostitic)—some constitutional source as the origin of the mischief, *e.g.* scrofulosis or syphilis.

Treatment.—Paralysis due to lesions which are destructive of the nerve are beyond medical aid. In cases suitable for treatment the agents used with most benefit are,—Blood-letting by leeches behind the ear of the affected side, or over the mastoid region. They ought to be applied repeatedly, especially if any pain exists on pressure in that region. Blisters, with dressings to keep them open, medicated by veratria or strychnia ($\frac{1}{25}$ to $\frac{1}{5}$ of a grain of *sulphate of strychnia* or of *veratria*), mixed with five or six times their weight of pounded sugar. Electrization in the form of Faradization by a Stöhere's two-celled volta-electric machine; or in the form of an *interrupted galvanic current*, from a *continuous current* battery (as from a Muirhead's battery of fifteen cells), the number of cells being very gradually increased.

In using Faradization each facial muscle should be separately galvanized, instead of attempting to pass the current through the facial nerve. The muscles are thus more powerfully influenced, and they are more apt to regain *voluntary* contractility at different periods, some muscles before others. At first the apparatus should be one capable of producing currents with very rapid intermissions; but when any muscles begin to *contract*, the intermissions should be few, and the sittings at long intervals, otherwise contraction of the muscles is apt to increase and become incurable.

The interrupted galvanic current, from a continuous current battery, induces contractions when the circuit is closed and when it is opened, and more powerfully at the moment of closure. It is said to restore to palsied muscles the power of contracting under the influence of Faradization. When improvement follows the use of the interrupted galvanic current from a continuous battery, the number of the cells should be gradually increased as the muscles get less susceptible to their influence. One of the electrodes should be gently moved along the surface of the muscle, just as the brush is used in Faradization.

When contracture of muscles supervenes, they ought to be stretched or pulled out mechanically. To remedy contraction of the buccinator, Duchenne recommends the use of a small billiard ball worn for a long time inside the cheek, to be replaced after a time by a larger one. The late Dr. Bazire gave favourable testimony to the value of this mechanical aid.

The daily action of the bowels ought to be carefully regulated, especially by such medicines as keep the colon in functional activity. *Bichloride of mercury* (*corrosive sublimate*) in small doses, continued till the gums are slightly tender, is sometimes of service.

In cases where rheumatism prevails or periostitic affections, small doses of *iodide of potassium*, persevered in for a lengthened period, are of service, with the frequent use of *phosphate of soda* as a daily morning aperient.

(*b*) SCRIVENER'S PALSY.

LATIN EQ., *Paralysis notariorum;* FRENCH EQ., *Crampe des écrivains;* GERMAN EQ., *Schreibekrampf;* ITALIAN EQ., *Crampo degli scrivani.*

Definition.—*Morbid excitement of the motor fibres of the nerves of the muscles of the fingers and thumb holding the pen, resulting in cramps, so as completely to prevent writing, and thus the condition becomes equivalent to a local palsy or paralysis.*

Pathology.—A want of coördination of the muscular movements engaged in writing seems to be the first pathological element in the disease. In this respect the disease approaches in form to locomotor ataxy, where there is partial loss of controlling power. The constant practice of the same movements sets up some irritation producing pain; and also every attempt to write calls forth uncontrollable movements in the thumb, the index finger, and middle finger, so that the pen starts up and down on the paper. The handwriting ceases to be legible—a mere scrawl results, or grotesque interrupted scribbling.

The more the patient persists in the attempt to write, the more does the difficulty of steadying the hand and using the pen increase. The visible and sensible contractions of the muscles of the thumb and fingers are soon followed by similar contractions of the fore arms, even extending in some cases to the upper arm. Apprehensive attention to the subject and dread of the occurrence of spasm are generally sufficient to ensure a paroxysm after the disease is well established. The hand and arm seem capable of every other combined movement except that of writing; and when all attempts at writing cease, then the spasms also subside and entirely disappear. On the other hand, the more the patient attempts to continue writing, so much the more violent does the spasm become. The disease is not entirely limited to the operation of writing. Shoemakers, milk-maids, or milkers of cows, goats, and ewes, nail-smiths, musicians, compositors, saddlers, sempstresses, and men who handle small hard articles with considerable muscular grasp are subject to similar cramps. Hence the disease is known under a variety of names—as *cobblers' spasm*, *milkers' spasm*, *nailers' spasm*, *writers' cramp*.

One theory regarding the production of the cramps implies that they are reflex through the excitement of the *muscular nerves* or *muscular sense*. Hence, holding the hand even in the *attitude* of writing, although it does not grasp a pen nor touch the paper, will induce the spasms.

On the other hand, it is believed that the irritation is similar to what takes place in the convulsive movements of chorea and stammering.

Causes.—The affection is much more frequent among men than women, and between the ages of thirty and fifty years. It chiefly occurs among professional penmen, clerks, teachers, and those who habitually write monotonously according to prescribed and constantly repeated forms—as in mercantile books and the like details of form in business. It rarely is seen among short-hand writers, whose brain is so constantly occupied with the subject in hand that attention to the mechanical process of writing seems in abeyance. Nor is it seen among judges, or lawyers, or professional men, who also write much, but with little attention to the mere mechanism of writing—so much so, that the writing of many such men is hardly readable, or at least may be legible only to a few when accustomed to read the writing. An eminent judge is said to have had three different hands of writing—one he could read himself, and no one else could read; another that his secretary or clerk could read, and which he could not himself read again, nor anyone else; and a third that neither himself, nor his clerk, nor anyone else could decipher. These results were not due to writers' cramp or spasm of any kind. The hard pen, the formal writing, and the mere mechanism of the art, seem to be the conditions tending to induce the characteristic spasms.

Symptoms.—The indications of the disease commencing are quite painless, beyond a feeling of tension or stiffness in the muscles of the arm and fore-arm or fingers only of the hand. There may be a feeling of fatigue of the hand, and a tendency to grasp the pen more firmly, especially towards the end of the day's work, which is generally relieved by a night's rest. It is not till the disease is fully expressed that convulsions or spasms occur. Movements become unsteady by the thumb being drawn towards the palm, while the index and middle finger become contracted with more or less rigidity.

Treatment.—In one case by Stromeyer, division of the muscle in which the cramp seemed first to commence had a good effect. In fourteen days after subcutaneous section of the long flexor of the thumb, the patient was again able to write. Dieffenbach often repeated a similar operation, but without success. Mr. Solly, in his lately published surgical experiences, describes several cases of this peculiar affection, in which tonic treatment, with rest from writing, was attended with good results. Niemeyer has been successful with the galvanic current. He applied the current to the muscles of the thumb and index finger. Exercise of the affected muscles materially tends to increase the disease. Complete rest from the usual mechanical use of the hand must be insisted on. Nourishing food, with a milk diet in abundance, or cod-liver oil are essential.

As to medicines, when the disease has not gone beyond the sensation of heat and cramps in the ball of the thumb, I have seen good results from the *syrup of the phosphates of iron, quinine, and strychnia;* and generally those remedies noticed under the subject of anæmia may be prescribed. Mechanical appliances, such as contrivances which fill up, as by a ball, the palm of the hand on which the fingers rest in writing, have been of service; and there are also appliances by which writing may be accomplished without the aid of the fingers, and which therefore may be of service in securing rest.

(*c.*) GLOSSO-LARYNGEAL PARALYSIS—Syn., GLOSSO-PHARYNGEAL PARALYSIS.*

Definition.—*Diminution and subsequent loss of the motor power of the tongue, soft palate, and lips, associated with structural changes in the roots of the motor nerves which supply the affected muscles. The disease progresses always rapidly to a fatal termination.*

Pathology.—Concurrent paralysis of the lips, tongue, *vellum, palate,* and vocal cords, together with the associated muscular movements of deglutition, have recently been recognized, as more or less capable of explanation by the close anatomical connection between the muscles supplied by the *vagus,* the *spinal accessory,* and the *lingual* nerves. The *lower* rootlets of the spinal accessory nerve (forming the external branch) arise, in common with the anterior roots of the spinal nerves in the cervical and brachial region, from the anterior grey substance of the spinal cord; while the *upper* rootlets (forming the internal branch) have a totally different and a double origin—one from a special nucleus continuous with that of the pneumogastric behind the central canal, and the other from the proper nucleus of the hypoglossal, in front of the canal. Some of the fibres of the hypoglossal seem to take their origin from the spinal accessory nucleus (LOCKHART CLARKE, *Beale's Archiv.* No. 3).

The anatomical lesion involves the centres of the *pneumogastric, hypoglossal* and *lingual* nerves; and in the paralysis under consideration there is *atrophy of the motor roots of these nerves, sometimes extending to the anterior roots of several of the upper spinal nerves, attended with more or less paralysis of limbs and incipient muscular atrophy.*

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* (Not recognized by the College of Physicians.)

Glosso-pharyngeal paralysis and progressive muscular atrophy are found to be attended with the same nerve-lesions—namely, atrophy of motor roots of nerves, cranial as well as spinal; and in all patients suffering at first from glosso-pharyngeal paralysis there is a tendency for the paralysis to become general (TROUSSEAU). Gradual disappearance of the nerve-elements, and proliferation of connective-tissue from the neurilemma, with intense fatty atrophy, are the characteristic anatomical lesions at the roots of the affected nerves.

In some cases diffuse sclerosis of the *medulla oblongata* has been found (NIEMEYER). According to Wachsmuth the atrophy of the nerves is secondary to "a cerebral affection of the *medulla oblongata* having its seat in the olivary bodies, and in the grey matter lying far back between the diverging lateral and posterior columns forming the floor of the fourth ventricle."

The disease, by French writers, is regarded as closely related to progressive muscular atrophy, with progressive palsy of cerebral nerves.

Symptoms.—The earliest and most noticeable are those which are due to palsy of the muscles of the tongue, the soft palate, and the lips, those of the larynx and pharynx becoming implicated at a later period. The patient cannot blow, whistle, nor spit, nor pucker up his mouth, and saliva runs from the mouth involuntarily. The origin, progress, and termination of the disease are so characteristic that, according to Trousseau, there is no other identical affection in the whole range of nosology. Embarrassment of speech first attracts attention. The tongue seems less supple, and the utterance becomes more and more thick. The food is apt to lodge between the teeth and the cheek, the cause of this being different from that which obtains in Bell's paralysis. In such facial paralysis it is due to paralysis of the buccinator muscle; here it is due to the circumstance that the tongue being more or less paralytic, awkward, and incapable at the tip, the patient is obliged to use his fingers to remove the food from between the teeth and the cheek, and so replace it on the tongue. Pronounciation of certain words is made through the nose. The vowels *o* and *u* cannot be properly sounded, on account of the deficient contractility of the *orbicularis oris* muscle. Saliva is apt to dribble from the lips and corners of the mouth. The paralysis continuing to progress, the tongue at last lies motionless in the hollow of the mouth, behind the lower teeth. Its apex and base are equally motionless, and not a word can be articulated. The shape of the tongue is also altered. It has sunk down in the centre, presenting a hollow in the middle line, with its edges raised. The soft palate also droops, and the tip of the uvula rests upon the tongue, and is generally callous or insensible to irritants. The first stage of deglutition thus becomes impossible. The morsels are swallowed by holding the head backwards, and facilitating their gliding down by fluids. Sometimes only a small quantity of the food gets into the œsophagus, the remainder being propelled upwards through the mouth and nostrils, and sometimes small portions of food will find their way into the larynx, causing great distress. The appetite remaining good, but swallowing being thus impossible, constant hunger aggravates the distress (TROUSSEAU).

The expression of the countenance is blank and strange. Excessive weakness of the respiratory movements is soon superadded to these already serious symptoms. The walls of the chest and the diaphragm scarcely move. If the patient be asked to blow out a candle he cannot do it. The flame will be scarcely agitated by his utmost efforts to blow upon it. Coughing is equally inefficient; so that if catarrh should supervene, there is great difficulty in expelling the increased secretion of mucus. Asphyxia is thus apt to prove fatal. The heart's action becomes abnormally rapid, but fever does not exist. The body temperature tends to sink below the normal, and betokens, with the other phenomena, the imperfect oxygenation of the blood. General debility now makes rapid progress, and the patient rather inclines to remain in bed, sitting up with his head supported on pillows, inclined to one side sufficiently to allow of the saliva flowing away which he is now unable to swallow. Sleep is disturbed by paroxysms of suffocation, and death is apt to ensue suddenly by cessation of the heart's action, unaccompanied by pain or noise (TROUSSEAU. See also Wilks in *Medical Times*, 1868, p. 281).

Diagnosis.—From the general paralysis of the insane, it is distinguished by the intellect of the patient remaining perfectly clear; and the gravity of the complaint impresses itself on the mind. The convulsive movements of the lips are also absent in general paralysis. In cases of hemiplegia, the palsy is unilateral. In double facial paralysis —a rare affection—the movements of the tongue are free, and deglutition remains efficient, all the muscles of the face are paralyzed, so that expression is as blank and fixed as marble, the patient laughs or cries as if from behind a mask (DUCHENNE); but in *glosso-laryngeal paralysis* the lower part of the face alone remains motionless.

Prognosis is unfavourable. The disease has always proved fatal. Its progress is generally rapid and continuous, although sometimes tedious. From three to six months it may not seem to make so rapid a progress as it does later; but as soon as deglutition becomes imperfect, death is rapid.

Treatment.—Faradization is of doubtful efficacy at the commencement, but gives relief at later periods, by temporarily restoring function to the affected muscles, to the lessening of the trouble of deglutition, and by exciting the respiratory acts.

One other form of *local paralysis* can only be mentioned here, as space cannot be given at present for its consideration,—namely, (*d*) *paralysis of the Serratus muscle*, which seems to be a peripheral nervous affection. The muscle is supplied by the *long thoracic nerve*, and the exciting cause of the palsy is obscure; but the nerve is very liable to injury in its lengthened course. The lower angle of the scapula becomes dislocated, and stands up like a wing, and the arm cannot be lifted above a horizontal line.

Another form may be mentioned as (*e*) *paralysis* by pressure on a nerve, as when one falls to sleep with the arm hanging over the back of a chair. When the limb recovers, it is preceded by the sensation of "pins and needles," due, I believe, to the return of vascularity to the sheath of the nerve.

SECTION VII.—FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

TETANUS.

LATIN EQ., *Tetanus;* FRENCH EQ., *Tétanos;* GERMAN EQ., *Tetanus,*—Syn., *Starrkrampf;* ITALIAN EQ., *Tetano.*

Definition.—*Involuntary, persistent, intense, and painful contractions, cramps, or spasms of more or less extensive groups of voluntary muscles; so that the whole of the body may seem to be affected, the spasm yielding and becoming intermittent in some groups of muscles, while at the same time it exists in others, or the continuous tonic spasms are characterized by paroxysmal aggravation of extreme intensity.*

Pathology.—This is one of the most formidable diseases of the nervous system. The body has often been most minutely examined, after the patient has died from idiopathic tetanus, without any lesion being discovered. When death has followed from traumatic tetanus, nothing has been found, in many instances, except, perhaps, the primary wound. In a few cases the membranes of the brain have been found congested; but not in a greater degree than might have been predicated from the violent and long-continued muscular action incident to the disease. In a fewer number of instances small patches of cartilages or of bony matter have been found on the spinal arachnoid membrane; but as these are often absent, they are not essential conditions of the disease. It seems proved, therefore, that tetanus commences as a disease of function; and, as Majendie has shown, if the spinal cord of a living animal be divided into as many segments as there are vertebræ—that the animal, if poisoned with strychnine, still becomes tetanic, although all direct connection of the muscles with the brain is destroyed—it seems probable that the cord, as high as the fifth pair, and not the brain, must be the great seat of this affection.

The morbid irritation of the motor nerves proceeds from the spinal marrow; yet real tetanic spasms are seldom seen in cases of grave organic disease of the spinal cord, and as a rule, lesions which give rise to great excitement or exaltation of function are not susceptible of anatomical demonstration.

When the disease commences, spasms are induced by trifling but very appreciable irritants, which, acting on the peripheral nerves, induce the spasms in a reflex manner. After several repetitions by such reflex irritation, the spinal marrow seems eventually to assume a condition of permanent excitement. Some organic change is established in the nerve-centre, which no longer needs the primary irritation in the circumference or periphery to maintain it. So long as the phenomena are peripheral and reflex, the removal of the offending part may arrest the disease; but when it has become central, it is probably always fatal. There are thus two forms of tetanus which demand recognition as early as possible.

My own observations on the specific gravity of portions of the cord in tetanus lead me to the belief that the blood-vessels, and thus vascularity, rather than nerve tissue lesion, have much to do with the phenomena.

In numerous cases I examined in Glasgow Hospital (see p. 232, *ante*), where I chopped up the cord into pieces, those pieces connected with the nerves going to and proceeding from the injured part, showed a marked increase in specific weight compared with other portions of the cord. (See articles on "Tetanus," by Dr. Lawrie, in *Glasgow Medical Journal*, No. 3, Jan., 1854.) Dr. Lockhart Clarke has since shown that even fluid disintegration of the grey matter of the cord takes place in cases of tetanus.

This disease was well known to the ancients, and is described by Aretæus with all his usual terseness and precision. It is the frightful accompaniment of wars and battles, but occurs from accidents, or spontaneously in many instances in civil life.

Cause.—Sometimes idiopathic in its origin (but more often traumatic), it is classed as of functional origin; and its treatment is assigned to the physician or the surgeon according to the latent or apparent source of the disease; but, as Le Gros Clark justly observes, such distinctions are purely conventional, since it matters not "whether an external wound be the cause of the disease, so far as the nature of the disease is concerned. Some antecedent disturbing cause exists, usually, if not invariably peripheral in its nature, and affecting some distant part of the nervous system. *Idiopathic*, therefore, is not to be understood as synonymous with "*causeless*" or "*spontaneous*." It merely means that the cause is unseen, or has not been identified or demonstrated, and is therefore probably unknown, or a mere matter of conjecture.

The morbid state of the spinal marrow, to which tetanus is due, is capable of being induced by a variety of noxious agents; but it is most frequently met with in armies on actual service, as the result of wounds, especially of lacerated, punctured, or gunshot wounds, or wounds made by large projectiles, as cannon-balls, bombs, or of the amputations rendered necessary by those wounds, or from wounds in which foreign bodies remain lodged in the part. Injuries of this kind are more dangerous upon the extremities than upon other parts of the body.

The conditions most favourable to the induction of tetanus are sudden changes of temperature, especially hot days followed by cold nights—so that the wounds are the predisposing, and exposure to cold, damp, and chill the exciting causes. When tetanus arises from the first of these causes, without any other being evident, the tetanus is said to be *traumatic*; when it arises from cold, damp, and chills, it is said to be *idiopathic*. The latter, however, is believed to be extremely rare in this country. The disease is liable to follow any kind of injury, from the most trifling cut or scratch to a compound fracture or most severe operation. Negroes seem more liable to the disease than Europeans. It follows also strains, contusions, and lacerations, and it is principally from these latter causes that it is met with in civil life.

Most authors consider it to be most common in hot variable climates, as that of Egypt. After the battle of the Pyramids, says Baron Larrey, upwards of 500 of the wounded were attacked with tetanus. The same authority adds, that the tetanus of Egypt was much more intense than that he had observed in Germany. He states, also, that this disease is much more common in all countries at those times of the year when the temperature passes rapidly from one extreme to the other, or in the spring, than in seasons when the temperature is more equal. Thus, after the battle of Eylau, fought during the depth of winter, not one of the guard, and very few of the line, were seized with this affection.

Besides wounds, strains, and contusions, some morbid poisons appear to produce this affection. Two men descended into a soap-boiler's vat to clean it out: on reaching the bottom, they both fell down in convulsions. They were quickly rescued, when it was discovered that *sulphuretted hydrogen* had been generated and remained at the bottom of the vat. Both of these persons were seized with tetanus, of which they died. *Strychnia, brucia,* or their salts, or vegetable matter containing either or both those alkaloids—as *nux vomica, St. Ignatius's beans,* or the juice of the *upas tree*—are also well known poisons, capable of producing this affection; and the poison of *cholera,* in severe cases, has also had the same result. These poisons, administered by the stomach or inoculated into the system, induce all the symptoms of intense tetanus, and which has therefore been termed *artificial tetanus,* or *tetanus toxicus.* There is no test by which the *artificial* can be distinguished from the *real tetanus,* by which the results of poisoning and of disease can be distinguished, except that the disease never proves so quickly fatal as the rapid cases of poisoning with *strychnia* (CHRISTISON). (See also "Report of Palmer's Trial.") The invasion of tetanus can scarcely be mistaken; but the general premonitory symptoms may be so undefined as to appear unimportant, and so escape notice; not so, however, the uneasiness about the neck, the sense of constriction about the throat, and the stiffness of the jaws.

A wound is generally the remote cause of true tetanus, and its nature and site appear to predispose to the disease. Thus it is most common after injuries of the ginglymoid joints, as that of the elbow or knee, or when the bone is extensively fractured or comminuted. Its occurrence is also more probable if a foreign body remains in the wound, and especially if, after amputation, a nerve has been included in the ligature round the artery. In other respects, the state of the wound does not appear to influence the attack, for it appears to take place equally whether it be open or cicatrized, granulating or suppurating, incised or contused; but if there be any difference, Larrey thinks the period of detaching of the eschar, especially if the stump be exposed to cold, is the most critical. It is singular, however, that time tends to destroy the predisposition given by the wound. Sir James Macgregor gives as the result of his great experience, that no person is attacked with tetanus after the twenty-second day from receiving the wound, a period which Sir Gilbert Blane extends to the fourth week. The interval between the reception of the injury and the first tetanic symptoms varies from the fourth to the fourteenth day, and rarely exceeds twenty-two days. Some time in the second week is the most common period. The more rapidly the disease comes on the more fatal will be the result.

The relative proportion which the occurrence of tetanus bears to various classes of surgical lesions are given by Mr. Polland as follows, from the Records of Guy's Hospital for seven years (see *Holme's System of Surgery,* vol. 1) :—

Major and minor operations,	1364	cases.	Tetanus	occurred in	1
Wounds of all varieties,	593	,,	,,	,,	9
Injuries and contusions,	856	,,	,,	,,	1
Burns and scalds, -	458	,,	,,	,,	3
Compound fractures, -	396	,,	,,	,,	9
	3667				23

It thus appears that tetanus is most frequently met with in the more severe varieties of injury and accident—such as compound fractures, burns, and injuries to the fingers and toes. It is still a matter of doubt whether the seat of injury has any influence in establishing the disease. One of our greatest authorites on Military Surgery observed it oftener after wounds of the elbow and knee (HENNEN); others again from injuries of the thumb and great toe. A popular belief prevails that wounds of the ball of the thumb are prone to induce tetanus.

All ages are liable to this disease, and even new-born children suffer from it, the "*trismus nascentium*" being ascribed to the tying of the navel-string. It occurs between the first and fifth day after separation of the navel-string. Tumbling boys are also frequently seized with this complaint. It is most common, however, in adult age; and if less frequent in old age, this circumstance is probably owing to persons in advanced life being little exposed to those accidents which usually produce it. Both sexes suffer from it; but men far more commonly than women.

Symptoms.—There is usually a certain order in which the different sets of muscles are afflicted. The muscles of the neck, jaws and throat are almost always the first to indicate the disease, as well as a peculiar expression of the face given by the contraction of the muscles about the eyebrows. There are five varieties of tetanus—*trismus*, *tetanos*, *emprosthotonos*, *opisthotonos* and *pleurosthotonos;* and when either of these terminates within eight days it is said to be *acute;* but if prolonged beyond that time it is considered *chronic.*

Trismus is that state in which the disease is limited to the muscles of the lower jaw and throat.

Tetanos is marked by the flexor and extensor muscles of the body generally being equally and strongly contracted, keeping the whole frame in such a state of tension that if you attempt to raise the leg, you raise the whole body, it being as inflexible as in death (BARON LARREY).

Emprosthotonos is when the flexor muscles bend the body forwards.

Opisthotonos when they bend the body backwards.

Pleurosthotonos is when they bend it laterally, or on one side only.

The frequency of occurrence of these different forms of tetanus is not accurately determined; but *trismus* is the most common: and though it may exist *per se*, it is generally the first and concomitant symptoms of all the other forms. After *trismus*, *opisthotonos* is far the most common, both in this country and throughout Europe; but Baron Larrey says that *emprosthotonos* was most common in Egypt. Of *pleurosthotonos* only a very small number of cases are to be found recorded in the whole annals of medicine.

The attack of either form of tetanus may be sudden; but more frequently it is preceded by an uneasy sensation and tension of the præcordia, followed by stiffness of the neck, shoulders, and lower jaw. There is a difficulty and uneasiness in bending or turning the head—a sensation like that commonly known as "*stiff neck.*" The mouth opens with difficulty, and at last the jaws close completely, sometimes gradually, but always with great firmness—so that the disease has popularly got the name of "*lock-jaw.*" At length the patient feels a sudden and painful traction of the ensiform cartilage; and this latter symptom is considered a pathognomic sign of the disease. The muscles concerned in swallowing become affected.

At this point the disease may stop, and the phenomena be limited to *trismus;* but more commonly the patient takes to his bed, and the disease assumes one of its severe forms, as of *opisthotonos, emprosthotonos, pleurosthotonos,* or of *tetanus.*

The pain in the sternum is one of the most distressing parts of the disease. It is an acute pain at the lower part of the sternum, piercing through to the back, and is believed to depend upon cramp of the diaphragm.

In *opisthotonos*, in addition to the *trismus,* the muscles of the face are generally spasmodically affected, for the forehead is wrinkled, the brow is knit, the corners of the mouth are drawn, giving to the patient a most wretched grin—the *risus sardonicus.* The angles of the mouth are drawn outwards, and the lips are drawn apart, so as to expose the firmly clenched teeth. The eyes also are almost motionless and sunk in the socket; and, during the attack, the tongue is projected against the teeth, so, except for the trismus, it might be caught by a convulsive snapping of the jaws, and severely injured. The characteristic of this form of the disease, however, is, that the flexors of the back are thrown into such powerful action that the spine becomes arched, and sometimes to such a degree that the body rests on the occiput and heels, as on the extreme points of the segment of a circle. A most graphic painting of such a patient, by Sir Charles Bell, exists in the Museum of the College of Surgeons of Edinburgh. The flexors of the back, however, are not the only muscles affected, for the shoulders are thrust forward by a strong action of the pectoral muscles, while the extremities are elongated and tightly braced by strong contractions both of their flexors and extensors. Indeed, the whole of these different sets of muscles are thrown into action at the same moment, as if by the discharge of a powerful galvanic battery. The contracted muscles seem as hard as wood, and are the seat of such frightful pain as can only be appreciated by any one who has suffered from severe cramp in the muscles of the leg. The shock is transitory; and, having passed off, an interval succeeds which varies from a few minutes to half an hour, an hour, two hours, or longer, according to the severity of the disease.

Exacerbations of the spasms commonly occur every ten or fifteen minutes, usually beginning by an increase of pain at the sternum, and lasting for two or three minutes. These paroxysms become more frequent—a shorter interval between—as the disease advances. But during this interval the patient lies as in his coffin, with his arms close to his sides, and his legs stretched out and touching each other, fearing lest the slightest motion should produce a recurrence of the attack. His nights, or such few as he lives through, are sleepless, or only marked by a few minutes' broken slumber. Such is an attack of opisthotonos. The other forms of the disease differ only by the different sets of muscles affected.

It is difficult to give an idea of the distressing violence of the spasms. They may be imagined when it is stated that Desportes gives a case in which both thighs were broken. But notwithstanding this strongly powerful action of the muscles, the patient's mind is seldom affected, and his pulse presents its healthy beat, only a little accelerated. The intercostal muscles partaking in the general spasm, the respiration is carried on principally by the diaphragm; and when the attacks are frequent the breathing is short and laborious.

The respiratory muscles are *specially* involved in tetanus; and the *diaphragm* is no doubt affected in company with other and voluntary muscles of inspiration and expiration. The excitement of the spinal cord, as a centre and source of reflex motor power, overrides the controlling influence of the brain. The inhibitory authority of volition is thus suspended, leaving the voluntary muscular system generally under the undisputed sway and dominion of excited reflex action (LE GROS CLARK).

Neither the nature of the wound nor its condition bear any relation to the intensity of the symptoms; and the tetanic spasms may not be developed till the causative lesion is healed, and may then prove speedily fatal.

The skin is, after a short time, covered with a profuse sweat, as during great exertion. The tongue is clean and moist; but the bowels are generally constipated, and the *sphincter ani* so contracted, that it is difficult to introduce a glyster-pipe. In cases in which tetanus supervenes on a suppurating wound, the sore dries up and is painful, while the muscles of the part are highly irritable.

The Intellect is not involved in tetanus; and in this respect it is more allied to chorea than to any other nervous disease.

In mild cases the paroxysm returns only three or four times in the twenty-four hours; while in severe cases it returns not only every hour or every quarter of an hour, but every motion of the body or attempt to open the mouth is followed by an attack.

In the last stage the situation of the patient is most pitiable, the spasm returning every few minutes, till he is at last cut off during one of unusual violence. Death usually results from a mixture of causes, but chiefly from *apnœa*, or "want of breath," due to spasm of the larynx, or to the fixed condition of the respiratory muscles lasting longer than the necessities of respiration can withstand; associated with *asthenia* from flagging of the heart's action. "Unrelaxing cramp, exacerbations recurring with increasing frequency and violence—the broken rest, the imperfect nourishment of the frame—the exhausting effects on the nerve-centres, resulting from the continuous demands on them by the unremitting spasms,—all aid in bringing to a speedy close the acute form of the disease; and the unhappy victim sinks into a state of prostration, both physical and mental, which brings with it some relief to his sufferings before he dies" (LE GROS CLARK).

The duration of this disease is various. In some instances death occurs in twenty-four hours; more commonly on the second, third, and fourth day, and is seldom protracted beyond the eighth. Some few persons survive till the seventeenth or twentieth day; and in such cases the disease generally terminates in recovery.

Diagnosis.—Independent of *Tetanus*, the jaw is sometimes locked by enlargement of the cervical glands, by difficult dentition of the wisdom teeth, and also in some forms of hysteria. The tumor, however, in the one case, and the hysteric symptoms in the other, are circumstances which readily enable us to distinguish them from *trismus*. The formidable phenomena of tetanus are seen in no other disorder except cholera; but the other differences between the two diseases are so extremely marked, that it is impossible not to distinguish them.

Prognosis.—The prognosis in this disease is always most grave. In the Peninsular war, although hundreds of cases were treated in every different manner, yet few, very few survived. In civil life the chances are something more favourable; and if the accident be of little moment, and the patient very young, he sometimes recovers. Dr. Parry writes, if the pulse be not more than 100 or 110, up to the fourth or fifth day, the patient almost always recovers. The danger decreases in general also in proportion to the duration of the disease, for few patients die after the fourteenth day. The danger in the early stages is to be estimated by the frequency and violence of the paroxysm.

As is the case with certain fevers, so is it with tetanus; it seems to have a definite course to run. It is in cases of tetanus that the maximum of body-temperature has been observed—112·55° Fahr. by Wunderlich, and 111·9° Fahr. by Lehmann; and fifty-seven minutes *after* death, in the first of these cases, Wunderlich observed the temperature rise to 113·675° Fahr. These rises are accomplished in very brief spaces of time. The Professor of Veterinary Surgery at Dorpat (Hofrath) also informs Wunderlich that he has observed temperatures above 107·6° Fahr. in fatal cases of tetanus in horses.

Treatment.—Baron Larrey affirms that this disease, if left to nature, is quickly fatal.

One of the most remarkable features of the complaint is the insensibility of the brain and nervous system generally to the action of our most powerful remedies; so that they are not merely inefficacious, but almost inert. Sir James Macgrigor says that all the most powerful remedies were fully tried in the Peninsular war, and that little or no dependence could be placed on any of them.

Opium was largely tried after the battle of Albuera, and given in the enormous dose of twenty grains every three hours; and yet it not only failed in curing the disease, but did not even produce sleep.

Mercury was tried after the battle of Salamanca, and to such a degree that strong mercurial ointment was rubbed in three times a-day in unlimited quantity. It entirely failed to cure the disease. One man, strongly under the influence of mercury, was seized with tetanus and died; while Baron Larrey's experience in Egypt led him to believe that mercurial frictions only aggravated the symptoms.

Opium and *mercury* were then combined; but, according to Sir James

Macgrigor, the combination was as inefficacious as their separate administration.

Wine and *brandy* were used in unlimited quantity; but without producing intoxication, or mitigating the symptoms.

Many other stimulants, as *musk*, *æther*, *camphor*, were afterwards employed, but equally without success.

Venesection had also a fair trial in several quarters, and in a great number of cases; but only one man recovered.

Tobacco glysters are not only not serviceable, but have been sometimes followed by the sudden death of the patient.

Digitalis has equally disappointed the hope which had been entertained of it; and one man is said to have died under its depressing influence.

Prussic acid has also been tried and failed.

Dr. Elliotson speaks in high terms of the *carbonate of iron;* and he has given it to the extent of 2 lbs. in the twenty-four hours; and under this treatment two out of three cases recovered. The instances, however, are far too few to enable us to decide on the value of this medicine, especially as a very long period has elapsed without any confirmatory evidence.

The warm bath has been thought serviceable in some cases; but patients have died while immersed in it. The cold bath is worse than useless: it is dangerous. Baron Larrey speaks of a patient who had twice gone into the cold bath, but with so little benefit that he absolutely refused to encounter a third. A blanket, however, was thrown over his head, and he was then plunged into the water. He died a few hours after, when it was discovered he had ruptured the *rectus abdominalis* muscle in all its thickness. Dr. Elliotson speaks of a case in which the patient was taken out of bed and placed in a tub in the middle of the ward, when a pail or two of water was dashed over him. The man fell dead as if he were shot. It appears that all the *heroic modes* of treatment which medicine offers have been tried and failed.

Much good, however, is gained by attempting to restore the secretions to a healthy state; also by supporting the patient, and by endeavouring to tranquillize the high irritation under which he is labouring. The medicines to be employed are moderate doses of purgative medicines, with *tinct. opii* ♏ v., or its equivalent, 10 grains of *Dover's powder*, given every three or four hours; and these conjoined with moderate quantities of wine, sago, or other nutritious diet. *Musk* also, in ten grain doses, has been given with some advantage.

Some authors lay much stress on a local treatment in traumatic tetanus. Baron Larrey, as the result of his great experience, says, "When it is caused by the wound, we should not hesitate to operate on the first symptom of tetanus, and thus, as far as possible, remove the causes of irritation. If tetanus follows amputation, he recommends the stump to be sprinkled with powdered cantharides; and in cases where a nerve has been included in the ligature, that the ligature be removed either by section or by actual cautery. In the British army, however, all these proceedings have been adopted, and with very little success. Amputation has been frequently performed without any mitigation of the symptoms. The wound has also been excised, submitted to actual cautery, been blistered, and dressed with every ointment; but in general the disease has run its course, either uninfluenced, or its fatal termination has been accelerated. Hennen has even seen the wound heal and the patient die on the same day. Nothing, in fact, is so unsatisfactory as the results yet obtained from either the general or local treatment of this fatal affection. Larrey has often attempted, from the difficulty of swallowing fluids that sometimes attends *tetanus*, to pass an elastic tube; but in all cases he says he met with a contracted state of the œsophagus impossible to overcome; while the attempt was constantly followed by the immediate occurrence of the severest spasmodic attacks.

"If, however," as Mr. Poland observes, "we can help our patient on one day after another, we gain much. Constant watching and constant attention are required by night as well as by day, and unflinching perseverance on the part of the sufferer to aid all efforts made in his behalf, besides the avoidance of all causes of excitement, and more especially cold air or winds; taking care to preserve a uniform temperature as much as possible.

No hope of recovery may be indulged in unless the spasmodic seizures continue to diminish in length and frequence, distinct relaxation of the contracted muscles occurring during those intervals of diminished spasm; and unless the patient becomes able to take food, recovery in the fortunate cases takes place with extreme slowness; and it is not till after the lapse of weeks that the muscles entirely lose their spasmodic tension. Russian vapour baths are well spoken of by Hasse and Niemeyer.

Too free use alike of *narcotics* and of *anæsthetics* must be guarded against. They are valuable aids in maintaining the existence of the patient from paroxysm to paroxysm.

The hypodermic injection of a solution of *curare* deserves extensive trial, commencing with such small doses as $\frac{1}{8}$ to $\frac{1}{4}$ of a grain, gradually increasing from $\frac{1}{2}$ to $1\frac{1}{2}$ grains. A solution of *one* to *two* grains in 100 drops of water should be made fresh, and 10 drops of this solution is injected as a dose. The action lasts about four or five hours, and upon this fact its repetition must be regulated (DEMME).

Aconite has been used in large and repeated doses by De Morgan, Smith, Sedgwick, Woakes, and Wunderlich. It ought to be used hypodermically, and there appears to be a great tolerance of the remedy.

Cannabis sativa or *Indian hemp* is a remedy of real value, if it can be got genuine and fresh. Three grains of the *extract*, or thirty drops of the *tincture*, may be given every half hour or hour till narcotism is attained. Cold should at the same time be applied to the spine (MULLER, O'SHAUGHNESSY, CHUCKERBUTTY).

The *extract* of *calabar bean* is also worthy of note—one grain by the stomach, or one-third of a grain, mixed with x or xv minims of water, and neutralized by carbonate of soda, by sub-cutaneous injection, repeated in two hours, and increasing the dose gradually.

The more or less obstinate constipation of the bowels which characterizes this disease, and which is accompanied by a fœtid state of the dejections, ought to be overcome by (1.) turpentine enemata, and (2.) calomel and jalap purgatives. This is most important.

HYDROPHOBIA.

LATIN EQ., *Hydrophobia;* FRENCH EQ., *Hydrophobie;* GERMAN EQ., *Hundswuth*—Syn., *Wasserscheu;* ITALIAN EQ., *Idrofobia.*

Definition.—*A disease peculiar to animals of the canine or feline race, the specific poison of which being implanted by them in man, or in other animals, produces a similar malady. The saliva or secretion issuing from the mouth of the diseased or rabid animal conveys the poison which inoculates rabies, either through a wound or through a thin epidermis without abraison. The period of incubation of the poison after inoculation varies from four to sixteen weeks, or even longer, before the malady becomes developed. The disease is characterized by severe constriction about the throat, spasmodic action of the diaphragm, and distress at the epigastrium: all of which are aggravated or brought about by attempts to take fluid, or by the least breath or current of air on the surface of the body, which produces, in the first instance, an effect resembling that produced upon stepping into a cold bath. Tenacious and clammy saliva issues from the mouth. Paroxysms of phrensy, or of uncontrollable impulsive violence (rabidity), supervene. The duration of the disease varies from* three *to* six *or* seven *days, the greater number of cases terminating in death on the second and fourth days from the accession of symptoms. Death is generally sudden, and unexpected at the moment.*

Pathology and Symptoms.—The saliva of the dog or other animal labouring under rabies is either the virus, or contains (as any *menstruum* would) the poisonous principle—the *animal poison* which by inoculation produces *hydrophobia* in the human body. The disease is so named, not because there is any dread of water, but because in man the most prominent symptom is an inability to swallow, or to attempt to swallow, any fluid, on account of the extreme spasms which the attempt produces.

The experiments of Hartwig have proved that the poison is of a definite character, that it may impregnate various substances, and that it retains its activity for a long period. Inoculations of the saliva of rabid animals succeeded in 23 *per cent.* of the animals operated on by him; and, according to Faber, out of *one hundred and forty-five* persons bitten by rabid animals in Würtemberg, only *twenty-eight* had hydrophobia.

Two points in the pathology of *rabies* are peculiar—namely, *first*, that a long period of latency exists in the human subject; and, *second*, that inoculation is not always followed by the development of the specific disease.

With regard to the first of these peculiarities, it is to be noticed that, although in some cases pain has been felt in the cicatrix a considerable time after the accident, and in a few a slight fever or a rapid pulse has been remarked to continue from the receipt of the injury to the outbreak of the malady, still the symptoms of the disease in man seldom show themselves sooner than the *fortieth* day after inoculation, and rarely after two years. A matured zymosis seems essential to the production of the full influence of the poison; and it may be that a double zymosis takes place, as in the case of the venereal virus, first in the part and afterwards in the system (MILLER), the result of which is either to multiply the poison or to increase its virulence.

Undoubted instances are, however, on record in which the characteristic symptoms appeared as early as the *twelfth* day (SIDEY), and on the *eighth* day (TROILLIET), who even quotes instances of their occurrence as early as the day following the injury. The duration of the period of incubation, however, is sometimes of extreme duration. It has been satisfactorily proved to extend over five and a half, six, or even nine months (BERGERON, BRANDRETH); and there is on record a large body of evidence in favour of the opinion that the incubation stage of hydrophobia may be prolonged not only over a series of months, but also of one year at least. An analysis of sixty authentic observations by Romberg has shown that the shortest interval between the introduction of the poison and the appearance of the disease is fifteen days, the longest from seven to nine months, and that the average period is from four to seven weeks. The inquiries of Drs. Hamilton and Hunter give to the majority of cases a period of incubation from *thirty* to *fifty-nine* days. In the *Transactions of the Vienna Medical Association*, a case is recorded of a period of incubation extending over two years (HASSINGER); but this is discredited both by the elder and younger Gurlt, of Berlin, whose experience in veterinary pathology has been very extensive.

In all such extremely long periods of incubation the question may be asked, whether the disease has been actually inoculated at a period so far back, or has there occurred a re-inoculation at some intervening period? It is known that the dog in the early stage of the disease has a disposition to lick the hands, face, or other exposed parts of persons, and especially of those with whom it is familiar; and there are cases on record where the disease has been implanted in this way. Mr. Lawrence mentions the following:—" A lady had a French poodle, of which she was very fond, and which she was in the habit of allowing to lick her face. She had a small pimple on her chin, of which she had rubbed off the top; and, allowing the dog to indulge in his usual caresses, he licked this pimple, of which the surface was exposed. Thus she acquired hydrophobia, of which she died." While this example teaches us that hydrophobia may be implanted without a bite being inflicted, in this almost unconscious manner, it ought to deter us from permitting such indulgences to a dog. The greatest anxiety and misery have frequently been experienced for many months by those who have been thus imprudent, owing to the circumstance of rabies having subsequently appeared in the animal so indulged (COPLAND). There are instances, however, recorded of very long periods of incubation after a bite, where subsequent inoculation, independent of a bite, could not have taken place. For example, there is a case published by Mr. Hale Thomson, in vol. i. of the *Lancet*. The subject of it, a lad aged eighteen, had been twenty-five months in close confinement in prison, and during that time had never been exposed to the bite of any animal. He had been bitten severely by a dog seven years before in the right hip, and the scar still remained. During the whole period he was under observation he was sullen, gloomy, and reserved, and was never known to look the person in the face to whom he spoke. Death occurred after a three days' illness, during which "the most decided symptoms of hydrophobia were manifested." On the 15th of May, 1854, a case was admitted into Guy's Hospital, under the care of Dr. Hughes, in which hydrophobia appeared to have been developed five years after the bite (*Med. Times*, 1854).

Such observations render it extremely probable that the period of incubation of the specific poison of hydrophobia is not yet defined; and the circumstances which, in man especially, seem to shorten the duration of this period, or prolong it, are in a great measure quite unknown. The shortest period, according to Niemeyer, is about *eight* to *ten days;* the longest *twelve* or *thirteen months;* in the majority of instances, about *forty days* after the reception of the bite.

The reasons for the great inequality in the periods of incubation are obscure; but there are some circumstances, however, which seem to show that during the long interval of apparent latency the quantity or the virulence of the implanted poison seems to increase, locally at least, if not also more extensively in the system.

First, In some instances there are evidences of a slow and silent change going on in the constitution, indicated by sallow looks, sunken eyes, a pulse somewhat accelerated, more easily excited and weaker, combined with symptoms of general debility (COPLAND).

Second, The observations of Dr. Marochetti, who visited the Ukraine in 1820, and who maintained that in that country characteristic pustules were observed to form beneath the tongue, near the orifices of the submaxillary glands, between the third and ninth day after the infliction of the bite. This observation was confirmed by M. Magistel, at Boulay, in France, in 1822, who noticed that the pustules formed from the *sixth* till the *thirty-second* day. He observed two forms of pustules, a crystalline and an opaque, the latter of which, when opened, left a small ulcerated cavity. They were situated on the sides of the *frœnum linguœ*, and on the lateral parts of the inferior surface of the tongue.

Third, Changes which take place in the cicatrix before the development of characteristic symptoms, indicate that the implanted poison there undergoes some process, the nature of which is as yet not known.

After the local incubation of the poison is complete, its specific action appears to be exercised upon the *medulla oblongata* and the *eighth pair of nerves*, and subsequently lesions of the structures supplied by the branches of the *eighth pair*. The action of the poison appears in the first instance to be made distinctly manifest by the œsophageal branch of the *eighth pair*, producing that derangement of function which gives rise to the characteristic symptom of the disease, or to the extreme difficulty of swallowing, especially of fluids; while the spasmodic catching of the breath, consequent even on touching the lips with any liquid, proves that the recurrent nerve is equally affected. Subsequently the eye and ear become distressed by every ray of light or impulse of sound, and likewise the sense of touch is most painfully excited on the slightest breath of air passing over the surface of the body, all of which distinctly show that the central and spinal nerves must be functionally affected. In a still more advanced stage the suspicion, the irritability, the violence, and generally the outrageous and uncontrollable derangement of mind which often seizes the patient, bringing on epilepsy and convulsions, show that the brain itself is likewise a principal seat of the action of this terrible poison, especially the region of the *medulla oblongata.* The effects of the hydrophobic poison are often so violent in the first instance as to cause the early death of the patient; and the bodies of many persons having been examined who had so died, not a trace of inflammation or other morbid phenomena were discovered. More commonly, however, some structural alterations have been found, limited to slight inflammation of the brain, the spinal cord, or of their membranes, and of the lungs, stomach, or structures supplied by the *eighth pair* of nerves. Still, the brain, the lungs, or the stomach may be either separately or conjointly affected—phenomena in no degree dissimilar to what have been observed in hooping-cough, where the poison seems to act chiefly on the vagus nerve.

The organic lesions which have been found after death in cases of hydrophobia are as follow:—

When the membranes of the brain have been found diseased, the appearances have been, great congestion, especially of the *plexus choroïdes,* also effusion of serum, sometimes muddy, into the arachnoid cavity, and into the ventricles. In an interesting case recorded by Dr. R. W. Cunningham, of Her Majesty's 4th Bengal Europeans, the layers of the arachnoid were found adherent in many places, especially along both sides of the longitudinal sinus. The adhesions were quite soft and recent, and flakes of coagulated fibrine floated in the fluid. The brain has, in some very few cases, been supposed to be harder or softer than usual, and to have more bloody points than in health. There has been no lesion noticed in the brain, however, that could be directly connected with the malady.

Changes in the *medulla oblongata* and the *spinal cord* have not yet received sufficient attention. In the case just referred to, related by Dr. Cunningham, there was a reddish spot in the substance of the *pons Varolii,* having the appearance of inflammatory softening. On the lower surface of the *medulla oblongata,* at the origin of the *seventh, eighth,* and *ninth pair of nerves,* the membranes were highly vascular, thickened, softened, and matted together; but the substance of the nerves at their exit, and of the *medulla,* seemed normal. There are strong reasons for believing that changes actually exist in these parts which escape the detection of our unaided senses, but which the specific gravity test, combined with microscopic examination, may yet demonstrate. The mucous membrane of the pharynx and œsophagus have been seen either greatly congested or diffusely inflamed, as also that of the stomach, and of the trachea and bronchia. The latter have been found covered with a considerable quantity of frothy mucus, while the pulmonary tissue has shown marks of inflammation, though more commonly only of great congestion. The salivary glands have likewise occasionally been observed increased in size, and vascular. In a case of hydrophobia which I had an opportunity of dissecting at Renfrew, near Glasgow, the most prominent morbid change was visible in the greatly increased vascularity of the lungs, and of the mucous membrane of the back part of the mouth, pharynx, and larynx, as far as the vocal cords. The whole of these parts were covered by a tenacious frothy mucus, tinged with blood. The glands surrounding the papillæ over the back part of the tongue were very much enlarged, not unlike what I have observed in severe cases of *cholera.* So also were the submucous glands of the pharynx, the epiglottis, and the larynx, even in its cavity, and of those beneath the tongue. Inflammatory appearances in these parts have been observed by Morgagni, Babington, Watt, Portal, Troilliet, Copland, and others.

Symptoms.—The wound inflicted by the bite, whether neglected or dressed, generally heals up kindly, leaving a cicatrix, and for a time the patient usually suffers no other derangement of health than the depression of spirits which his apprehensions are calculated to excite. A few weeks or a few months having elapsed, the latency of the poison terminates, and the disease is formed. The course of the affection is usually divided into three stages, the first stage comprising the symptoms which precede the difficulty of swallowing; the second commences with the difficulty of swallowing, and terminates with the overthrow of the mind; the last stage embraces all the concluding phenomena.

The first stage commences in a few instances by the patient's attention being aroused by a numbness extending towards the sensorium from the injured part (which, if an extremity, may become tremulous); or pain is felt in the cicatrix, sometimes severe and sometimes trifling, and which shoots up the bitten limb, following in general the course of the nerve towards the trunk. It shoots as if towards the heart, but there is no evidence of lymphatic absorption. Pain, however, is by no means constant, and is for the most part absent. In the latter case the first symptom is chilliness, with headache, or a slight attack of fever, and the patient is more excited or depressed than usual. These premonitory warnings last but a few hours, or at most a few days, when the fatal but characteristic symptom, "the difficulty and dread of swallowing"—a symptom which distinguishes this malady from all others—appears, and the hydrophobic stage commences.

The second or hydrophobic stage is ushered in with a great difficulty, if not an utter impossibility, of swallowing any liquid—a symptom which generally comes on suddenly; and such horrible sensations accompany the effort, that whatever afterwards even recalls the idea of a fluid excites violent agitation and aversion.

The muscles of *deglutition* seem to be specially affected, while those of *mastication* are not so. The symptoms point to special implication of the *eighth pair of nerves.* "The distressing thirst, accompanied by the dread of making the effort to satisfy it; the wild and wandering expression of the countenance; the suffused eye; and, beyond all, the helpless, purposeless, unremitting restlessness of this disease, suggestive of the undefined apprehension of something more terrible than death itself, under which the senses reel and the intellect staggers,—distinguish *rabies* from *tetanus* and all other convulsive affections, and mark it as the most dreadful of diseases to suffer or to witness" (LE GROS CLARK). "To command the hydrophobic patient to swallow is to tell him to strangle himself" (MEAD).

Some patients who have been able to give some account of themselves describe the hydrophobic sensation as a rising of the stomach which obstructs the passage; others, as a feeling of suffocation or a sense of choking, which renders every attempt to pass liquids over the root of the tongue not only impossible, but which excites convulsive action in the muscles of the larynx, pharynx, and abdomen. In this state, says Dr. John Hunter, "the patient finds some relief from running or walking, which shows that the lungs are not yet the seat of any great oppression."

The hydrophobia, or inability to swallow fluids, is shortly accompanied by an increased flow of saliva, termed the "hydrophobic slaver." This secretion, as the disease advances, is not only copious but viscid, so that it adheres to the throat, and causes incessant spitting; and the quantity expectorated may be taken as the measure of the violence of the disease. The ejection of the saliva from the mouth, as it forms, is mainly owing to the dread of attempting to swallow it, causing the spasmodic paroxysms of suffocation. By some this increased flow of saliva is considered as an effort of the system to eliminate the poison through these excretory glands; and therefore mercury, in large doses, to promote salivation, has been recommended to promote elimination in this way, and to reduce the extreme excitability of the nervous system (LIGGET), but with no such result.

The aversion to fluids is no sooner established than another series of symptoms of dreadful severity, or a highly exalted state of every corporeal sense, is added. Indeed, it is hardly possible to depict the sufferings of the patient from this cause; for not only does he shrink at the slightest breath that blows over him, but the passage of a fly, the motion of the bed-curtain, or any attempt to touch him, produces indescribable agony, almost amounting to convulsions. Dr. Elliotson states that the effect produced by these causes very much resembles that produced upon stepping into a cold bath. The sense of sight is no less a source of terror than that of touch, for the approach of a candle, the reflection from a mirror or other polished surface, occasions the same distressing effect. The hearing is as strongly affected as the other senses, so that the least noise, and especially that of pouring out fluids, throws him into a fearful paroxysm. An attendant who sat up with a hydrophobic boy made water within his hearing, which threw the sufferer into a most violent agitation. The degree to which this painful state of the senses arrives may be understood when it is stated that Majendie records the case of a deaf and dumb child who heard distinctly in this stage of the disease. The patient, thus incessantly harassed and pained by every circumstance around him, becomes peevish and irritable, and at length sees his family, relations, and strangers, with feelings of dislike and aversion, and sometimes apparently with horror.

The third stage commences by the cerebral functions becoming disturbed, the mind being either filled with dreadful apprehensions, or being so completely overthrown that paroxysms of uncontrollable impulsive violence follow. A rabid impulse overtakes the patient to tear in pieces who and whatever opposes him. This rabid impulse greatly distresses him; and it is often strongest against those to whom he is most attached, although he struggles to suppress it. In this stage horror is strongly depicted on the countenance; every symptom is aggravated, the saliva grows thick and ropy, while the poor sufferer, not daring to make the slightest attempt to swallow, spits it out incessantly, oftentimes with frequent retchings and vomiting. In this state he sometimes turns black in the face, falling into convulsions, in which he expires; or, exhausted by his great efforts, a sudden calm ensues, and, as if nature gave up the struggle, he dies without a groan.

Remote Cause.—Hydrophobia originates in animals of the canine and feline races, as the dog, the fox, the wolf, the jackal, and the cat, as a specific inoculable disease, but from what peculiar source is altogether undetermined. It is probably at all times to a certain extent endemic, and occasionally epidemic among these animals. It has been supposed that it is excited in them by the great heat of the dog-days, or by the *œstus veneris;* but Troilliet has shown that canine madness occurs with nearly equal frequency in winter, spring, summer, and autumn. The poison is not peculiar to any country. Rabies is found equally in Europe, Asia, and America. Neither is it limited to climate. It prevails in the frozen regions of Canada, as well as in the East and West Indies. The difficulties attending any explanation of the origin of this poison are at present not to be surmounted; but hydrophobia once originated in the animals that have been mentioned, they have the power of reproducing it by their bite, not only in each other, but probably in all warm-blooded animals, certainly in all domesticated animals, as the horse, the elephant, the sheep, the ox, even in the common fowl, and in man. It will be necessary to the proper understanding of hydrophobia to give a short outline of the disease as it occurs in the dog, so constantly associated with us in domestic life, and the principal source of the disease in the human subject.

The symptoms of this formidable affection, as witnessed in the dog, are some singular departure from his ordinary habits, such as picking straws or small bits of paper off the floor, and swallowing them; licking the *noses* of other dogs, or other cold surfaces, such as stones or iron. Besides this, he is observed to be more lonely, shy, and irritable; his voice is so changed that his bark would not be recognized by those who have known his voice before; and he is less eager for his food, or refuses it altogether. His ears and his tail droop; his look is suspicious and haggard; and sometimes, from the very commencement, there is a redness and watering of the eyes. In a short time saliva begins to flow from his mouth, he "slavers," his fauces may be seen to be inflamed, and he is feverish. The animal, though highly irritable and easily provoked, still obeys the voice of his master, and it is remarkable "that the dread of fluids and even the sight of them—so striking a feature in man—is often wanting in dogs and other amimals, for many dogs lap water during the disease" (Youatt). In many dogs the symptoms never rise higher than these; but in others there is a repugnance to control, and a readiness to be aroused to extreme rage on the appearance of a stick, whip, or other instrument of punishment, or on any attempt at intimidation, which strikingly characterizes the disease. In this state, however, he seldom fights a determined battle, but bites and runs away; still even this mitigated irascibility usually ends in indiscriminate aggression, till at length he dies, apparently of convulsions or asthenia, or from mere nervous excitement and functional derangement. Majendie has inspected the hydrophobic dog, and found no characteristic morbid change. In all cases, however, in which the poison has had time to set up its specific actions, the principal lesions of structure are found to be in those parts supplied partially or entirely by the eighth pair of nerves. The tongue is swollen; the fauces, the salivary glands, and the mucous membrane at the back of the larynx behind the epiglottis, are more or less inflamed. The bronchial membrane is also occasionally inflamed, and so is the mucous membrane of the stomach, which generally contains a strange mixture of straw, hair, paper, hay, horse-dung, and earth, showing the peculiar morbid propensity of the animal; or, being void of those substances, it contains a fluid resembling the deepest-coloured chocolate. Such are the symptoms and phenomena of hydrophobia in the dog, the chief source, perhaps, of this fatal malady to the human race.

The susceptibility of the human subject to this poison is by no means universal, for only ninety-four persons are known to have died out of one hundred and fifty-three bitten, making the chances of escape as three to two nearly. It has been thought this occasional immunity does not arise out of any want of susceptibility to the action of the poison, but from the person being bitten through his clothes, and the dog's tooth, consequently, having been wiped clean from all venom. Menières, however, says he met with seven cases in which the dog must have bitten through several folds, and yet they all proved fatal; showing, as he imagines, the little importance of dress as a protection from this malady.

Neither age nor sex is exempted from hydrophobia; but no instance is known of any person being affected with hydrophobia unless antecedently bitten by a rabid animal capable of communicating the disease.

It is a question of much moment whether the saliva of a patient labouring under hydrophobia will or will not communicate the disease. It may be stated as an undeniable fact that, during the many years hydrophobia has been studied, no instance is known of its having been communicated from one human being to another, although many instances have occurred of the attendants having been bitten, or otherwise accidentally inoculated with the saliva of the hydrophobic patient. The only circumstance which makes this statement at all questionable is, that Majendie and Breschet inoculated two dogs with saliva taken from a diseased patient, shortly before his death from rabies, and that one dog shortly afterwards died of hydrophobia. Persons have also been seized with rabies in consequence of having wiped their lips with napkins or cloths, or other articles, which were soiled with the saliva (ENAUX, CHAUSSIER, and AURELIANUS).

The dog's tooth generally implants the poison, or at least some abrasion appears to be necessary, either of the cutaneous or mucous surfaces. The ancients were aware of this, for Celsus observes that the integrity of the lining membrane of the mouth is necessary to the operation of the Psylli, whose office it was to suck out the poison after the bite of a rabid dog; and Dioscorides expressly orders them first to wash their mouths with astringent wine, and afterwards to lubricate the cavity with oil. With regard to dogs, Meynill observes that "such of them as have been thought to become affected merely by the contagion of the same kennel will generally be found, upon minute examination, to exhibit the marks of bites, though concealed by the hair." When a scratch or other abrasion exists, a rabid dog merely licking the part is sufficient to implant the poison of rabies.

Diagnosis.—When hydrophobia is fully formed there is no disease with which it can be confounded; but there are many reported cases in which the imagination of a patient bitten by a dog has been so powerful as to induce symptoms resembling the disease. In hysteria the difficulty of swallowing exists, but no other symptom. Tetanus is the disease with which rabies is most apt to be confounded; yet the differences are sufficiently marked. The spasm of the muscles is more continued in tetanus; less remitting, and never intermitting. The jaw is usually much in motion in hydrophobia, in frequent attempts to clear the mouth and throat from the peculiar tenacious mucus; in tetanus it is fixed. Tetanus is rarely attended with aversion to liquids; on the contrary, the bath is grateful; nor are the tetanic paroxysms increased by the sight, hearing, or touch of fluids. Also, tetanus makes its accession usually at a much earlier period after infliction of the injury. Physiologically, while tetanus is a disease of the true spinal system, hydrophobia involves the brain also, as evinced by the disorder of intellectual function and special sense, even early in the disease. Further, the two diseases differ greatly in their mode of induction. Tetanus, in the traumatic cases, is caused by irritation of a nerve, and by disease of the spinal marrow in those which are idiopathic. Hydrophobia is the result of a specific poison introduced into the circulation, and thence affecting the nervous system as a poison would (MILLER). While in tetanus the stimulus which excites the paroxysms "operates through the true spinal cord, in hydrophobia it is often conducted from the ganglia of special sense, or even from the brain, so that the sight or sound of fluids, or even the idea of them, occasions, equally with their contact, or with that of a current of air, the most distressing convulsions" (CARPENTER).

Prognosis.—There are few instances of any patient or animal suffering from this disease having recovered.

Treatment.—As there are but very few authenticated cases of recovery from hydrophobia, so there are few instances of any mitigation of the symptoms by the use of medicine. All that remains is to mention the most leading experiments that have been made, with the hope that, as they have not been successful, they may not be wantonly repeated.

Dr. Hamilton gives twenty-one cases, and adds—"many hundreds more are on record," in which *venesection* has been unsuccessful, though frequent and copious. *Opium* has been given by Dr. Babington, to the enormous amount of 180 grains of solid opium in eleven hours, without the slightest narcotic effect, or the slightest mitigation of the symptoms. Nord has given a drachm of *belladonna* in twelve hours, without any benefit. Dr. Atterly gave to a child eight years old two drachms of *calomel* by the mouth, and rubbed in two ounces and a half of strong mercurial ointment in a few hours, with an equal want of success. A case, however, is related by Ligget, which is said to have been successfully treated by half-drachm doses of *calomel*, given to the extent of ptyalism, induced in three days, after four and a half drachms of calomel had been taken. The case really appears to have been one of hydrophobia; and recovery is said to have been complete by the twelfth day (*Amer. Quar. Journal of Med. Science*, Jan., 1860). *Iron, arsenic, nitrate of silver, camphor, musk, cantharides, turpentine, tobacco, acetate of lead, ammoniacal solutions of copper, hydrocianic acid, galvanism, strychnine, nitrous oxide, chlorine, and guaiacum*, have all been given in equally large doses, but have signally failed. These include some of the most powerful medicines in the Pharmacopœia; and in addition to these, Ploucquet, in his *Literatura Medica Digesta*, has enumerated nearly 150 others.

The failure of every remedy by the mouth, and the inefficacy of *opium*, of *morphine*, and of *laurel water*, even when injected into the veins, so convinced Majendie that in hydrophobia the constitution was armed against the action of any medicinal substance, that on a patient labouring under this disease being brought to the Hôtel Dieu, he determined to rely for all treatment on an *injection of warm water into the veins.* The patient, at the time of the operation, is represented as being absolutely insane, so as to require to be restrained. In this state, and with a pulse of 150, Majendie injected into his veins, in the course of two hours and a quarter, two pints of water, at the temperature of 100°. At the conclusion of this operation the pulse had fallen to 80, and the patient recovered his senses, so that restraint was no longer necessary. The sequel, however, renders it doubtful whether this mitigation was desirable, at the price of the intense suffering which followed. The poor man lived eight days afterwards, but the despondency and mental agitation quickly returned, and at the end of three days the poison (or the state of the blood induced by it and the warm water) appeared to set up a new series of actions on the synovial membranes of the wrists, elbows, and knees, attended with excessive pain, so that he was unable to bear the weight of the bed-clothes, and he died in great torture. The articulations thus affected were found, on *post-mortem* examination, to be greatly inflamed, and their cavities filled with pus. This case is remarkable as being the one in which life was prolonged for the greatest period of time recorded of this disease. The experiment has since been repeated by Gaspard and others; but the mitigation, if any, has been so slight and transient as to give no encouragement for repeating it; and, tried on the rabid dog by Youatt and Mayo, it proved eminently unsuccessful.

The property which some animal poisons have of controlling and of interrupting the actions of other morbid poisons on the constitution has caused even animal poisons to be tried in the cure of this disease. The rapid and powerfully acting poison of the viper led to the hope that the bite of that reptile might prove an antidote to the hydrophobic virus; but the experiment, tried in France, Germany, and Italy, upon animals, has been entirely unsuccessful. M. Grindard conceived that the vaccine virus might influence hydrophobia, and he vaccinated a hydrophobic child in three places, and afterwards injected five charges of vaccine lymph into the veins; but the child died without any marked remission, and in the usual time. The following draught has been found rather to promote euthanasia than to hold out any prospect of cure:—

℞. Spirit. Æther. Sulph., Tinct. Opii, ā ā ♏xx.; Spirit. Ammon. Aromat., ʒss.; Chloroform, ♏xx.; Mist. Camph. ℥iss.; *misce.* To be given as often as may be *considered safe* (Cunningham, Carden).

On the same principle chlorodyne may to be given. The vapour bath is sometimes useful in moderating spasm.

Preventive Treatment.—The probabilities are, that unless the operation of excision, or cauterization, be performed within a few minutes after the bite of the rabid animal, it is impossible to save the patient from the fatal disease, which, according to the susceptibility of his constitution, may threaten him at any moment. In all probability no prophylactic medicine exists in nature, and the administration of any potent substance by way of prevention is worse than useless; for, without protecting the patient, it may injure his constitution. Mild remedies, if they tend to tranquillize his mind and appease his apprehensions, may be innocently employed.

The theory which maintains that a zymotic incubation first takes place in the wound, by which the poison is originally implanted, suggests the most rational prophylactic—namely, to destroy entirely by *potassa fusa* the whole cicatrix, where practicable, or by some other surgical means entirely to remove it, at as early a period as possible, and *previous* to the occurrence of symptoms. When premonitory symptoms are first observed, the following plan has the recommendation of Dr. Maxwell in *The Indian Journal of Medical and Physical Science,* and of Dr. Copland, namely,—(1.) That the original cicatrix be freely laid open, and suppuration from it speedily and freely produced and maintained for several months. (2.) The nerves, or nerve, leading to the part are to be divided without delay, the more remote from the wound the better. (3.) Free perspiration should be promoted by the hot air bath. (4.) Bleeding from the arm to syncope in robust persons with sthenic symptoms, or cuppings on the nape of the neck, are modes of practice indicated by the lesions found after death.

INFANTILE CONVULSIONS.

Latin Eq., *Membrorum distentio infantalis;* French Eq., *Convulsions de l'enfance;* German Eq., *Kinderkrämpfe;* Italian Eq., *Convulsioni de 'bambini.*

Definition.—*Convulsive seizures, consisting of contraction of muscles by gradual but rapid shortening of the muscular fibres, causing such hardness and stiffness of the muscle or limb that it cannot be overcome. This period of tonic contraction is followed by clonic spasmodic phenomena, characterized by* *the occurrence of alternating movements of contraction and relaxation, independent of the will, which is as powerless to suspend or moderate them as to excite them* (TROUSSEAU).

Pathology.—Convulsions may occur from the time of birth until the end of the seventh or eighth year, and arise from the following causes:—

(1.) From manifest anatomical lesions of the nervous system, especially during the progress of acute diseases of the brain or spinal marrow, and which are analogous to forms of epilepsy arising from cerebral tumors and other chronic disorders of the brain.

(2.) As the expression of many very different diseases, or the premonitory beginning of them; for example—

(*a.*) Of *Epilepsy.*

(*b.*) *Hysteria.*

(*c.*) *Chorea.*

(*d.*) *Tetanus.*

(*e.*) Blood-poisoning, as of uræmia and of narcotic poisons.

(*f.*) Material of contagion of certain diseases, and which by convulsions usher in the attack as of *scarlet fever, measles,* or *small-pox.*

(*g.*) The high fever-heat of such diseases as pneumonia and other inflammatory diseases of childhood.

(*h.*) The result of terror and other mental emotions, causing excitement of the cerebral ganglia, transmitted thence to the medulla oblongata.

(*i.*) Irritation of peripheral nerves from teething, intestinal worms, indigestion, or painful injuries of the skin, like burns, or from application of blisters, or sinapisms, or painful eruptions, which, being transmitted to the medulla oblongata, induce that morbidly irritable state which is expressed by threatening "reflex convulsions."

(3.) Convulsions occur as the expression of a *hereditary predisposition.* This nervous susceptibility manifests itself in different generations, in the same or in different ways. It commonly happens that parents—mothers especially—who in their infancy were subject to fits, give birth to individuals who, in their turn, are affected in the same way (TROUSSEAU). Niemeyer and Duclos also bear testimony to the fact, that infantile convulsions are often congenital, all the children of a family being sometimes affected in the same way.

(4.) Convulsions also occur as the result of *insufficient feeding,* or of bad food; or in those who have lost large quantities of blood from *spontaneous hæmorrhage, venesection,* or *leeches,* or who had profuse *diarrhœa* persisting for a long time. In proportion as the nutritive and vegetative functions are feeble and languishing, so will nervous phenomena be mobile, exalted, and irregular. The dependence of the normal state of the nervous system on the blood and the nutritive functions being mutual, is most strikingly marked in children, confirming the law enunciated by Hippocrates—*sanguis moderator nervorum* (TROUSSEAU).

(5.) As a consequence of albuminuria—as an acute affection—as a result of scarlet fever, or of Bright's disease.

(6.) Traumatic injury—such as penetration of the brain by fine needles. Examples of this occurrence are cited by Trousseau and Underwood, where, on removal of the child's cap, a bit of coloured thread twisted on the scalp being laid hold of, a long needle to which it was attached was pulled out.

In the *acute exanthemata* and in *pneumonia*, convulsions in children often seem to take the place of the chill or rigor, which is the precursory sign in older people of the commencement of such acute diseases; and which in reality is a convulsion of a small degree due to alternate contraction and relaxation of muscles.

Morbid Anatomy discloses many morbid conditions of a very different nature, associated with fatal cases of convulsions; but the cases may be broadly classed into—(1.) Those which arise from manifest anatomical lesions of the nervous system; and, (2.) Those which seem to be caused by no material change, or at least where the most inquisitive examination after death fails to reveal the existence of any organic lesion to which the convulsions may be ascribed. To the convulsions associated with the first of these two classes the name of *symptomatic* has been given; to those of the second class, the name of *idiopathic convulsions,* as well as *eclampsia,* have been assigned.

But although no anatomical change can be demonstrated, it is not to be at once inferred that they are independent of any material affection seated in the nervous system. For the most part the lesions that have been found in the nervous centres in these and similar diseases are for the most part secondary—the result or consequences of the disturbances in the nervous system. Morgagni expressed this belief long ago, when he wrote that "the cause of convulsions, which consists in an invisible change that has occurred in the brain and nerves, cannot be detected by our senses after death. Its effects alone are seen, and these vary according to the violence and duration of the convulsions." The more often, therefore, convulsions take place, the more liable will the child be to the occurrence of permanent and visible lesion of the nervous system as a result of them. Their repeated recurrences tend to weaken the Intellect and impair the general health.

It is the *idiopathic convulsions*—*eclampsia*—those which are connected with well defined physiological conditions, which ought to be considered under the head of "Infantile Convulsions." Those which are symptomatic, such as the convulsions of *cerebro-meningitis,* or of other diseases, form part of the history of the particular disease of which they are one of the expressions. When, however, they are the result of the existence of those diseases, and come in at or toward their termination, instead of at the commencement, then they properly belong to this class—that of *eclampsia,* the particular disease being then regarded as their exciting cause.

Circumstances apparently the most insignificant may bring about convulsions in children *pre*disposed to them; so that there are children who are liable to be convulsed with as much facility as other children will sleep, dream, or become delirious. This predisposition is chiefly hereditary.

The rigidity of *tonic contraction,* for however short a time, always precedes the *clonic spasms.* These may be very violent, while the preliminary contraction may be very slight and transient. Hence, the tonic contraction is apt to be overlooked, or lost sight of. In other cases the *tonic contraction* alone constitutes the convulsion. A period of rigidity is the essential element of all convulsions. It is never absent, and may alone be present. *Collapse, stupor,* or *coma* may also occur, but they are no necessary part of the seizure, but the effect of the convulsions.

Symptoms.—Infantile convulsions may come on like an epileptic fit in the adult, suddenly, and with no premonitory warning to indicate the invasion of the disease. They are best described by Trousseau, as follows:—"The child may utter a cry, lose consciousness, become rigid as a deal board, and ultimately commence to struggle with a fixed chest and suspended respiration. The face, pale at first, becomes red and livid, and the eyes fill with tears, which run over the cheeks, and the veins of the neck are turgid, and project like knotted cords. The *clonic spasms* then set in, characterized by disorderly and involuntary contractions of many muscles,—the limbs are alternately flexed and extended; the fingers and toes successively bent and stretched out, separated from or approximated to one another, but most frequently in a state of forcible flexion; the thumb is adducted and hidden by the fingers. The head is drawn backwards or is bent forwards; and sometimes it is pulled laterally by irregular and jerking rotatory movements. The muscles of the face share in the *general convulsions;* the eyes are the seat of jerking movements, and roll in their sockets; they are generally drawn up under the upper eyelid; more rarely they are pulled downwards, and there is *strabismus convergens.* The labial commisures are dragged upwards and outwards,—hence the distorted face is sometimes frightful to beholders; and then, on each convulsive shock, the air passes through the kind of funnel formed by the corners of the half-opened buccal orifice, making a suction noise, accompanied by a flow of frothy, and sometimes of bloody saliva. The tongue is apt to be protruded, and may be bitten, and so lacerated by the teeth. As the muscles of the trunk are affected during the tonic stage, the inspiratory muscles are fixed, and the larynx spasmodically contracted, no longer permits the free passage of air. Contraction and convulsion of the abdominal muscles cause expulsion of urine and of fæces. The *clonic spasms* are at first rapid and limited, but become slower and more extensive, till at last a deep inspiration, followed by complete relaxation, announces the end of the fit. The child is then apt to fall into a state of somnolency and stupor. Such a fit may last one or two minutes, and may be repeated over and over again during half a day, a whole day, or more days, even to eighteen days, in one case, during hooping-cough."

A repetition of fits may be anticipated, when the sleep is not sound, after the first fit has subsided, and when the child throws itself about, gnashes and grinds its teeth, and twitches its limbs from time to time.

There are also localized convulsions to be recognized, such as *partial convulsions* of one-half of the body, of the trunk of the body, of the muscles of the face, of the muscles of the eye.

Another form of partial convulsion consists of partial spasms of the pharynx, larynx, and muscles of respiration, especially accompanied by convulsions of the globes of the eyes, to which the vague names of "*inward fits,*" "*inward convulsions,*" or "*inward spasms,*" have been given.

Children a few days after birth are apt to suffer from slight convulsions of this kind, expressed by the rolling about of the eyes during sleep—the eye-balls turned upwards with the corneæ under the upper lids, gentle moaning, difficult breathing, twitches of the muscles of the face, flushing, or lividity of the face. It is to those phenomena that nurses have given the name of "inward fits."

Of all the causes mentioned in connection with convulsions, none are so common or universal as *indigestion.* Hence the quantity and quality of the food and condition of the evacuations ought to be inquired into at the first.

Prognosis.—If the tonic stiffness or spasms continue affecting the trunk, the sudden arrest and complete suspension of the respiratory functions which it entails may be fatal in due course of time; so also "*inward fits*" implicating the larynx.

Death by asphyxia is the usual mode of fatal termination, or some times by syncope.

Convulsions which take the place of chill, occurring at the outset of diseases, are not generally attended with danger; but those which occur during the course of general diseases, such as eruptive fevers, enteric fever, pulmonary or intestinal inflammation, and the like, and especially towards their close, are significant of great danger, arising from grave complication in the condition of the patient. Convulsions under such circumstances generally usher in death. They are more particularly liable to occur thus in cases of scarlet fever, in the third stage, associated with commencing albuminuria.

Generally speaking, convulsions are less dangerous in proportion as they are more easily excited. Danger is great in attacks during the first months of life; in older children prognosis is more favourable.

The frequency and fatality of convulsions diminish as the development and organization of the nervous system becomes more perfect as age advances.

Treatment.—In most cases it ought to be expectant to this extent, that the clothing must be loosened completely, so as to admit air freely, and *anti-spasmodics,* such as *ether* alone, or combined with *musk* or *belladonna,* may be given, till the progress, duration, seat, or probable cause of the convulsions may be ascertained. An *emetic,* a *purgative enema,* a *searching purgative,* such as *calomel,* combined with *jalap* or *rhubarb,* may be indicated; or the removal of a pin in the dress, which had been long pricking the skin, or too tight a dress, may, on removal or rectification, cause the convulsions to cease.

A clyster of one part vinegar and three parts water, the application of cold compresses to the head, or of leeches behind the ear, if the convulsions do not subside, are remedies advocated by Niemeyer.

If symptoms denote stupor, cold affusion should be prescribed; if, on the other hand, they denote some collapse, *wine, camphor, musk,* and other *stimulants* must be used.

If indigestion is traced as the source of the fits in an infant or young child, a few drops of *aromatic spirit of ammonia,* or the same quantity of *ether,* may be given in a tea-spoonful of water, or a drop of *anise oil,* rubbed up with sugar, may be given from time to time, by laying the powdered sugar so saturated on the tongue of the child.

If dentition is causing the convulsions, the dense tissue of the gums over the tooth ought to be relieved by lancing it, or cutting it across over the crown of the tooth.

Dr. Tanner advises the administration of *bromide of potassium* as a medicine from which more may be hoped for than any other. If there be inability to swallow, it ought to be given in *enemata* of *beef-tea.*

Where great restlessness prevails, *hydrocyamic acid,* with *tincture of hyoscyamus,* may be given.

Compression of the carotid arteries and chloroform inhalation, or a mixture of ether and chloroform, are only to be had recourse to in cases of prolonged duration, depending on such irritation as that of dentition. Anæsthesia thus induced is of great value.

Trousseau is strongly opposed to blisters and general "revulsions to the skin," except in some cases of "inward fits," involving the diaphragm, or the heart. In such case the action of the remedy must be immediate, as by *ammonia.*

EPILEPSY.

LATIN EQ., *Epilepsia;* FRENCH EQ., *Epilepsie;* GERMAN EQ., *Epilepsie*—Syn., *Fallende krankheit;* ITALIAN EQ., *Epilessia.*

Definition.—*A complex nervous state, in which, as a rule, a sudden and complete loss of Consciousness prevails, associated with convulsions, as if tonic at first, but which subsequently become chlonic, and ultimately impede the respiratory process.*

The attack, lasting from two to twenty minutes, is followed by some exhaustion and sleep (Petit Mal).

The expression of the epileptic state varies from the most severe paroxysm to simple vertigo, a momentary suspension of Consciousness, a fixity of gaze, a totter of step, and a confusion which appears and disappears almost instantaneously, and which only the patient can recognize.

Pathology.—This disease has been known from the earliest antiquity, and is remarkable as being that malady which, even beyond insanity, was made the foundation of the doctrine of possession by evil spirits, alike in the Jewish, Grecian, and Roman philosophy. The interest and importance which attaches to *epilepsy* cannot be better expressed than has been done by Sir Thomas Watson. He writes that it "is scarcely less terrible to witness when it occurs in its severer forms than *tetanus* or *hydrophobia;* but it is not attended with the same urgent and immediate peril to life. Yet it is, upon the whole, productive of even more distress and misery, and is liable to terminate in even worse than death;—a disease not painful probably in itself; seldom immediately fatal; often recovered from altogether; yet apt in many cases to end in fatuity or insanity; and carrying perpetual anxiety and dismay into those families which it has once visited," (Lecture xxxv.) Again, fully impressed with the responsibility entailed on the physician in the diagnosis between hysteria and epilepsy, and the necessity of its being certain and accurate in either case, he says, "It is a dreadful announcement to have to make to a father or mother that their child is epileptic," (Lect. xxxviii.)

About seven *per cent.* of the cases of nervous diseases are due to epilepsy (Reynolds).

Unlike chorea and tetanus, there is an interruption alike in consciousness and insensibility, which is as essential an element in the epileptic fit as the convulsions.

Convulsions, unconsciousness, and loss of sensibility, are the essential elements of an epileptic fit.

By clinical observation and experiment, it is presumed that the excitement of the motor nerves, of which the convulsions are the exponent, proceeds from the *medulla oblongata* and the portion of brain lying on the base of the skull. That such is the correct pathology of epilepsy is shown by Niemeyer under the four following heads :—

(1.) By the interruption of the functions of the hemispheres, which accompanies the convulsions. It is not probable that motor impulses proceed from the hemispheres at a time when the irritability of the other ganglion-cells and nerve-fibres is extinguished.

(2.) Because convulsions, similar to epileptic convulsions, can be excited by continuous excitement of the basilar portion of the brain by means of the induction apparatus, while no such result is obtained by a like irritation of the various parts of the hemispheres.

(3.) Because *Küssmaul* and *Tenner,* in their experiments upon animals, could still produce convulsions of a decidedly epileptic character after extirpation of both hemispheres.

(4.) *Schroeder van der Kolk* has found that, in all bodies of epileptics where the disease had been of long standing, besides numerous inconstant lesions, there was always a dilatation of the arterioles and capillaries of the medulla, with thickening of their walls. He believes that epileptic convulsions depend mainly upon an increased afflux of arterial blood to the medulla oblongata. There is no doubt, moreover, that the morbid irritability of the medulla which occasions epilepsy may arise without any increase or diminution of its supply of blood, merely from the improper character of its nutriment and from the admixture of certain materials in the blood. It must also be admitted that the *medulla oblongata* may be thrown into an irritated condition by the transmission of a morbid impression from remote regions of the nervous system, whether central or peripheral. It is well known that *neuromata* and *cicatrices,* or *tumors* pressing upon peripheral nerves, have sometimes occasioned epilepsy, and that the epilepsy has ceased after section of the affected nerve or after removal of the cause. Perhaps, also, cerebral tumors and other diseases of the brain and spinal marrow may induce epilepsy in a similar manner by gradual transmission of a morbid irritability to the *medulla oblongata.* This supposition has received strong support from the result of recent experiments by *Brown-Séquard,* in which dogs, whose spinal marrows had been injured, suffered from convulsions, although not immediately, but some time after the receipt of the injury.

It is difficult to say why the morbid irritability of the motor nerves is not continuous, but merely occurs in paroxysms, with intervals which frequently are of very long duration.

It has been affirmed that in fifteen out of twenty cases, in which the brains of epileptic patients have been examined, the structure of that organ has been in every respect healthy. Even when the patient has died during the paroxysm, the brain has in many instances been found congested only. Epilepsy has been therefore regarded as a functional disease, the particular seat of lesion not being determined.

But although epilepsy may exist without any obvious disease of the brain or of its membranes, it must be admitted that the brain and its membranes are occasionally found in every state of disease to which those parts are liable. Thus, the membranes may be inflamed, thickened, or ossified, and with every variety of exudation; or the substance of the brain may be indurated or softened—the seat of abscess, of cancer, of tubercle, or of other structural disease. Any such structural disease is then considered to give rise to the epileptiform attack. Dr. Sieveking, whose researches into the nature of this disease have been most prolonged and laborious, once showed me an interesting old German work by T. and C. Wenzel, in which the epileptic state was shown to have been invariably found associated with a morbid state of the *pituitary* body in the *cella turcica*—a spot of the encephalic region very rarely examined in *post-mortem* examinations. On the other hand, Niemeyer states (very laconically) that "alteration in the appendages of the brain, which Wenzel mentions as a constant lesion in epilepsy, is absent in the majority of the cases."

The tendency of modern pathology seems to be to connect the epileptic seizure with a variety of pathologico-anatomical lesions of a variable and inconstant kind, such as have been mentioned.

There are also cases referred to causes of an eccentric or peripheral nature, such as to uterine or ovarian disease, which are thus said to act upon the brain through the medium of the nervous system, in some way as yet unknown—*reflex epilepsy.*

Dr. Todd developed a theory of the disease, suggested by the occasional occurrence of epilepsy with renal affections. He held that the peculiar features of an epileptic seizure are due to the gradual accumulation of a morbid material in the blood, until it reaches such an amount as to operate upon the brain, as it were, in an explosive manner. In other words, the influence of this morbid matter, when in sufficient quantity, excites a highly polarized state of the brain, or of certain parts of it, and these discharge their nervous power upon certain other parts of the cerebro-spinal centre, in such a way as to give rise to the phenomena of the fit. This theory assumes that the essential derangement of health in epilepsy consists in the generation of a morbid matter which infects the blood, and it supposes that this morbid matter has a special affinity for the brain, or for certain parts of it, just as *strychnine* exercises a special affinity for the spinal cord. According to this theory, the disease ought to have found a nosological place amongst the *constitutional diseases.* But, to give a definite character to such a humoral theory, it were necessary to discover some morbid matter in the blood in every case of epilepsy.

"This desideratum has as yet been only partially obtained. The clue to a discovery of this kind was first given by the observations of Prevost and Dumas upon the effect of excision of the kidneys. These observers found that the removal of the kidneys always led to an accumulation of a considerable quantity of urea in the blood, followed by convulsions and coma—an epileptic state. After this, clinical observations by practical physicians showed that disease of the kidney was apt to be followed by attacks of convulsions and coma, when the excretion of urine fell in quantity to a very low amount; and it was found that, in such cases, a considerable quantity of urea was present in the blood. A connection was clearly thus established between the presence of urea in the blood, defective renal action, and the epileptic condition; but whether the active poison is urea cannot yet be decided. Frerichs, indeed, has lately affirmed that it is carbonate of ammonia, a product of the decomposition of urea. But even this is still *sub judice.* All that we really know is, that in certain states of diseased kidney, when the excretion falls below a certain point, urea will accumulate in the blood, and epileptic seizures will ensue; and, should the patient die, we find no brain-lesion to explain the phenomena; but we find unequivocal evidence of diseased kidney" ("Clinical Lectures," by Dr. R. B. Todd, *Med. Times and Gazette*, Aug. 5, 1854).

In the present state of our knowledge, therefore, and knowing that there are a great many cases in which the epileptic phenomena have recurred during a long period, and in which *post-mortem* examination reveals no lesion with which symptoms can be connected, it is then better to consider epilepsy as an intrinsic disease of the brain, because the most constant and marked groups of phenomena are referable to the functions of the central parts; while the loss of consciousness, associated with excessive mobility, leads one to regard those parts of the brain in the immediate vicinity of the *cella turcica* and basilar portion of the occipital region—for example, the *central ganglia* or *medulla oblongata*—as parts where, in future, morbid anatomy may yet discover a lesion.

On the other hand, epilepsy in very many cases must be regarded as "a disease of the whole man, and not of any one organ or system of organs alone;" and if lesions of a more or less constant kind be ever found in the brain, they would merely stand in the same relation to epilepsy that morbid states of the kidney do in relation to the whole phenomena of Bright's disease, regarded as a *constitutional disease.* Epilepsy, therefore, might with fair reason be regarded in this respect as a *constitutional disease,* with intervals of apparent freedom, and with times at which the disease culminates in the characteristic paroxysm. In order fully to appreciate the nature of epilepsy, as Dr. Sieveking justly remarks, a careful study of the general condition of each patient is necessary, and especially of the phenomena which may show themselves in the intervals of freedom from the paroxysms. Nutrition changes in epilepsy may be connected with a constitutional state of ill-health, such as gout, scrofula, or the like, the paroxysms having a remote origin in the state of the blood; and when once set up, they bring the *medulla oblongata* and *upper* part of the spinal cord into that condition of excessive and perverted functional activity which maintains the epileptic state. Arterial spasms and the consequent cerebro-spinal anæmia is believed to be the first step of this disordered movement. The pale face, the loss of consciousness, the contraction of the muscles of the chest and larynx at the commencement of the fit, are all considered to point to this cerebral condition.

The scantiness of our knowledge regarding the causes of epilepsy renders statistics of very secondary importance regarding the conditions of its development. In the words of Niemeyer, "we do not know of one single agent of which we can certainly predict that it will produce epilepsy; yet we must admit that all the assigned causes of epilepsy by themselves are incapable of producing it, and that the co-operation of the second and unknown factor is always requisite."

It is a very common disease. Six in one thousand individuals are epileptics. Attention must not be limited to the paroxysm alone.

Symptoms.—Epilepsy may be grave or slight. The attack often occurs without any previous warning; so much so that Georget estimates that in 95 cases out of 100 there are no premonitory symptoms. These warnings are known by the term "*aura*." They comprise all the premonitory symptoms which may prognosticate the approach of a fit. Dr. Sieveking has noted such "warnings" in 48 out of 104 cases—a little more than 46 *per cent.* Many patients, however, on the approach of the fit, have vertigo or headache; some swelling of the veins, or throbbing of the arteries of the head; while others again have ocular spectra, or affections of the other senses. Dr. Gregory used to mention in his lectures the case of an officer whose paroxysm was always preceded by the spectre of a little old woman dressed in a blue cloak, who issued, as he imagined, from the corner of the room, and knocked him down with her stick. Dr. Fothergill attended a Quaker who always fancied he saw his garb covered with spangles before he fell into the fit. These ocular spectra are very numerous; but the most common are flashes of light, tadpoles, flies, coloured areolæ around the flame of the candle, black dogs, and white horses. Others have hallucinations of hearing, as the ringing of bells, or the roaring of the sea; while others again are annoyed by the smell of disagreeable odours, or by the sensation of unpleasant tastes. When the sense of touch is the seat of the hallucination, the term "*aura epileptica*" is used to express it. In these cases the patient has often the sensation of a fluid creeping from the fingers or toes upwards towards the trunk; others feel as though a spider or other insect were crawling over the skin. Dr. Elliotson speaks of a patient that had two *auræ*, each of which ran along the dorsum of each foot, ascended up the front of the legs and thighs to the trunk, where they broke into five streams, all of which again met at the epigastrium, and, having reached this point, he fell into the fit. The late Dr. John Thomson, of Edinburgh, relates an instance of an epileptic "*aura*" commencing in an old cicatrix in the side; and Sir Thomas Watson mentions the warning sensation as originating in the thumb of one of his patients, which presently became twisted inwards; and he could sometimes prevent the complete expression of the fit by tying his handkerchief tightly round the throat. Esquirol relates the case of a woman, in which the *prodrome* consisted in the patient turning round for a considerable time; and another of a man, who ran with all his might, till at length he fell down, overpowered by the disease.

Although these sensations (*aura*) may be subjective, and experienced only in the skin, and not following the course of any particular nerve, yet their subjective origin may "be due to some injury done to, or some morbid impression made upon, an *afferent* nerve," as well as to some morbid condition of the brain itself.

Two forms of epileptic seizures are to be recognized, namely—

(1.) Lapses of consciousness only.

(2.) Lapses of consciousness, with the accompaniment of slight convulsive movement.

In the former—the genuine "*petit mal*"—the patient has simply become giddy (*epileptic vertigo*) often amid business or conversation. He must then either seat himself, or he staggers, or sinks slowly to the ground without any outcry. His face is pale, his eyes are fixed. A few convulsive twitches play over his face and the extremities, especially the upper ones. He recovers in a few minutes, looks wildly around him, and after the lapse of a few minutes, appears to be himself again, and able to resume his usual avocation (NIEMEYER).

A still more mild and rudimentary form of epilepsy is the *vertige epileptique*, where consciousness alone is clouded for a few seconds, the countenance pale, and the eyes fixed; but the patient does not fall. He may lose hold of anything he has in his hand at the time.

Regarding the frequency of the occurrence of individual symptoms, Dr. Sieveking met with headache in 56 out of 104 cases, or in a ratio of 53·8 *per cent.*; and the pathological import of the symptom varies much according to the period at which it is met with. It may be connected with the fit etiologically; or it may be a consequence of the attack; or it may be a casual coincidence (*On Epilepsy and Epileptiform Seizures*, second edition, p. 54).

According to Reynolds, the relative frequency of the different classes of premonitory symptoms is as follows:—*Mental and emotional*, in 11·1 *per cent.*; *sensational symptoms*, in 19·8 *per cent.*; *motorial phenomena*, in 8·6 *per cent.*; *vascular* and *secretory disturbance*, in 3·7 *per cent.*

In the *adult*, whether the warning symptoms be or be not present, the attack usually commences by the patient uttering a cry, losing on the instant all consciousness, and falling down in convulsions, his mouth covered with foam.

The more immediate or directly precursory symptom of this event is a momentary and death-like paleness of the face at the time of the fall, which is immediately followed by the flushed face (TROUSSEAU, G. JOHNSON).

The convulsions vary from the most trifling and transitory convulsive movement to the most frightful, terrific, and long-continned struggles. In mild cases only one limb is convulsed; in others only the face, the lip, or the eye. Esquirol gives the case of a lady whose fits were so slight that although often seized on horseback she never fell off. In a few seconds she recovered, and resumed the conversation by finishing the sentence she was expressing. In this case, however, the epileptic cry and the convulsed eye denoted the true nature of the attack. Another lady, advanced in life, suffered from fits so slight that she preserved her seat in the chair; and except for the occurrence of some slight convulsive motions about the mouth, followed by a short sleep, the attack would have passed unnoticed. Attacks so mild often occur many times in the day, last about five minutes, and appear for a time to leave no feeling of ill-health behind.

In severe forms of epilepsy the convulsions are more formidable; the hair stands on end, the forehead is wrinkled, and the brow is knit. If the eyelid be opened, the eye is seen to be injected, sometimes convulsively agitated, at other times in a state of strabismus, and sometimes fixed: more commonly the eyelid is quivering, and half open, so as to show the white of the lower portion of the conjunctivæ. The face is red, or livid and swollen, the teeth generally clenched, and the lips covered with foam; sometimes, however, the mouth is open and the tongue thrust forward; and should the masseter muscles now act spasmodically, it may be bitten through, or otherwise much injured, and the foam consequently be mixed with blood. The force with which the jaw closes is so great that teeth have been known to be broken and the jaw luxated. The limbs, also, are violently convulsed, thrown about in every direction, and with such power that it often requires three or four persons to prevent the patient seriously hurting himself. In these convulsions, also, the hands are strongly clenched, and the body is often arched backwards (*opisthotonos*); when, on the muscles relaxing, the patient may fall to the ground with great force. While the limbs and trunk are thus powerfully agitated, the muscles of the chest are often spasmodically fixed, so as hardly to permit the acts of respiration.

The functions of organic life are also implicated in this scene of tumult. The pulse is generally frequent, sometimes hard and intermittent, and at other times scarcely perceptible, although the heart's beats are strong and tumultuous. The respiration is stertorous, the stomach and bowels troubled with borborygmi, the skin bathed in sweat, while the urine, semen, or fæces are occasionally emitted. Blood sometimes flows from the eyes, ears, or nose, frightfully expressive of the violence of the attack.

When the paroxysm has reached its crisis the muscles relax, the convulsions subside, the respiration becomes more free, the pulse more regular, and the countenance more natural; and at length the patient falls into a heavy sleep, from which he awakes sometimes in good health, but more often shaken, exhausted, and suffering from severe headache, which lasts some hours or even days. In neither case, however, has he the slightest consciousness or remembrance of what has passed. In other instances the termination of one paroxysm is but the beginning of another, and the succession is occasionally so continued that the attack, with short intermissions, may last twenty-four or forty-eight hours, or even longer.

When *children*, from teething or other causes, are seized with epilepsy, the attack is often preceded by a spasmodic affection of the larynx, causing the whooping or crowing sound so well known to every practitioner; but it may, and often does, take place without any warning. In the former case the child perhaps is in his best health, but on awakening is seized with the characteristic whoop, often accompanied by a spasmodic flexion of the thumb against the palm; or the fingers are clenched, or the toes bent. These symptoms may recur a varied number of times, till at length, with or without this warning, the eye is seen staring, fixed, or convulsed; the face and extremities pale or livid; the hand clenched, the body rigid, and the head and trunk curved backwards. The fit is now formed; and if we examine the *fontanelle* we find it distended and pulsating. These symptoms generally last only a few minutes, when a strong expiration takes place: a fit of crying succeeds, and the child, much exhausted, recovers its Consciousness, and after a short interval generally falls asleep. These convulsions seldom occur during the early periods of lactation, nor until the commencement of the period of dentition, and rarely after three years of age.

The duration of the paroxysm in children seldom lasts more than a few minutes. In the adult it often does not exceed that period; but in many cases it lasts half an hour to two hours; while in others the greater part of the day passes before the paroxysm terminates.

It seldom happens that the paroxysm occurs but once. In the mildest case in the child it is commonly repeated three or four times in the course of the first three or four years of childhood, while in other cases it will occur three or four times in the day; and in severe cases the child is hardly out of one fit before it falls into another, till at length they gradually subside. In the adult the frequency of the fit varies extremely in different patients. In some instances there is an interval of several years; at others it returns annually, or every six months, or mensually, weekly, or even daily; while others will have twenty or thirty fits in the course of the same day. The period of the day at which the attack takes place is also very varied, for it may occur during the day, at night when asleep, or in the morning when just awakening.

Causes.—When epilepsy is the result of a powerful original tendency, it often occurs without any apparent cause, and when the patient is in his best health. The effects of moral causes in its production are so well known that Raphael has introduced into his picture of the Transfiguration a boy falling into an epileptic fit. Fright is considered a very common cause. Dr. Webster says that one of the worst cases he had ever seen was that of a young female who was frightened by seeing a young man dressed in a white sheet, personifying a "ghost."

Besides moral causes, errors in diet, excess of any kind, blows on the head, every structural or functional disease of the brain, and especially insanity; or any severe disease, as *fever*, or *small-pox*, are all powerful remote causes. Amongst soldiers in the guards, in the experience of Dr. Graham Balfour, the fits, with one exception, seemed to be brought on by gorging the stomach, usually with beer. He became so satisfied of this that, when turned out in the middle of the night to a man in a "fit," the excitation of vomiting generally relieved the paroxysm.

In children the irritation of teething is the most common cause; and, indeed, in France epilepsy is often termed "*mal des enfans.*"

Puberty is the next most frequent period at which it occurs; and its frequency as a primary disease decreases from that time till fifty, when it again increases, from the tendency the brain now has to insanity and to structural disease.

The following table of cases, collected by Dr. Reynolds, shows the influence of age in the production of epilepsy:—

Age at Commencement.	Males.	Females.	Total.
Under ten years,	10	9	19
Between ten and twenty years,	66	40	106
Between twenty and forty-five years,	25	20	45
Over forty-five years,	1	1	2
	102	70	172

Thus the period from ten to twenty is the one of greatest frequency of the beginning of epilepsy, and is that period of life which embraces processes of the second dentition, and of the establishment of puberty.

The larger number of cases in this group showed their first symptoms of epilepsy between the ages of thirteen and seventeen inclusive.

A comparative immunity from attack was present from twenty-five to thirty-five; and the greater proportion of cases forming the third group were seized at or about the age of forty years.

In cases where there is a hereditary taint, epilepsy is developed at earlier ages; and at an earlier age among boys than among girls.

Commencing.	Hereditary.	Non-Hereditary.
Under fifteen years,	83·38	46·15
Above ,,	16·56	53·82

Hereditary predisposition plays an unmistakable part in the production of epilepsy. Its existence or presence is demonstrable in nearly one-third of all the cases. The disease is apparent, especially in persons descended from epileptic parents, particularly epileptic mothers, as well as individuals whose parents or ancestors have become insane or intemperate (NIEMEYER).

As epilepsy is common in idiots whose heads are deformed, it has been affirmed that mankind become more liable to this disease in proportion as the facial angle approaches to 70°. There are many exceptions, however, to this statement. In infancy, and under seven years, epilepsy occurs in nearly equal proportions in both sexes. After puberty, when the distinction of sex is marked, some authors contend that it is more common in males than in females; Dr. Elliotson thinks in the proportion of 27 to 11; Esquirol, however, states that, on comparing the number of epileptics at Bicêtre and at Saltpêtrière, the number of women attacked was one-third greater than that of the men. In an analysis of the returns of the Registrar-General, given by Dr. Sieveking, with reference to the mortality from the disease in either sex during the past seven years, it appears that 6,729 were males, and 6,149 females, giving a relative proportion of 52·26 males to 47·73 females. Sir Thomas Watson also states that he has seen "more epileptic boys and men than girls and women." Dr. Webster is of opinion that the disease is on the increase in this country.

Dr. Sieveking could trace hereditary tendency in 13·4 *per cent.* of his cases. Dr. Webster believes, from a combination of his own investigations with those of Esquirol and others, that *one-third* of the cases may be traced to hereditary descent. There is no doubt that a tendency to the disease is frequently hereditary. It may pass from parent to child; or it may skip over a generation or two, and appear in the grandchild or great-grandchild. The scrofulous diathesis is also a strong predisposing cause of epilepsy.

Of other exciting causes "there are certain vices," writes Sir Thomas Watson, "which are justly considered as influential in aggravating, and even in creating, a disposition to epilepsy. Debauchery of all kinds, the habitual indulgence in intoxicating liquors; and, above all, the most powerful predisposing cause of any, not congenital, is *masturbation*—a vice which it is painful and difficult even to allude to in this manner, and still more difficult to make the subject of inquiry with a patient. But there is much reason to be certain that *many* cases of epilepsy owe their origin to this wretched and degrading habit; and more than one or two patients have voluntarily confessed to me their conviction that they had thus brought upon themselves the epileptic paroxysms for which they sought my advice," (Lect. xxxvi.)

Sir Charles Locock attributes the great increase of the disease during late years to the cause last mentioned in the above quotation (*Med.-Chir. Society's "Report;" Medical Times and Gazette*, May 23, 1857). On the other hand there is a want of direct proof that epilepsy and masturbation have any special relation to each other; and on this point Dr. Reynolds observes "that the one is a tolerably prevalent disease, and the other a very widely distributed vice. There are multitudes of epileptics with regard to whom no such suspicion could ever be entertained; and there are, it is to be feared, much larger multitudes of masturbators who have never become epileptic. When, therefore, we find the two elements combined in the same individual, it is necessary to observe some caution in our attempt to interpret their relations."

Dr. Clymer also is of opinion that the influence of excessive venery or of masturbation in the production of epilepsy is undoubtedly overstated. The serious nervous disturbances to which both of these actions give rise, he considers to be of a very different nature from those of epilepsy. He has seen many confirmed masturbators of both sexes without a suspicion of epilepsy.

Irregularity and perverted state of the menstrual function, associated with hysteria, is another frequent source of the malady. The repression of eruptions, and especially those about the head, are also to be set down as causes which bring about the development of the disease; so are some of the *constitutional diseases*, such as *rheumatism*.

Diagnosis.—An immeasurable responsibility is associated with the diagnosis of such a disease; and, as already seen, the very slightest cue may be all which may be given to distinguish the epileptic state. It is especially to be distinguished from *apoplexy* and *hysteria;* and the following are the classical grounds of diagnosis as given by Dr. Reynolds:—

1. *The Mental State* of the epileptic is thus far characteristic. By far the greater number exhibit a deficiency of the powers of the Will in relation especially to *Thought, Emotion, Sensation,* and *Mobility.* The mind is inclined to wander in a half-abstracted state, and without

energy of purpose. There is little or no power of attention or concentration of Thought, and there is a slowness of apprehension, with defective Memory. The Emotions and their expression are undirected and uncontrolled. The patient can only give unsatisfactory and often totally unmeaning accounts of sensations experienced. Something is felt to be wrong, but the place can hardly be fixed upon; and if the head, thorax, abdomen, or limbs are referred to, the patient is rarely able to express what he has experienced. A "working in my inside" is the comprehensive phrase commonly used to express their indescribable sensations. There is also a characteristic sluggishness and clumsiness of the voluntary movements. The walk and manner of the patient become ungainly. He rolls along rather than walks, stumbling over objects in his way, in an unnecessarily awkward manner. The countenance tends to be dull, expressionless, and morose. These phenomena may be so slight as almost to escape detection, and may in many cases be overcome by a determined effort of Will. Sometimes, on the other hand, they are extremely well marked, and graduate into utter stupidity and dementia with paralysis.

2. The *Motorial* and 3. *Sensorial* phenomena are such as have been described under the head of *symptoms.*

The attacks may be distinguished into two groups, namely:—(1.) Those in which the loss of Consciousness is complete, associated with violent spasmodic movements. This group comprehends "*le haut mal*" of the French authors, and the laryngismal and tracheal epilepsy of Dr. Marshall Hall. (2.) Those in which one element predominates much over the other, even to its entire exclusion;—(*a.*) Attacks in which loss of consciousness being complete, there is little or no spasmodic movement. This class includes "*le petit mal,*" or "*vertigo epileptiforme,*" of the French, and the syncopal attacks of Dr. Marshall Hall; (*b.*) Attacks in which there is marked general or partial spasms of the muscles, somewhat of a tonic kind, but in which there is no appreciable loss of Consciousness. Such seizures constitute the "abortive" attacks of Dr. Marshall Hall.

One individual afflicted with epilepsy frequently presents every variety of these attacks, while any one form may exist alone; but the essential features of a fully expressed epileptic attack cannot be mistaken. They consist of—(1.) The simultaneous occurrence of the following symptoms:—Complete loss of Consciousness, general quasi-tonic contraction of the muscles, impeded respiration, darkened face and surface generally, with distended jugular veins, dilated pupil, distorted features, throbbing carotids; (2.) These phenomena are quickly followed by—persistent loss of Consciousness, clonic violent muscular contraction, laborious respiration, with tracheal gurgling noises; slight return of colour in the face and body generally; oscillation of the pupil and eyeball; chewing movements of the jaws, and foaming at the mouth; (3.) The gradual cessation of these symptoms, and the production of another stage, marked by the following characters:—Return of Consciousness for a short time, with an aspect of astonishment, alarm, and suspicion; and then followed by drowsiness or profound coma; occasional semi-voluntary movements, such as change of position, laboured slow respirations, with stertor and tracheal rattle, paleness of face, coldness of surface, with perspiration; the pupils often contracted, and the conjunctivæ injected; (4.) After sleep the patient becomes more natural in manner, and feels some headache and general soreness.

In the diagnosis of epilepsy it must be always borne in mind, especially in dealing with soldiers, seamen, prisoners, mendicants, and vagabonds, or others with whom powerful motives often prevail to feign diseases, that epilepsy is perhaps more frequently attempted to be copied than any other affection; and often with wonderful success. The means of detection consist,—(1.) In cross-examination as to the consistency or inconsistency of the accounts of the fits, and general description of the attacks. This can only be well done when a perfect knowledge of the symptoms and grounds of diagnosis are familiar to the examiner. (2.) By observing whether or not a situation (favourable always to the malingerer) is chosen for the seizure. (3.) True epileptics seek retirement, and are frequently hurt by their falls. Feigned epileptics delight to exhibit in public, and rarely sustain any bodily injury. (4.) Let the eyes be closely observed. In true epilepsy they are partly open, with the eyeballs rolling and distorted, the pupils dilated, and not contracting by the stimulus of light. The feigning epileptic prefers to shut his eyes completely; and may occasionally be seen to open them to "take a peep," so as to ascertain the effect of his exhibition. *His iris always acts on exposure to the light.* (5.) The skin of an impostor generally perspires from his exertions; that of a true epileptic in the paroxysm is generally cold. (6.) An impostor will not readily bite his tongue or void his excrements or urine. (7.) Tests peculiar to beadles and police constables consist in dropping melted wax upon the suspected feigning person, putting some gin into the eyes, pressing the thumb nail with force under that of the supposed impostor—an experiment productive of sudden, excrutiating and harmless pain. (8.) The mere speaking of or proposing some severe remedy in the presence of the patient is sometimes enough to detect imposition. Sir Thomas Watson (from whose *Lectures* these statements have been condensed) specially recommends a very harmless and ingenious device—namely, in the hearing of the would-be patient, gravely propose to pour *boiling water* on his legs as a remedy, and then to proceed actually to pour *cold water* upon them. Three humorous instances of detection are thus related by him:—

> "Dr Cheyne mentions an instance in which one table was placed upon another, and a soldier who was supposed to be shaming was laid upon the upper one while his paroxysm was on him. The fear of falling from such a height soon stopped his convulsions. Mr. Hutchison relates the case of a sailor who was suspected to be a cheat, in whom the convulsions were instantly removed by blowing some fine Scotch snuff up his nostrils through a quill. This brought on another kind of fit, namely, a fit of *sneezing*, which lasted nearly half an hour; and there was no return of the epilepsy so long as Mr. Hutchison remained in that ship. He tried the same expedient in cases of *real* epilepsy, but never could produce any similar effects, although the patients were not snuff-takers. There was a beggar in Paris who often fell into epileptic fits in the streets. One day some compassionate spectators, fearing that he might injure himself in his

struggles, got a truss of straw and placed him upon it; but when he was in the height of the paroxysms, and performing remarkably well, they set fire to the straw, and he presently took to his heels," (Lect. xxxvi.)

The sphygmographic characters of the pulse in epilepsy have been proposed as an aid in the detection of true from feigned epilepsy.

In true epilepsy the pulse curves are large, the line of ascension is high, with well marked dicrotism, and these characteristics last from thirty minutes to several hours. Similar pulse traces cannot be got from persons made to gesticulate violently, from walking or rapid running, nor from any other form of severe exertions. In the cases of feigned epilepsy the pulse traces bear no resemblance to those of true epilepsy; and as the sphymographic characters of true epilepsy remain for some time after the paroxysm, in all cases of suspected malingering the pulse traces should be taken several times during the hour after the end of the attack—real or feigned (Voisin in *Annals d'Hygiéne Publique,* Avril, 1868).

Another most reliable distinction between true and feigned epilepsy is that dwelt upon by Trousseau as the *initial facial pallor,* and also *the dilated pupil,* already mentioned, even on exposure to bright light, which cannot be imitated. It is, unfortunately however, of too short duration to be of much practical use in many cases of feigned disease.

Nearly all malingerers keep the fit up too long, and devote too much attention to certain symptoms which they believe to be pathognomonic, such as turning the thumbs in to the hand, and foaming at the mouth.

Prognosis.—Epileptic convulsions during teething generally subside about the second or third year; children, likewise, first seized between three and four years old, are often cured, or the disease subsides at puberty, except when hereditary.

Epileptics attacked after puberty are generally incurable, and especially when epilepsy is conjoined with insanity. Pregnant women attacked with epilepsy are in great danger.

As to the positive certainty of any cure for the disease, a proper feeling of scepticism prevails. In the majority of cases no anatomical lesions exist, even after a long series of years, in which the recurrence of the fits have been more or less constant. According to the belief of Dr. Sieveking and of others, a diathesis is necessary to their occurrence, and this may be suppressed or held in check; but it is very doubtful if it can be eradicated.

Niemeyer writes that recovery must be regarded as rare, in spite of the opposite opinion of many observers. Nocturnal epilepsy is of a specially malignant and obstinate character.

Generally, the more distinctly the malady is traceable to hereditary tendency, and the more plainly it depends upon structural disease of the brain, the longer it has lasted, the more violent the fits, the more frequent their occurrence, and the deeper the impression which they leave behind them, the less is the chance of recovery (NIEMEYER).

Nevertheless, well-selected remedies have a power in repressing the paroxysm, and often of indefinitely postponing it;—more especially dietetic and regimenal treatment.

The duration of the disease before treatment is commenced has an obvious influence over its curability. "It is seldom," writes Dr. Watson, "that any permanent ill effect can be noticed as having been left behind by any one single fit; but, unhappily, this cannot be said of their repetition." "More, probably, depends," he continues, "upon the *repetition* of the fits than upon their precise *nature* or *severity*." "Every successive attack strengthens the *habit*, and renders the individual more obnoxious to future seizures; every arrest or postponement of a seizure is so much gain in favour of the patient, not only by avoiding the pain and the risk of the isolated paroxysm, but still more by diminishing his future liability to the disease" (SIEVEKING, l, c., p. 212).

Aretæus, in describing the symptoms of epilepsy, has not neglected to speak of the baneful influence of this disease on the Intellect, of the Memory being lost, of the Imagination being impaired, and of the functions of the brain being, in many patients, so subverted that they fall into incurable insanity. Esquirol gives the cases of 385 epileptics under his care, in the Hospital Salpêtrière, and he states that four-fifths were more or less insane. The remaining fifth had preserved their reason, but, he adds, "a reason so broken!"

> "A single paroxysm often leaves the patient in a worse condition than that in which it found him; but this is not perceptible to an ordinary observer until after the alteration has been rendered apparent by repeated fits and repeated small additions to the permanent injury. The friends of the patient remark that his Memory is enfeebled in proportion to the number of the attacks; that his mental power and intelligence decline. His features even assume by degrees a peculiar character, and too often he sinks into hopeless fatuity, utter imbecility, or confirmed insanity. It is this tendency which renders epilepsy so sad and fearful a disease. . . . Cases do occur in which epileptic persons preserve their faculties to a good old age; but those who are early epileptic do not often attain old age; and whenever the disease comes on, if it repeat itself frequently, it is followed much more often than not by impairment of the Mind, or by some apoplectic or paralytic affection," (WATSON, Lecture xxxv.)

Acuteness of judgment is lost, Memory and power of imagination diminish, the gentler and nobler impulses recede more and more; while the excited and unbridled propensities, lasciviousness, and gluttony, too often impel the patient to commit violent or criminal actions. They often avoid society; are odd, capricious, very troublesome to those around them, and apt to burst into violent fits of anger. The personal appearance of the patient also undergoes a change in cases of long standing epilepsy (NIEMEYER). Esquirol also notices the coarseness of the features of an epileptic, his swollen eyelids, his thick lips, faltering look, and general clumsiness of body.

Such are the phenomena associated with the paroxysms of epilepsy —a disease not only frightful from the violence of the symptoms, but also from the serious effects it may produce on the moral character, as well as on the physical frame of the unhappy patient. While some may fall into the fire, and may be burnt to death, others fall into the water, and may be drowned, although the pool may be but a few inches deep (CHEYNE). Bruises and fractured limbs are also not unfrequent. Many epileptics have a convulsive action or *tic* of the muscles of the face, or their legs waste, and are unable to support the weight of the body. In some instances the leg has been flexed under the thigh—a contraction which has lasted more than a year; while in others the patient has become paralytic.

Treatment.—The treatment divides itself into what is to be done during the paroxysm, and subsequently during the intervals.

When adults are labouring under the paroxysm, little, in general, can or ought to be done except bringing the patient into fresh air, taking off what may be around the neck, and baring the chest, together with the more imperative duty of preventing the patient doing himself any injury. Bleeding, so often had recourse to, is rarely found beneficial; except, however, as regards females, in cases of suppressed menstruation. If, however, the paroxysm be greatly prolonged, the application of cold to the head, and opening the temporal artery, where symptoms of excessive cerebral congestion are obvious, may be of some service, as in cases recognized as plethoric.

It is of great importance to shorten the paroxysm as much as possible. Dr. Sieveking recommends that a trial be made of the galvanic current during the fit. The conductors should be moistened sponges, so as to insure the passage of the current to the deeper-seated tissues.

The paroxysm over, the probable exciting cause of the paroxysm should be investigated, and, if possible, removed; the state of the bowels should be particularly inquired into and regulated, and leeches should be applied to the temples if the headache be severe. In women, also, the catamenia, if defective or excessive, should be remedied. These few simple rules are of the first importance, not only as removing the immediate inconveniences incident to the attack, but also as a means of prolonging the interval, and perhaps preventing its future occurrence. We must take into account, and search out for all causes which may have contributed to the malady. In assuming the charge of an epileptic, it is necessary to commence first, by regulating the external relations of the patient, his habits and his bodily health, so that every suspicious condition of his life to which the origin of the disease can in any way be ascribed may be corrected (NIEMEYER). In a few instances the patient, by the adoption of certain rules, is cured, and the prevailing principles of treatment in epilepsy mainly consist in local derivation, or counter-irritation directed to subdue cerebral congestion, and in the use of such tonic remedies as may be indicated by a careful inquiry into the condition of the individual organs, and how their several functions are performed. The intensity of the headache suggests more or less active counter-irritation by *blisters, dry or wet cupping, ointment of tartrate of antimony, setons,* or even *the actual cautery applied under chloroform.*

The remedies that have been employed are *valerian, iron, zinc, quinine, musk, opium, asafœtida, the iodide and bromide of potassium, camphor, ether, and the preparations of turpentine.*

The *nitrate of silver,* once esteemed a specific in this complaint, has not only failed, but, by occasionally staining the *rete mucosum* of a dingy hue, has often permanently disfigured the patient. Of the long catalogue of remedies which has been mentioned, each medicine is perhaps useful for a few weeks; but after that period its good effects are, for the most part, lost; so that it would appear to act rather mentally than physically in removing the cause and altering the morbid tendency.

"Whatever remedies or course of treatment you pursue," writes the late Dr. Todd, "do not appear to despond, or use any other language to the patient than that of hope. Avoid extravagant promises, as inconsistent with that love of truth which ought to characterize every professional man; but unless you have the strongest evidence against it, do not yourself, nor allow your patient, to abandon hope" ("Clinical Lectures," *Medical Times*, August 12, 1854).

The employment of purgatives is indicated for the removal of waste matter, to act as a derivative from the head, to expel foreign matters or worms from the intestines, and generally to regulate the bowels; and the laxatives most suited to epileptics are *rhubarb, compound colocynth pill, aloes, castor oil, taraxicum, sulphur in combination with magnesia or rhubarb;* and the Pullna bitter water imported from Bohemia, containing, as it does, *sulphates of soda, of lime, of potash, and of magnesia, carbonates of lime and of magnesia, chloride of magnesium, and phosphates of lime* with *free carbonic acid*—about 200 grains of saline matter in a pint, so that half a tumblerful taken in the morning generally produces a full pultaceous evacuation.

The judicious use of an anthelmintic sometimes frees the patient from the disease as well as from a tape-worm or other parasite, which not unfrequently may be the *eccentric* source of the fits. Independently of its anthelmintic properties, however, both Drs. Watson and Sieveking strongly recommend the use of turpentine. It may be given in half drachm doses every six hours.

If a scar, foreign body, tumor, or *neuroma*, be found pressing on a peripheral nerve, its removal by operation may cure the epilepsy.

The preparations of iron and zinc are the most useful tonics; and for the general principles on which iron may be administered, the reader is referred to pages on *anœmia.*

The remainder of the text on the subject of treatment is well given by Dr. Clymer as follows:—

Dr. Trousseau believed that *belladonna* is the least inefficacious of all the remedies for epilepsy, and from its use not only obtained alleviation and improvement in many cases, but was able to count upon a certain number of cures (*Clinique Médicale*, t. ii., p. 95). His formula is:—

Atropiæ Sulphatis, gr. j.; *Sp. Vini Gallici*, ♏c. M. One drop of this solution is to be given every day in the morning, if the attacks happen in the daytime; or, in the evening, if during the night; the dose to be increased by one drop for each succeeding month, and to be always taken at the same period of the day. It should not be pushed beyond the first toxical indications. If the dose is not well borne, it should be increased only every second or third or fourth month. When improvement is apparent, the quantity given in the last dose should be continued for some time, and then gradually lessened, and finally stopped, to be, after an interval, resumed.

"A year is scarcely sufficient to test the influence of *belladonna*, and if in the succeeding year some improvement follows, the treatment is to be followed for two, three, and four years" (*l. c.*, p. 95).

Dr. Trousseau, however, in practice seems to have adopted a mixed treatment, giving *belladonna* in the morning, with the *nitrate of silver*, the *sulphate of copper*, and the *lactate of zinc*, in the evening, alternating these last every ten days. The following are his modes of administration:—

℞. *Argenti Nitratis*, gr. ij.; *Pulv. Acaciæ, Aquæ destil.*, āā q. s. Div. in pil. x. One every night. Or,—

℞. *Cupri Sulphatis*, gr. xx; *Pulv. Sacchari*, ʒj. Div. in pil. xx. Two every night. Or,—

℞. *Zinci Lactatis*, ʒj; *Pulv. Sacchari*, ʒij. Div. in pulv. xx. One every night. Or, the zinc may be made into pills with the conserve of roses.

Within the past ten years the *bromide of potassium* has been largely used in epilepsy, and the testimony to its utility in the disorder is uniform and strong.

Dr. C. B. Radcliffe writes:—"In the summer of 1858 I began to give this medicine almost promiscuously in cases of epilepsy, and epileptiform disorder, and from that time to this (1864), I have been continually finding fresh reasons for persevering in the practice. . . . The conclusion at which I have arrived is, that *bromide of potassium* is the only remedy in epilepsy upon which most dependence can be placed."

The late Dr. Bazire, in a note to his translation of Trousseau's *Clinical Lectures*, speaks thus of the experience of the physicians of the Hospital for the Epileptic and Paralyzed, London, and of his own, with this remedy:—"The results obtained are such as to warrant the conclusion that it is infinitely superior to all other remedies that have been recommended against epilepsy. It is certainly far superior to *belladonna* in its power of diminishing the frequency and severity of epileptic attacks and epileptiform seizures in general. Nay, more, of warding off the attacks, lengthening the intervals between them, and, in some cases, of bringing on a cure" (vol. i., p. 99).

"The most recent, and a very high, authority, Dr. J. Russell Reynolds, in his excellent article on Epilepsy (*System of Medicine*, vol. ii., 1868), says:—'*Bromide of potassium* is the one medicine which has, so far as I know, proved of real service in the treatment of epilepsy. . . . In large doses it has scarcely ever failed to give much relief. . . . Given in doses, ranging from ten to thirty grains, three times daily, it has had these effects:—In some cases it has completely cured the patient, and the cure has been permanent for years, and is so now. In others it has arrested the attacks, so that none have occurred for periods varying from a few months to two or three years; but on the omission of the medicine the seizures have returned. In such cases the attacks have again ceased on the readministration of the medicine. In a third series of cases it has diminished the frequency and severity of the seizures, but has not removed them altogether; the patients, while taking the bromide, have had one-half or one-third the number of fits to which they were habituated. Such patients have gone back to the old frequency of recurrence when the drug has been omitted, and have again improved when it has been readministered. In a fourth, but very much smaller number, it has been good for a time, and then has appeared to cease; and in a fifth, and yet smaller proportion, it has been apparently without any appreciable effect. . . . It is to be demonstrated that there is yet something "specific" in the action of *bromide of potassium*'" (p. 280–1).

"Dr. Voisin, of the Salpêtrière Hospital, says, that it does not cure absolutely, but diminishes the disorder in a marked degree, lessening and even suppressing the nervous erethism" (*Bul. de Thérapeutique*, 1867).

"Dr. Edward Fox reports fifty-two cases in which *bromide of potassium* in not less than twenty-grain doses was found 'satisfactory,' and ten cases in which it wholly failed, in one of which *belladonna* was more successful" (*St. George's Hospital Reports*, vol. ii., 1867).

"Dr. Brown-Séquard, than whom few have had as large experience in this disease, began to use the *bromide of potassium* in large doses towards the end of 1860. He writes:—'I was soon led to associate the *iodide of potassium* with the *bromide:* and it became almost at once evident that, in most cases of epilepsy (whether idiopathic, symptomatic, or sympathetic, but especially in that form, which is much more common than it is admitted to be, in which that convulsive affection is allied with, or due to, a congestion of the base of the brain or its meninges), these two remedies did more good than either of them alone. By the end of 1861, after I had ascertained that the *bromide of ammonium* has a special therapeutic influence in cases of congestion of the *medulla oblongata* and of the upper parts of the spinal cord, I began to associate that salt with the *bromide* and *iodide of potassium* in the treatment of epilepsy. . . . That mode of treatment has been submitted to an extensive trial, which leaves no doubt as regards its superiority. Although certainly it does not often cure permanently, it usually diminishes considerably, the violence and the frequency of the attacks, and is much more successful than the various modes of treatment by the best remedies against epilepsy, such as *atropine* or *belladonna*, the *sulphate of copper*, the *nitrate of silver*, *strychnine*, *valerian*, *zinc*, *digitalis*, or the means of counter-irritation by applications of ice, *moxas*, *croton oil*, &c., on the spine or the head'" (*Lectures on the Diagnosis and Treatment of Functional Nervous Diseases*, 1868, p. 81–2). His usual prescription is:

℞. *Potassii Iodidi*, ʒj; *Potassii Bromidi*, ℥j; *Ammonii Bromidi*, ʒijss.; *Potassæ Bicarbonatis*, ℈ij; *Infus Calumbæ*, f ℥vj. M.

A teaspoonful before each of the three meals, and three teaspoonfuls at bedtime, in a little water.

"In syphilitic epilepsy, the *iodide of potassium* is increased to five or six drachms. Where the attacks begin with violent laryngismus, the *bromide of ammonium* is raised to three or four drachms, and the *bromide* *of potassium* diminished by two drachms. The following rules are laid down as to their use:—

"The quantity of these medicines to be taken daily should be large enough to produce an evident though not complete anæsthesia of the fauces and upper parts of the pharynx and larynx, this quantity varying, with the patient's idiosyncrasy, from forty-five to eighty grains of the *bromide of potassium,* and from twenty-eight to forty-five grains of the *bromide of ammonium,* when only one of these salts is given, and a lesser quantity of each, but especially of the *ammonium,* when given together.

"These remedies very rarely produce a good effect in epilepsy without causing an acne-like eruption in the face, arms, neck, shoulders, and there seems to be a positive relation between the intensity of the eruption and their efficacy; it is important, therefore, when there is no eruption, and also when it begins to disappear, to increase the dose, unless the dose given in the twenty-four hours is already so large that any increase brings on great sleepiness in the daytime, a decided lack of will and of mental activity, dulness of the senses, drooping of the head, considerable weakness of body, and a somewhat tottering gait.

"It is never safe for a patient taking these drugs to be even one day without them, so long as he has not been quite free from the attacks for at least fifteen months.

"The debilitating effect of the bromides ought to be lessened by the use of *strychnine, arsenic,* the *oxide of silver, ammonia, cod-liver oil, cold douches* or *shower-baths,* and wine and a nourishing diet. There is an antagonism between *strychnine* and the *bromides,* and, when prescribed together, the dose of the *bromides* must be increased.

"*Iron* and *quinine* should not be given in epilepsy, unless complicated with *anæmia* or malarial poisoning, except, perhaps, the double salt of the *citrate of iron* and *strychnine.*

"A gentle purge every five or six weeks maintains the power of the bromides" (Brown-Séquard, *l. c.,* p. 84–6).

"In night-seizures the *bromide of potassium* would seem to have less influence than in day-fits" (Duckworth Williams).

"From Dr. Clymer's personal experience with the *bromide of potassium* in epilepsy, from all the published evidence of its effects in the disorder which is definite, and not vague and general, and from the inquiries he has made, and what he has seen in the practice of physicians who have long and properly employed it, he must repeat what he wrote in the first American edition of this work (1866) namely, that 'he has never seen nor heard of, nor found recorded one single well-authenticated case of the permanent cure of epilepsy, under its use alone, or when combined with either of the other salts of *potassium* or *ammonium,* however long or judiciously administered. As the evidence now stands with respect to the value of this medicine in the treatment of epilepsy, it may be said that it most often exercises at once a marked influence over the severity of the paroxysms, and lengthens the intervals between them; that in cases of the disorder apparently pathogenetically similar to those that have been benefited by its employment, it may have no good effect; that in cases of long standing it frequently fails to give any relief; that its control over the disorder seems to be in inverse proportion to its duration; that when its administration is continued for a long time, the remedial power at first shown is apt to diminish and finally to cease altogether; and, lastly, that there is as yet no proof of its curative property."

"Clinical familiarity with epilepsy, and a history of its therapeutics, go to show that there are no grounds for belief in any specific against this affection, and the physician who promises a cure, or even relief, by means of any one remedy in this disorder, runs the risk of damaging not only himself, but his art. It cannot, however, be doubted, that, in a certain number of cases of epilepsy not apparently caused by any coarse cerebral lesion, the severity and frequency of the seizures may be greatly abridged, so that the sufferers may be brought to enjoy often a great degree of immunity and comfort; the proportion of such cases to the whole number afflicted, is, perhaps, few, but is still sufficiently numerous to give hope and encouragement to further trial. Such results, however, can only be surely gained by means which will increase and develop vital power generally.

"But it should be borne in mind that a tonic treatment does not alone consist in the administration of a tonic drug. Whilst many cases of epilepsy demand the use of tonic medicines to fulfil certain present indications, they should in no sense be looked on as curative, or even remedial, but only as adjuvants, and when they have done their work should be laid aside. Of this class of remedies, none, perhaps, is more valuable than *arsenic*, given in minute doses. Where there is *anæmia*, *iron*, or *manganese* is required. But a general restorative system must be adopted and persevered in.

"The food should contain, as soon as the state of the digestive organs will permit, a certain proportion of fatty and oily constituents, and in most cases, *cod-liver oil* may be given with advantage. The *hypophosphites*, as a vehicle for the introduction of *phosphorus* into the system, would seem to be of service in improving the general nutrition.

"The maintenance of the activity of the cutaneous function is of the first importance. In the beginning an occasional vapour or hot-air bath may be taken; afterwards tepid salt or fresh-water baths, followed by a general grooming of the skin. Exercise in the open air, and gymnastics, both measured by the strength of the patient, are to be insisted upon, as well as such means as will promote the expansion of the lung-tissue. With physical training, mental and moral training should be combined.

"Such are the general principles of the rational or restorative treatment of epilepsy, which, it is believed, will give a larger measure of success than a reliance upon any one of the innumerable specifics that have had questionable and temporary repute, and which have been happily styled by Dr. Trousseau, 'therapeutic rubbish' (*fatras de moyens thérapeutiques*). But if a successful issue is to be had, perseverance and confidence, both on the part of the physician and the patient, are chief conditions of success, and this should be fairly stated at the outset to the sufferer and his friends."

SPASM OF MUSCLE.

LATIN EQ., *Spasmus musculorum;* FRENCH EQ., *Spasme;* GERMAN EQ., *Krampf;* ITALIAN EQ., *Spasmo.*

Definition.—*Idiopathic spasms are tonic or clonic constructions of muscles, not attributable to disease of the brain or spinal marrow.*

Pathology.—It is difficult to identify any anatomical lesion in connection with this functional disturbance, which some believe is associated with trifling and transient lesions of the nerves and their sheaths. Some regard them as a kind of rheumatic affection, and attributable to hyperæmia, with œdema of the neurilemma (NIEMEYER).

Muscular spasms are apt to occur during the course of, or succeeding convalescence from acute or chronic disorders, such as *typhus, enteric fever, malarious fevers, Bright's disease,* and *epidemic diphtheritis;* and it is argued that because these diseases have a pernicious effect upon the assimilation and nutrition of the tissues, that the spasm is therefore probably idiopathic. Cramps are the result of some such slight derangement of the nerves as have been noticed.

Various motor nerves are subject to such excitement as leads to spasms of the muscles to which they belong.

When the morbid impressions act in such quick succession that the muscular contraction is continuous, the condition is known as *tonic spasms.*

If the *spasm* of the muscle relaxes at intervals, but so incompletely, that *spasm* still continues, the condition is spoken of as *clonic spasm.*

Spasms, cramps, or *hypercenisis* occur in various situations, more or less limited to certain muscles or groups of muscles.

(*a.*) *Spasm of facial muscles,* sometimes called *mimic spasm of the face,* or *convulsive tic,* is generally due to some noxious influence acting on the surface of the face itself, such as *cold, contusions,* or other *lesions;* but whether the influence is direct or reflected is unknown in most cases. Sometimes the spasms are believed to be thus far reflex that they seem to arise from the irritation of such remote organs as the rectum or intestines from parasites, or the uterus in *hysteria* or diseases of that organ.

Sometimes the spasm is confined to the eyelids, or to the muscles of the ear, or to the lips, a lip, or portion of a lip. When fully expressed, in the words of Romberg, "grimaces occur, either intermittent or constant, involving one side of the face, and more rarely both sides. In the former case they consist chiefly of elevation or depression of the *occipito-frontalis* muscle. Corrugation of the eyebrows, blinking and closure of the eyelids, twitching and sniffling of the *alæ nasi,* and drawing up and down of the corners of the mouth. These attacks set in suddenly, and as suddenly subside, to recur with equal suddenness at short intervals. In permanent tonic contraction of the facial muscles, the furrows and hollows in the affected side of the face are deeper, the tip of the nose, the commissure of the lips and the chin are drawn to the convulsed side. The muscles are hard and tense, and impede motion so much that one eye cannot be so completely closed as the other" (quoted by Niemeyer).

(*b.*) *Spasms in the region of the spinal accessory muscle* are sometimes assigned to twists of the neck, cold, or disease of the vertebræ. The spasms are either of a tonic or clonic character, and expressed in the muscles supplied by the nerve, *i.e.*, the *trapezius* and *sterno-cleido mastoideus,* and according as one or other muscle is more affected, so are the movements of the head in one direction or another. The paroxysms generally cease during sleep, and the clonic spasms develop in a slow and gradual manner.

When the muscles on both sides are similarly affected, the movement is that of incessant nodding (salam convulsions), an instance of which occurred in a soldier at the invalid depôt at Fort Pitt, Chatham, in 1861; and about the same time and place another soldier had lateral spasms, so that the head rolled constantly from side to side, except when asleep, and the occipital region was soon bare of hair by the incessant rolling on the pillow.

(*c.*) *Idiopathic cramp of muscles of the limbs* is not uncommon as a form of rheumatism among children, and is sometimes the result of cold or of reflex intestinal irritation.

The calves of the legs are the most frequent seat of cramps, which often awake the patient suddenly by their pain, and the desire to grasp the limb or press the foot against a firm resistance.

(*d.*) *Scriviner's spasm* ought rather to be considered here as a local spasm or cramp, for such is the cause of the paralysis.

(*e.*) *Spasms of the muscles of the bladder* frequently arise from the irritation of foreign bodies in that cavity, such as *calculi;* and the combined conditions lead to some organic lesion.

Treatment in all these cases must be directed towards the removal of the probable cause, and improvement of the general health.

The causes of spasm of the bladder have been arranged by Romberg into *cerebral, spinal,* and *reflex.* Mental emotion, such as will induce the "goose skin," through the sympathetics, will, under like circumstances, also induce violent contraction of the *detrusor urinæ,* with intense inclination to pass water, and even to the extent of passing it; so that individuals have been known to micturate from terror or sudden fright.

Vesical spasm is generally of reflex origin, and induced by such conditions as irritation of the urethra, as by introduction of a catheter, by irritation of the *rectum,* as from anal fissure or hæmorrhoids, or irritation of the womb.

LARYNGISMUS STRIDULUS—*Syn.*, SPASM OF THE GLOTTIS; SPASMODIC CROUP; CHILD CROWING.

LATIN EQ., *Laryngismus stridulus;* idem valent, *Spasmus glottidis, Angina spastica, clangor infantium;* FRENCH EQ., *Spasme de la glotte;* GERMAN EQ., *Millarsches asthma,*—Syn., *Krampf der glottis, Spasmus glottidis;* ITALIAN EQ., *Spasmo della glottide.*

Definition.—*A paroxysmal spasmodic disease, in which the muscles of the glottis are contracted, the vocal cords tightly stretched and so approximated,* *and the glottis temporarily, partially, or completely closed. The affection depends upon some morbid excitement of the* par vagum, *direct or reflex, and is only seen in infants, especially during the period of first dentition, and in children before the completion of the second year; and in the great majority of cases between the fourth and tenth month.*

Pathology and Symptoms.—During such partial or complete closure of the glottis, the entrance of air into the lungs is impeded or arrested, so that breathing becomes suddenly difficult; inspiration is effected by rapid spasmodic efforts, accompanied with laborious motions of the larynx, during which the child crows like a cock. More than 300 children in England and Wales die yearly from this affection, of whom about 200 are infants under a year old.

Congenital predisposition exists, as in many families almost all the children in succession are affected (ROMBERG). Children brought up by the bottle, and in towns, are also said to be more liable to the disease than children brought up in the country, and suckled at the breast.

There are neither symptoms of fever nor of inflammation, but suddenly, and without premonitory warning, except occasional but slight breathlessness or wheezing, the breathing is performed by violent and noisy inspirations, with each of which the peculiar squeaking or crowing sound is heard.

The eyes become fixed and glaring, and the face is expressive of great distress. The head is thrown backwards, and the spine is bent as in sposmotonos. If the paroxysms persist and recur for any length of time, the face and extremities become bluish or purple. At length the paroxysm terminates by a forcible expiration, generally followed by a fit of crying when the child, completely exhausted, falls asleep. It is the sudden, violent interruption of respiration by spasm which is pathognomonic of the seizure. There is no cough, and no hoarseness—the *par vagum* alone is affected, so that *croup*, or *laryngeal catarrh* is not to be confounded with the *laryngismus stridulus.*

The spasmodic nervous nature of the affection is still more obvious from the fact that in some cases there are also at the same time spasmodic contractions of the fingers and toes, or of the hands and feet; and occasionally general convulsions may occur, when the little patient may perish. In many cases also the thumbs and fingers are forcibly and involuntarily inflected into the palms of the hands, and one or two toes, generally the large one and that next it, are, in like manner, forcibly inflected towards the soles of the feet. These motions are attended with feelings of pain, and any attempt to extend either the fingers or toes always causes pain and suffering. Sometimes the affection commences by these spasms of the fingers and toes preceding the crowing inspiration. Dr. Craigie mentions a case in which these symptoms preceded the laryngeal phenomena for weeks before a paroxysm of spasmodic breathing took place. The duration of the disease varies from three or four weeks to several months. The duration of the fit lasts from a few minutes to ten or a quarter of an hour; and several fits may take place in the course of a day, after which a week or more may pass without their repetition. It is in very bad cases that the fits repeat themselves frequently, and in which the general convulsions are apt to occur, and the case to prove fatal. Even when a child has remained for months without a paroxysm, there is always a tendency to relapse. During these fits of suspended respiration, there is great fear of death, and the child may really seem dead, till after a period of about two minutes the characteristic gasp is followed by the thin stridulous breathing.

Morbid Anatomy.—A strumous habit of body must be looked upon as a predisposing cause of the disease. The *bronchial glands* and the *thymus* gland were observed long ago, by Peter Frank, to be enlarged in this disease. Since then, the disease has been sometimes referred to the *thymus* and sometimes to the *bronchial gland enlargement*, by Hood, Marsh, Ley, J. H. Kopp, Caspari, Paganstecker, and Hirch. These morbid conditions act through pressure on the *par vagum* and *recurrent branch* of the *inferior laryngeal* nerve, the *superior laryngeal* remaining unaffected. These enlargements, however, are now known not to be constant; and there is evidence to show that *cerebro-spinal* irritation is the more immediate cause of the disorder, as has been shown especially by Dr. Marshall Hall. The irritation which establishes the reflex excitement consists mainly of three kinds:—

(*a.*) The irritation of teething through the *trifacial nerve.*

(*b.*) The irritation of the gastric intestinal tract by overfeeding or improper food, through the *pneumogastric nerve.*

(*c.*) The irritation of constipation, diarrhœa, or intestinal obstruction, acting through the *spinal nerves;* and the action of cold on the skin.

These act through the medium of the *spinal marrow*, and influence—(1.) *The constrictor of the larynx*, through the *inferior or recurrent laryngeal nerve;* and, (2.) The *motions of respiration*, through the *intercostal* and *diaphragmatic* nerves.

Indirectly, the condition is excited by the passage of liquids into the larynx in sucking or swallowing; or from slight catarrh of the larynx.

Treatment.—The warm bath, and hot water to the under part of the body, with sinapisms to the extremities, purgative enemata, cold affusion to the head and face; slapping the chest and nates with a wet and cold cloth; exposure to a sudden current of cold air; movements of the arms for artificial respiration, taking care to draw the tongue forwards; the vapour of ether or ammonia applied to the nostrils, and, as a last resource, opening the windpipe, embrace all the appliances for resuscitation of the child.

During the intervals of paroxysm means must be taken to prevent its recurrence. These mainly consist of attention to the state of the alimentary canal. A searching purgative is generally called for to clear out the contents of the bowels; *calomel, jalap,* and *castor oil* are the most useful agents.

Belladonna, to the extent of $\frac{1}{6}$th of a grain thrice daily, combined with *bromide of potassium* or of *ammonium* may be of use. Sinapisms and linaments to the spine are also of use. The diet ought to be carefully regulated.

Change of air is generally called for, and the tepid and cold bath must be in daily use.

SHAKING PALSY.

LATIN EQ., *Paralysis agitans;* FRENCH EQ., *Paralysie tremblante;* GERMAN EQ., *Paralysis agitans;* ITALIAN EQ., *Paralisi tremula.*

Definition.—*Involuntary tremulous motion, with lessened muscular power, agitation, and continuous shaking, commencing in the hands, arms, or head, and gradually extending over the whole body.*

Pathology.—The disease is generally one of advanced life, and progresses slowly—so slowly that cases within my own knowledge have continued during the past twenty years with little or no change. The Intellect remains unimpaired, and it is only when the movements become so constant as to prevent sleep and lead to exhaustion that any danger is to be feared. Extension of the disease is marked by deglutition and mastication becoming difficult, by the walk becoming unsafe from tripping up of the feet. The constitution begins then to suffer, and general paralysis with coma finally prove fatal. It is a state of decay.

CHOREA—*Syn.*, ST. VITUS'S DANCE.

LATIN EQ., *Chorea:* FRENCH EQ., *Chorée;* GERMAN EQ., *Veitztanz*—Syn., *Chorea:* ITALIAN EQ., *Corea.*

Definition.—*An irregular convulsive action of the voluntary muscles, of a clonic kind, especially of the face and extremities. The movements are either entirely withdrawn from the control of volition, or but little under the direction of the will.*

Pathology.—The history of this disease is a sad picture of superstition. As late as the close of the fifteenth century it does not appear to have been studied by physicians, but was supposed to depend on supernatural causes, or what was termed "demoniacal possession." In Germany it was said for two centuries to have been epidemic, and the patients, probably many of them maniacs, were wont to join in frantic dances; and as late as 1673 they went in procession to the church of some favourite saint, of whom St. John, St. Guy, and St. Vitus were the most reputed. Hence the name of St. Vitus's Dance, by which the disease is sometimes described. As physical remedies were supposed to be unavailing in such a disorder, the priests said masses, sung hymns, and sought by such means to exorcise the foul fiend, believed to be in possession of the patient.

The morbid appearances of the body which have been observed in cases of chorea have not as yet thrown much light on its pathology. Sydenham, Cullen, Rostan, Bright, Stoll, Pinel, and others, who have had frequent opportunities of examining cases of this disease, failed to detect any other morbid appearances than those which were commonly seen in other affections of the brain and spinal cord. Accordingly many various pathological views are entertained regarding chorea, which may be classed as follows:—

(1.) By some pathologists chorea has been regarded as a disorder entirely functional or dynamic, and independent of organic change.

(2.) It is believed by some, and not without good reason, that the blood, at all events in some cases of chorea, is primarily diseased, or becomes so constitutionally, the precise nature of the change being as yet unknown.

(3.) Associated with some other diseases, whose pathology is better known, chorea has been regarded either as a concomitant feature or as a necessary consequence of their previous existence: such, for instance, as *rheumatism*, and *diseases of the heart.*

(4.) Pauses in muscular movements occurring during sleep, and from the action of chloroform, render it probable that the perversion of motor influence is derived from the brain rather than the spinal cord.

Much evidence has been brought forward in favour of the humoral or rheumatic character of the disorder. Dr. Copland has the merit of having been the first to indicate the complication of chorea with that class of diseases, (*London Medical Repository*, vol. xv.) His views have been subsequently confirmed by Drs. Prichard and Roeser, and more recently by the elaborate researches of Dr. Begbie and Dr. See. Numerous instances have also been adduced by Andral, Bouillaud, Bright, Mackintosh, Watson, R. B. Todd, Kirkes, and others, in which diseased conditions of the heart and pericardium have been attended with, or have given rise to, spasmodic diseases of the nature of *chorea*, *paralysis*, *mania*, or *dementia;* and the evidence of these writers is amply sufficient to prove that a considerable number of individuals affected with *chorea* have suffered from *cardiac* or *synovial rheumatism* previously. Dr. Sidney Ringer has observed one or two cases of chorea with considerable elevation of the temperature, but without any of the ordinary evidences of *rheumatism.* He believes such cases may tend to show that even in those in whom there has been no evidence of previous rheumatism, it nevertheless probably existed, but was latent in respect of all the symptoms except elevation of the body temperature. But it is unquestionable that all have not so suffered; and indeed the history of the majority of the cases clearly shows that chorea has a more intimate connection with *disorders of the intellect*, such as *imbecility*, than with perhaps any other morbid state. The presumed blood-condition, similar to that which exists in *rheumatism*, can only thus be regarded as one of many occasional causes, the real essence of the disease being a perverted nervous function, as Dr. Reynolds writes, and with whose remarks I am pleased to find the views here stated agree.

Cardiac affections are apt to supervene in cases of chorea, differing in kind as well as in degree, namely,—(1.) Rheumatic endocarditis, or pericarditis, resulting in organic change; (2.) Functional derangement and cardiac murmurs, due to an impoverished condition of the blood (Romberg); (3.) A chronic affection of the heart itself, "evidenced by the existence of a systolic murmur at the left apex, which cannot be referred to inflammation or organic change of the mitral valve, which has not the usual accompaniments of a hæmic murmur, but which does seem plausibly ascribable to diordered action of the muscular apparatus connected with the valve" (Walshe).

As in the case of most diseases expressed during life merely by perverted functional activity, morbid anatomy is often at fault. Our means and appliances for the accurate appreciation of nervous lesions especially are but rude compared with the fine and delicate textures with which we have to deal. When patients suffering from chorea have died, and the brain has been carefully examined, the most experienced observers have failed to detect any constant lesions by which the occurrence of the symptoms could be explained. This negative evidence no doubt points to some morbid condition of the blood as an essential element in chorea. Rostan had once an opportunity of examining a woman upwards of fifty, and who, from her childhood, had laboured under chorea of the whole of the left side of the body, and of which the limbs were atrophied. "I expected to find," he says, "atrophy of the right side of the brain, but there was nothing morbid; at least, after a most careful examination, I could see nothing." Dr. Bright has given one case which he had an opportunity of examining, and which gives equally negative results. It was that of a young woman aged seventeen, who had formerly laboured under this disease. She had been free from it for four years, when she formed an attachment, was forsaken, was attacked with chorea, and died. The attack was of great severity; she tossed herself about in all directions, bit her tongue, and was with difficulty in any degree controlled. On examination there was a slight effusion in the arachnoid cavity, more *puncta cruenta* than usual, and five or six bony plates opposite the *cauda equina*—phenomena common in many diseases of the brain or cord, and of course incapable of having any pathological significance assigned to them in relation to chorea. It is equally impossible to fix upon any other organ or part of the body in which anything like constant structural lesions have been observed, susceptible of being associated in explaining the nature of chorea.

The structures which most obviously manifest disordered action during life are the nerves and muscles; and for the following reasons we are led to believe that they are maintained in their disturbed and excited state by some morbid condition of the central parts of the brain, and not of the spinal cord, either directly or by reflex action.

1. *Clonic spasm*, of the incessantly repeated character peculiar to chorea, is not a phenomenon of persistent spinal irritation; while *tonic spasm* is a mark of such a condition (REYNOLDS).

2. The movements can generally be in some measure controlled by the Will, unless they are very severe; and even then they are so controlled to some extent (REYNOLDS).

3. The spasmodic contractions cease during sleep, whereas phenomena of an excito-motor character are increased by the removal of Volition. Fixing the attention also to some other object likewise diminishes the intensity of choreic movement (REYNOLDS).

4. The special occasions of increase or of induction of choreic movements are the attempts at volitional action and emotional changes (REYNOLDS).

5. The phenomena of chorea during life, in accordance with the views expressed by Drs. R. B. Todd and Carpenter (which are now very generally received) tend to refer the exciting cause of the disease to

changes going on in the central ganglia of the brain, such changes being expressed in a healthy state through "Volition, Perception, or Emotion, or the balancing and co-ordinating of movements."

6. Experiments on living animals, and observations in morbid anatomy, tend to prove that injury to the *optic thalami* is productive of considerable disturbance to the movements of the body.

7. An opportunity was afforded me, when Pathologist to the Glasgow Royal Infirmary, of examining carefully a case of chorea, which terminated fatally after a most violent attack, the acute symptoms lasting ten days. The result of the examination showed some decided changes in the *corpora striata* and *optic thalami,* sufficiently indicated by the following observations :—

> "The specific gravity of the *corpora striata* and *thalami optici* was different on the two sides of the brain: those on the right side were of the specific gravity of 1·025, those on the left side of 1·031; and this difference appeared from the hydrostatic experiments, as well as from those made with the gravimeter, confirming in some measure the accuracy of the general result.
>
> "The vascularity of these central parts of the brain, when compared with the grey matter of the spinal cord, (which was healthy) was so well marked as to leave no doubt of its abnormal increase.
>
> "Microscopic examination confirmed the existence of increased vascularity, for numerous capillary vessels, in unusual abundance, existed in every section examined. Some of these were irregularly dilated, as in a varicose condition, and were filled to a greater or lesser extent with the red corpuscles of the blood. The amount of granular substance in these parts of the brain, on both sides, appeared to be greater in proportion to the fibrous substance than in the same parts of healthy brain with which I compared them" (Contributions to Pathology, *Glasgow Med. Journal* No. I., 1853).

The late Dr. R. B. Todd remarked that "further observations on this subject are greatly needed, and will no doubt throw much light on the pathology of chorea and other allied affections."

Dr. Walshe, the Emeritus Professor of Medicine of University College, was the first to call attention to a not unfrequent occurrence in the course of chorea, namely, a high specific gravity of the urine. The high density of the urine is most marked where the choreic movements are most active; and no doubt it indicates increased waste of tissue, consequent on the disturbed state of the muscles and nerves (*Lancet,* Jan. 27, 1849, p. 85). In the acute case of chorea, so carefully recorded by Dr. Walshe, four phases were observed in the characters of the urinary discharge, namely—During the first five days it was "*febrile;*" that is, of high specific gravity, deep brownish gold-colour, strong urinous odour, and depositing lithates in abundance. *Second,* There came a period during which a great excess of urea gave a special character to the fluid, while alternating improvement and recrudescence marked the course of the chorea. This superabundance of urea is, in the present state of our knowledge, referred to the muscular waste entailed by the constant convulsive movements. *Next,* there appeared oxalates in the urine passed on the twenty-sixth day, on which day the improvement in the case was so marked that the child might have been considered convalescent. *Subsequently*, an abundant precipitation of phosphates took place, the indubitable result of previous nervous waste.

These observations, originally made by Dr. Walshe, were subsequently confirmed by the late Dr. Todd, and by Dr. Bence Jones.

In a case recorded in his most interesting *Clinical Lectures on Paralysis, Disease of the Brain, and other Affections of the Nervous System*, where the urine was carefully examined from day to day by Dr. Todd, the density of the urine was shown never to have fallen below 1·019, and frequently reached 1·030, and once was found as high as 1·035. As the patient improved in health the urine fell in specific gravity, but was never below 1·019. Lithate of ammonia was nearly always present, and oxalate of lime was frequently found mixed with it. An excess of urea was frequently present. In another case he records the specific gravity of the urine as ranging between 1·030 and 1·040, and afterwards falling to 1·020 and 1·022. Generally speaking, he found the density of the urine highest in those cases in which the movements were most general and most active, and falling steadily with their diminution, and with the restoration of a greater controlling power on the part of the patient.

Symptoms.—Chorea principally consists in singular and involuntary movements of one or more limbs, which prevent the patient from being able to lay hold with certainty of any given thing, or to carry that object, be it a spoon or a glass, with any certainty to his mouth, or to any other place. These symptoms are developed so gradually, that in most cases the disease is not recognized till it has made some progress. The symptoms reach a certain point of intensity, and remain at such a point for a variable period. Premonitory symptoms are neither frequent nor characteristic; but a certain susceptibility to nervous disturbance, and irascibility of temper, are not uncommon. General ill-health is not unfrequent, arising from various causes, as delay of menstruation, and constitutional morbid states, such as *rheumatism*, or the existence of some other general disease. The commencement of the symptoms is often at first insidious, but more commonly gradual, and sometimes sudden. They consist at first simply of restlessness, or of hurried and somewhat clumsy movements. The left side and the upper limbs are frequently affected first; but subsequently the whole body is involved. The lower limbs are generally as much affected as the upper, and the patient can with difficulty walk in a straight line, or if he does, it is always by a series of movements which tend towards the object, counteracted by another series which altogether diverge from it,—his feet turning in and out, upwards and downwards, in every possible direction. The muscles of the face and neck are sometimes seized with this species of convulsion, when the head is not only tossed about, and the mouth contorted into the most singular grimaces, but it may require two or three persons to feed the patient—one or more to hold, and another to watch the proper moment to pop the food into the mouth. Sometimes the motor nerves of the fifth pair are affected, and then the jaw closes with a loud snap, or the articulation of voice is affected, or the effort of swallowing difficult.

Thus the essential phenomena of chorea are motorial, consisting of spasmodic involuntary contractions of the muscles. These have been classified by Dr. Reynolds as follow:—(1.) *Clonic spasms;* of great frequency, unattended by pain, resembling the restless movements of a child who has been irritated or put out of temper. Such spasms occur independently of any attempt at voluntary movements, and are in slight cases almost unobserved. (2.) The patient is agitated by all sorts of odd motions, and has often a vacancy of countenance which gives him a fatuous appearance. These symptoms are constant during the day, but during sleep they generally cease altogether. They affect both sides, as a rule, and in a very few cases one side only. The patient is then said to labour under *hemichorea.* The child's health is generally good; his pulse natural; and his bowels, though occasionally constipated, are by no means uniformly so, but for the most part act regularly. The spasms are generally increased by Emotion, and, while they persist during the day, disappear during sleep. The heart acts regularly, probably owing to the anæmic state generally associated with chorea. Dr. Addison describes a bellows murmur, often mitral, but sometimes aortic, and probably due to the same cause.

Causes.—The disease frequently attacks children otherwise in good health, and without any obvious cause. When any cause is assigned, it is usually terror. Somebody has pretended to cut off the child's head, and perhaps has drawn the back of a knife across the throat; or a person dressed in a white sheet has personified an apparition. The symptoms have been known to follow the fright in a day or two, at other times about three or even six weeks have elapsed before the disease became manifest. The causes producing this affection, however are generally referred to mental impressions. A woman in the fourth month of her pregnancy had a frightfully disgusting object thrown at her bosom. She continued for two months in a state of extreme nervous illness from this circumstance, but recovered, and went her full time, remarking, however, that the child was extraordinarily lively in the womb, and that she was often overcome with the sensations it produced. At birth, the child (a girl) displayed the writhing motions of chorea, and continued to suffer throughout life. When she was about thirty years of age she had the appearance of an elderly child, with a head remarkably small, and a mind hardly removed from complete idiocy (Mayo).

Chorea is limited, or nearly so, to early life, and is rarely seen after twenty. Dr. Heberden states it to be most frequent between the ages of ten and fourteen, and also that it is more common in the female than in the male, three-fourths of the patients under his care having been females. Dr. Rufz regards the ages from five years to fifteen as the most liable to chorea; and that girls are three times more frequently sufferers than boys. Dr. Todd's experience shows that chorea generally occurs between nine and fifteen years of age. It is really a disease of childhood, and is greatly more prevalent at the period of *second dentition* and at the period of puberty than at other periods; and although symptoms somewhat resembling those of chorea are sometimes seen at the adult period of life, and at more advanced ages, still such cases are exceptional. It may even be questioned whether they are due to exactly the same morbid condition as that which gives rise to the ordinary clonic convulsions of early life. The probable influence of the rheumatic constitutional state, or of some other unknown constitutional diathetic condition, has been already noticed.

Before the sixth year of life the disease is rare, and is equally uncommon after the age of fifteen. In certain cases a hereditary tendency to the disease has been observed (NIEMEYER); and the mimic influence of example is not to be lost sight of as a cause, especially in the epidemics of chorea, which are apt to prevail in boarding schools; and, lastly, reflex causes, such as *irritation from worms in the intestines, onanism, pregnancy,* are each to be considered.

Prognosis.—The recovery of the patient, with very few exceptions, may be always prognosticated. The disease will in general gradually decline, with complete removal of the spasms. The mean residence in hospital for cases of chorea has been found to be thirty-one days (RUFZ), although recovery is not complete at that time. Those cases only are apt to terminate fatally which occur during an attack of rheumatism or pericarditis, or when the disease assumes an intensely acute form, the patient losing rest at night and becoming exhausted; then emaciation progresses rapidly, and death occurs in from nine to twenty days. (See account of such a case by Dr. Wm. Weir, in *Glasgow Medical Journal*, No. I., 1853.) Dr. Walshe's experience leads him to believe that when the disease is slowly ushered in, it is more obstinate and enduring than when it is suddenly developed. An irregularly remittent course he regards as an inherent quality of the disease, (*Lancet*, l. c.) When of long duration the accuracy of judgment becomes impaired, and disorders of the intellect are apt to follow. The course of the disease is essentially chronic, rarely terminating before six or eight weeks, and as often prolonged for three or four months. In rare instances it becomes persistent, and lasts more or less through life—marked by remissions and exacerbations.

Treatment.—The indications of cure are (1.) To remove, if possible, all morbid states of the body which may tend to aggravate the disease, such as *constipation, anœmia, amenorrhœa, worms;* (2.) By well-regulated purgative medicines, to subdue any cerebral congestion; (3.) To sustain the strength and improve the vigour of the nervous system by tonic and stimulant medicines, by food, and by the cold bath.

The particular tonic is not of much moment. Dr. Wood recommends the *powder of the black snake root* (*cimicifuga*), in doses of from half a drachm to a drachm, or from one to two fluid ounces of a decoction; or from one to two drachms of a saturated tincture should be given three or four times a day, and continued for several weeks, the dose being gradually increased till it produces headache, vertigo, or disordered vision. The *sulphate of zinc* has also had the credit ascribed to it of curing a large number of cases, beginning with a grain in the form of a pill, three times a day, and increasing the dose till it reaches seven or eight grains daily. The *preparations of iron* are also frequently resorted to with benefit.

Dr. Walshe did not find any of these remedies at all useful in the cute case he has described. Of all the remedies he tried, the "*extract f cannabis Indica*" was followed by the most satisfactory results. It xercised a sedative influence on the muscular action in a marked degree, nd that immediately. *Nitrate of silver* he also found to have no mean nfluence in aiding the cure. The *Indian hemp* was given in doses of ne-fourth of a grain of *the extract* thrice daily. The dose was subse- uently increased to half a grain, and at the same time one grain doses f *nitrate of silver* were administered, and a draught containing eight lrops of *dilute nitric acid*, (*Lancet*, l. c.) Dr. Corrigan had also previously ised *Indian hemp* with much success in chorea (*Med. Times and Gazette*, 845, p. 29; also *Dublin Hosp. Gazette*). The student is recommended o consult a valuable paper, "On the Uses of Indian Hemp in Nervous)iseases," by Professor Russell Reynolds, in Beale's *Archives*.

Arsenic, in the form of *Fowler's solution*, is well spoken of by Romberg, n doses of three to five drops.

Trousseau and Niemeyer consider *narcotics* to be of great value in the orm of large doses of *morphine*; others are of opinion that *narcotics* are iot well-borne in chorea.

Gentle applications of the galvanic current along the spine, the atient standing erect, have been successful in the hands of Benedikt. The current should be just strong enough to be felt distinctly, without ain. Chapman's ice-bags to the spine are also of use. *The tincture of Calabar beans* (ʒi. to ℥i. of alcohol), beginning with twenty minims three imes a day, is recommended by Dr. J. W. Ogle—the dose to be ncreased by ten minims a dose, up to ʒi.

Chloroform has been used with benefit to control the violent move- nents.

But, the best treatment is the improvement of diet, change of urroundings, and moral discipline. Hence it is that children with horea improve so much in well administered hospitals, where the food s abundant and discipline well maintained. Anæmia is constantly ssociated with it, and the treatment must be restorative and tonic.

Of other classes of stimuli, *camphor* in five grain doses has acquired nuch reputation, especially after the discharges have become healthy y the action of purgatives. Many young women, also, who attribute he attack to fright, frequently get well from the simple administration f the *spirit of nitrous ether* in one fluid drachm doses three times a day, ombined with the officinal *camphor mixture*. The catalogue of remedies roposed is endless. In many instances, however, the above medicines re continued for weeks without any manifest improvement. In such ases the cold bath, or the cold shower bath, is an excellent adjuvant; nd, unless the patient is suffering from some structural disease, the ase uniformly yields to this conjoined treatment; great care and ttention being bestowed on regulation of the diet, which should be ight, nutritious, and easily digested.

HYSTERIA.

Latin Eq., *Hysteria;* French Eq., *Hystérie;* German Eq., *Hysterie,* Italian Eq., *Isterismo.*

Definition.—*A complex morbid condition of all the cerebral functions of a chronic kind, probably associated with some morbid state of the emotional or sensori-motor centres, and presenting every variety of alteration, so that the phenomena of hysteria are protean, and simulate or mimic the phenomena of almost every other disease, while the most common and characteristic features of the affection are certain motorial changes of a convulsive nature, and usually of paroxysmal occurrence.*

Pathology.—Three theories have been entertained relative to the nature of this disease, and to the primary seat of the affection :

(1.) Some, with the ancients, refer it to a morbid condition of the nerves of the uterus, and organs of generation, because the disease is almost exclusively peculiar to females between the age of puberty and that of the extinction of the sexual functions, and is accompanied by morbid conditions of the sexual organs, especially *infarctions* of the womb, ulcerations of the *os uteri* and *flexions of the uterus, dermoid cysts of the ovaries, irritation of the genitals* from sexual excitement, imperfectly effected or excessive *coitus,* or *onanism.*

(2.) Others consider it exclusively due to a morbid state of the cerebral structures.

(3.) A third class refers the phenomena to a morbid excitability, due to "*nutritive derangement* of the whole nervous system, *both central and peripheral*" (Hasse), which renders it liable to be thrown into disorder by causes insufficient materially to disturb its action in health—thereby implying paralysis of some nervous centres.

A tendency, either congenital or acquired by injudicious training, has an obvious influence upon the induction of *Hysteria.* It rarely manifests itself before the 12th or 15th year of life; and, although it seldom appears in old age, it frequently outlasts the period of child-bearing.

Post-mortem examinations of the bodies of those who have died from other diseases, while suffering from hysteria, have yielded negative results.

Symptoms.—The forms and degrees of hysteria are so numerous that the difficulty of describing this disorder is very great. The modifications of age, temperament, states of nervous sensibility, physical and moral education, and grades of society, so influence its aspect that it is only possible to give a mere general outline. It is usually divided into three forms : *first,* that which is characterized by what is termed the "*globus hystericus,*" in which the sensation of a ball rising in the throat, or a feeling of suffocation, is experienced by the patient, but without convulsions ; *second,* its paroxysmal form, or that in which the *globus hystericus* occurs with convulsions; and *third,* those irregular and anomalous phenomena which often manifest themselves during the intervals of severe attacks.

The milder forms are those which terminate without the formation of the paroxysm. They commonly begin with pains in the epigastrium; in the left side, or in some other part of the abdomen; or the patient is unusually nervous, her feelings excited or depressed. These symptoms having existed for a longer or shorter period, the patient experiences the sensation of a ball, the "*globus hystericus*" rising apparently from the lower portion of the abdomen, and proceeding upwards with various convolutions to the stomach, thence to the throat, and causing sometimes an intense sense of suffocation. At this point the slighter forms frequently cease, but are followed by headache, stiffness of the neck, general weariness, a profuse discharge of a light-coloured limpid urine, and by great flatulence, the abdomen becoming almost instantaneously distended.

When hysteria assumes a paroxysmal form or "fit" it may be preceded by the pains and mental feelings which have been described; but not unfrequently the attack is sudden, and is often caused by some transitory occurrence. In such a case the patient bursts out into a fit of immoderate laughter or crying, the *globus hystericus* begins to form and to rise, and no sooner reaches the throat than she falls to the ground, apparently unconscious and violently convulsed. The fit is now said to be formed, and while in general the convulsions are easily controlled, yet —not only in the strong and plethoric, but sometimes also in delicate-looking slight-made girls—many persons are sometimes necessary to restrain the patient, who writhes her body to and fro, agitates her limbs in various directions, and beats her breast repeatedly with her arm and hand. During the fit the patient also often knocks her head against the bed or floor, tears her hair, screams, shrieks, laughs, cries, or sobs alternately. The respiration is slow, and is rendered still more laborious by spasms about the pharynx and glottis, so that the patient often grasps her neck and throat, or rubs or strikes the epigastrium and side with her hand. During this struggle she may bite her own arms or those of the bystanders, and will sometimes move round the room while lying on her back, by means of the muscles of the dorsal region. The abdomen is often singularly distended with flatus; but in other cases the muscles of that region are tense and irregularly contracted. The pulse is in some cases increased by the violence of the exertion, but in others its beat is natural. The veins of the neck are distended, the carotids beating with more than usual violence. The face is flushed, and "the head is generally thrown back, so that the throat projects; the eyelids are closed, but tremulous; the nostrils distended; the jaws often firmly clenched; there is no *distortion* of the countenance, and the cheeks are at rest," unless when giving expression to some of the above-mentioned phenomena. The temperature of the extremities is often lower at the commencement than natural, so as to cause a momentary shivering, but as the paroxysm forms, the heat is usually restored and sometimes increased. The phenomena attending the subsidence of the paroxysm are very various; sometimes a flood of tears, a fit of laughter, or an exclamation, is followed by a great flow of limpid urine, after which the recovery is generally rapid and complete. In other cases the action of the stomach becomes reversed; and the sympathizing attendant, perhaps watching the patient with the tenderest care, receives its whole contents over her person, after which the patient may lapse, most unconcernedly, into a profound sleep. In others, again, the fit only partially passes off, and the patient lies, to a certain extent, sensible of what is passing about her, perhaps jaw-locked, the secretion of urine suspended, unable to talk, and often obliged to be fed. The fit having entirely subsided, the patient lies exhausted and unwilling to be disturbed, and although more or less conscious of what has passed, she wishes to be thought ignorant of all that has taken place. A want of consciousness may exist when the fit assumes a severe or epileptic form; but this is not a common symptom of the purely hysterical convulsion. In some few cases the patient appears to be delirious, and makes the most extraordinary noises, such as barking or howling like a dog. The duration of the fit varies from a few minutes to two, three, or more hours. These fits readily recur, and no sooner is one fit ended than the patient suffers from another; and in this manner the whole attack may last twelve, twenty-four, or even forty-eight hours. In general the intervals are much longer, and not subject to any general law of recurrence, except that they are more common about the period of menstruation.

In the interval between fits, the symptoms are extremely anomalous and irregular, and more strange and difficult to describe than even those of the paroxysm. Some have their senses so acutely alive, that although the window and bed-curtains may be drawn, still they are pained with light, and the slightest noise distresses them. In some, again, the sense of touch is so exquisite, that they can scarcely bear the weight of the bed-clothes; and to others odours are similarly intolerable. Besides this extreme acuteness of the senses, others suffer pains under or in the mammæ, known to surgeons as the "hysterical breast," lumbar pains, pains in the hip-joint, headache fixed to one spot, *clavus hystericus*, and palpitation. Pain in the region of the spine is also frequent, and often so intense and so exquisitely increased by pressure that it has often been mistaken for ulceration of the intervertebral cartilages. The late Sir B. Brodie recorded that he had seen numerous instances of young ladies condemned to the horizontal posture, and to the torture of issues and setons for successive years, whom air, exercise, and cheerful occupation would have cured in a few weeks. As to painful affections of the joints, it has been stated by the same high authority, that at least four-fifths of the females among the higher classes who are supposed to labour under diseases of the joints, are suffering from hysteria, and from nothing else. The morbid sensibility is chiefly in the integuments, as in the case of the hip-joint, and if they are slightly pinched or drawn from the subjacent parts, the patient complains more than when the head of the femur is pressed against the acetabulum. There is likewise no wasting of the *glutei* muscles, nor flattening of the *nates*, nor painful starting of the limbs. In some instances the patient becomes paraplegic, and is unable to walk, while others suffer temporarily from *hemiplegia*.

It is the extreme acuteness and exquisite sensibility of the senses in hysteria which has led those less skilled in female arts to believe in the instances of animal magnetism and mesmerism they seemed to exhibit, which formerly attracted so much public attention. A most interesting account of the vagaries of hysteria may be read in Sir Thomas Watson's Thirty-eighth Lecture, *On the Practice of Physic.*

Diagnosis.—The best diagnostic guide may be obtained by classifying the symptoms according to Dr. Reynold's plan, as followed in the description of the previous diseases. There is to be noticed—

1. *The Mental State.*—Volition is deficient and misdirected. The Emotions and Ideas exhibit excessive activity; and to the combination of these two conditions is to be attributed many of the peculiar and characteristic features of the disease. *Assertions* by the patient are being constantly made—such as that she cannot control her Thoughts, Emotions, Expressions, or general voluntary movements: or that she cannot move this or that limb, that she cannot open her eyes, that she cannot stand or walk; and if she makes the attempt under such impressions, she certainly fails; and she may simulate the real inability so completely, and so well, that it seems almost incredible that nothing but defective Will is the real source of the failure. If, however, some strong Motive, Emotion, or Sensation come into operation, she may for a moment forget her condition, clap the hitherto moveless hands together, open the closed eyelids, and, with the rapidity and energy of robust health, run across a room or up a staircase with her *quasi*-palsied limbs. It does not appear that with all this there is any intention on the part of the patient to deceive any one, more than herself. It is truly a morbid mental condition on her part, and she doubtless believes in the real nature of her symptoms. Often, also, a species of delirium prevails, in which nonsensical sentences are pronounced in an excited manner. Uncontrolled sobbing, sighing, and laughing are alternately produced, or accompany each other. Somnambulism, ecstasy, or hysteric coma (which is rare), may prevail. Often the expression of the face is insane. A listless, abstracted, vacant look pervades the countenance, as if the individual cared nothing for the things of this world. Combined with this condition there is restlessness and impatience of temper, with monosyllabic talking.

2. *Sensorial.*—The pain of hysteria, which may be anywhere, but most commonly in the head and mammary region, is always described as "intense," "horrible," or "agonizing;" and it is increased when the attention is directed towards it, but lessens when the attention is withdrawn. The patient generally shrieks when the skin is touched. *General hyperæsthesia,* or "nervousness," as it is commonly called, is scarcely ever absent, and may exist for years as the only expression of the disease. It is evinced in several ways; such as:—unusual acuteness of the senses, neuralgias, tenderness, as pressure of the spine, painful affections of the joints (*Arthropathia hysterica*), noises in the ears, spots before the eyes.

On the other hand, anæsthesia may prevail, although it is very difficult to determine whether pain is felt or not by operations which usually cause pain, such as pricking, pinching, or burning parts of the skin.

Palpitation of the heart, or pulsations of vessels, is complained of by nearly all; so is pressure and fulness over the stomach, as *cardialgia,* thirst, desire to micturate, and the like.

3. *Motorial* phenomena, when voluntary, are performed sluggishly and imperfectly. The other phenomena in connection with the motor power are exhibited in convulsions or paralysis. It is the convulsive paroxysms of hysteria which may be mistaken for epilepsy. When they occur, it is almost always at the period of puberty in young females. They frequently attend the menstrual period, and are preceded by the premonitory phenomena of hysteria already described, and which re-appear towards the close of the convulsions. There is probably never complete loss of Sensibility and Perception. The spasmodic movements are general, and of reflex origin, causing morbid excitement of the motor nerves, proceeding from the *spinal marrow* and *medulla oblongata.* The face undergoes little alteration. There is commonly a contractile movement of the eyelids. The patient appears to see, and there is no marked change of the pupil. Foaming at the mouth or a bitten tongue is rare. The attacks are sometimes of considerable duration, and the respiratory movements become very disorderly. After the paroxysm has passed, there is no marked stupor, but merely general exhaustion; and loss of Consciousness appears to be very seldom complete, and never occurs at the outset of the attack. " The patient," writes Sir Thomas Watson," is often able to repeat (although she may not always choose to confess it) what has been said by the bystanders during the period when she seems insensible. This is a point of distinction well worth remembering, for more reasons than one. It not only helps the diagnosis, when the fact comes out, but it suggests certain cautions to ourselves. We must take care not to say anything by the bedside of a hysterical patient which we do not wish her to hear; and we may take advantage of her apparent unconsciousness, and pretend to believe in it, and speak of certain modes of treatment which she will not much approve of, but the very mention of which may serve to bring her out of the fit."

The less expressed forms of hysteria cannot be confounded with the less expressed forms of epilepsy. The non-convulsive form of epilepsy is exclusively expressed through disordered sensorial states, such as by vertigo and a suspension (however brief and transitory) of the mental powers. The non-convulsive forms of hysteria, on the other hand, are chiefly expressed in derangement of the organic functions of the thorax and abdomen (FOVILLE, WATSON).

It is often difficult to distinguish between the many painful affections of the joints which arise from hysteria and the formidable diseases of these parts which they simulate, mimic, or copy; and many mistakes have been made fatal to health and even to life. The character, however, of the patient, her time of life, her general good health, the intermitting nature of the pain, and its following the course of the nerve, enable us generally to determine with much accuracy between these different classes of disease. The most common mistake, however, is that of considering the pains under the mammæ as pleurisy or disease of the liver, or the abdominal pain as peritonitis or enteritis, thereby leading to an abuse of bleeding, blistering, and the administration of mercury. The state of the pulse, however, the general good health of the patient (" for," writes Dr. Wood, " one of the most striking circumstances connected with the disease is the general integrity of the nutritive process—the patient continues plump and rosy"), and most commonly the existence of some uterine irritation, furnishes sufficient grounds for diagnosis between these different diseases.

Nausea, eructations, borborygmi, or *tympanitis, palpitation of the heart,* with *syncopal feelings, frequent micturition* of clear pale urine, are characteristics of the hysterical state. A certain constitution is also characteristic of the sufferer from hysteria. The external conformation of the features of the face is often of itself sufficient to indicate the existing tendency. The "*facies hysterica*" may be recognized by the remarkable depth and prominent fulness of the upper lip, which is more or less thick. There is also a fulness of the eye, with a tendency to drooping of the upper eyelids.

Causes.—The remote causes of this affection are rather moral than physical; and in a young person predisposed to the disease almost any mental emotion will excite it, as anger, disappointment, jealousy, protracted expectation, the loss of a husband, a friend, or a child; indeed, all that brings the Passions, Emotions, or Affections of the mind into play is a cause of this disease; and many women cannot go to church, or witness a tragic representation, without suffering from hysterical paroxysms.

This disease almost exclusively attacks females between the ages of fifteen and thirty, or during that period of a woman's life when the generative functions are fully developed and in their greatest vigour. Those most liable are the unmarried or continent, and those that labour under amenorrhœa or menorrhagia. The married woman often suffers just after conception, or before parturition, or subsequently as a consequence of protracted suckling. The barren woman, however, is more liable, and probably from her mind being acted upon by a greater number of exciting causes, such as disappointment in the prospect of being a mother. Taking classes of women, the higher classes, from their artificial modes of life, are greater sufferers than the lower orders.

Although hysteria is a disease almost peculiar to woman, it is not entirely so, but occasionally affects the male sex under conditions of mingled debility and excitement. Shakespeare makes Lear exclaim, when Gloster relates the cause of his being put in the stocks—

> "Oh, how this mother swells up toward my heart!
> *Hysterica passio!*—down, thou climbing sorrow,
> Thy element 's below!"

The predisposition to the disease, however, is most manifest in that peculiar condition of the nervous system for which we have no more precise or definite expression than *nervous irritability* or *mobility, nervousness* or *hyperœsthesia*—a condition which is more common in women and children than in men, and more common in all persons when in a state of weakness than when in the full enjoyment of muscular strength. In women the affection is more common about the menstrual periods, and immediately after parturition, than at other times: more common likewise among those in whom the monthly discharge is habitually *excessive,* or *altered* (as in *leucorrhœa*), or suddenly *suppressed,* or more gradually obstructed (as in the different forms of *amenorrhœa*). In this condition of mobility both Sensations and Emotions are intensely felt, and their agency on the body is stronger and more lasting than usual; continued voluntary efforts of Mind, and steady or sustained exertions of the voluntary muscles, are difficult or impossible; the muscular motions are usually also rapid and irregular.

Prognosis.—The ultimate result of these cases, though often long and tedious, is always favourable. "In nine hundred and ninety-nine cases out of a thousand, hysteria is attended with no ultimate peril either to mind or body" (WATSON). The disease may continue for years with varying intensity; but there is no fixed period of its duration; and the malady may run into *epilepsy* or disorder of Intellect.

Treatment.—The treatment may be divided into what should be done during the fit, and into what should be done afterwards.

When the patient falls in a fit of hysteria, the first thing to be done is to loosen everything tight about her person. The window should be opened, and the cold air allowed to blow over her. She should then be laid in the horizontal posture on a bed, or on the floor, as a means of rendering the circulation through the brain more equal, and to enable us the more readily to control the convulsive movements of her body. This being done, many modes of further proceeding may be followed. Some recommend, in plethoric cases, that the patient should be bled—a remedy certainly in many instances manifestly improper, and in all of doubtful efficacy. When the jaw is locked, Dr. Wood recommends that an enema, consisting of the yolk of an egg beat up with two drachms of asafœtida, with half a pint of water added, may be administered; or, still better, an enema of turpentine, in which half an ounce of turpentine is similarly mixed with the yolk of an egg, and half a pint of water added. These remedies, in some instances, he adds, instantly remove the affection, but in other cases not for some hours. Another remedy is to fill the mouth with salt: "You generally see them come round if you fill the mouth with salt." The remedy, however, which supersedes all others, and is unquestionably the best, is a good drenching with cold water. "I believe there is more virtue in cold water than in any other single remedy" (WATSON). If the patient lie on the bed, the head should be drawn over its side, and a large quantity of water poured on it from a considerable height out of a pail, jug, or other large vessel, and directly over the mouth and nose of the patient, so as to stop her breathing and compel her to open her mouth. This practice is generally introduced into hospitals; and until it was adopted it was not unusual to see three or four patients in hysteria in the same ward, and at the same time. Under this practice, however, an hysterical case is rare, and the fit seldom occurs twice in the same person, and never becomes epidemic.

After the paroxysm is over, if the patient complains of continued headache, a few leeches to the temples may be necessary, especially if the urine be small in quantity and high-coloured; but in all other cases leeches, blistering, or cupping should be avoided, as tending rather to aggravate than to control the disease. The next object is to regulate the bowels by such remedies as may be necessary, and at the same time to support and tranquillize the patient by stimulants, such as *ether* or *asafœtida,* combined with *hyoscyamus* in the form of tincture, the *syrup of poppies,* or small doses of *morphia.*

Niemeyer recommends the *chloride of sodium* and *gold* as a restorative nerve tonic. He prescribes it in the form of a pill, as follows:—

℞. *Auri. chlorat natronat,* gr. v; *Gum. tragac. Co,* ʒi.; *Sacc. Alb., q.* s.; *misce.* Divide into forty pills, of which one is to be taken an hour after dinner, and another an hour after supper. After a time, two are to be taken as a dose at one time, and increased gradually till eight pills a day are taken; and a cold shower bath twice daily.

The state of the uterine functions must always be inquired into. If leucorrhœa be present, or the menstruation be profuse, the *mineral acids,* or the *bitartrate of potass* will be found most efficacious, by restoring a more healthy state of the deranged organs.

The urine is often suppressed for a time after an attack of hysteria; but unless the bladder be sensibly, and perhaps painfully distended, no attempt should be made to draw the urine off. Something more should be hazarded to avoid this necessity, for the catheter once passed, that operation will require to be performed morning and night, perhaps for several months.

CATALEPSY.

Latin Eq., *Catalepsis;* French Eq., *Catalepsie;* German Eq., *Catalepsie;* Italian Eq., *Catalepsia.*

Definition.—*A sudden suppression of Consciousness; but instead of falling down convulsed, as in hysteria, the patients maintain the position in which they were when the attack commenced. The limbs and trunk persist in a state of balanced muscular contractions; the same expression of countenance which may chance to be at the moment of seizure is preserved. If sitting, the patient continues to sit; if standing, he continues to stand; and if occupied in any mechanical employment, he continues fixed in one attitude; and if he is under the influence of any Passion, the countenance retains its expression.*

Pathology.—This is an extremely rare form of nervous disease, apparently intermediate between hysteria and epilepsy; but probably more allied to hysteria. It affects the two sexes nearly with equal frequency. Dr. Reynolds has observed the cataleptic state ensue in cases of chronic ramollissement of the brain, and in tubercular meningitis; and Dr. Laycock compares the condition to the state presented by the so-called "*brown study.*" The combination of fixed attitude and of unvarying expression gives to the patient the air of a statue rather than that of a living being, and he appears as if suddenly changed to stone. The most remarkable circumstance, however, in this disease is, that the attitude of the body and position of the limbs admit of being changed almost into as many new forms as a painter's lay figure, and the new position, however inconvenient, and almost volitionally impossible, is preserved till again changed, or until the paroxysm has subsided.

Besides this singular state, Consciousness is suspended, and the patient neither receives any impression from external objects, nor retains any recollection of what has happened during the fit.

In this respect the disease approaches in character to epilepsy. The organic functions of life, however, continue to be performed, though feebly. The pulse and respiration are regular, only the former is smaller and the latter less frequent than in health. The colour of the countenance is either pale or undergoes no change. The fit may last a few minutes or a few hours, and is said to have continued in some cases three or four days. The patient at length awakes as from sleep, and generally with a deep sigh, when all the functions of the body are suddenly restored. The attack is generally sudden, and without any previous symptoms; but it is sometimes preceded by headache, stiffness of the neck, or some obvious torpor of the mind or body. The return of the paroxysm is very uncertain, but the disease seldom subsides with the first attack. The following case, given by Dr. Gooch, will best exemplify this affection:—

A lady who laboured habitually under melancholy, a few days after parturition, was seized with catalepsy, and presented the following appearances:—She was lying in bed motionless, and apparently senseless. It was thought the pupils of her eyes were dilated, and some apprehensions were entertained of effusion on the brain; but on examining them closely it was found they readily contracted when the light fell upon them. Her eyes were open, but there was no rising of the chest, no movement of the nostril, no appearance of respiration. The only signs of life were warmth and a pulse which was 120, and weak. Her fæces and urine had been voided in bed. In attempting to rouse her from this senseless state the trunk of the body was lifted up and placed so far back as to form an obtuse angle with the lower extremities, and in this posture, with nothing to support her, she continued sitting for many minutes. One arm was now raised, and then the other, and in the posture they were placed they remained. It was a curious sight to see her sitting up staring lifelessly, her arms outstretched, yet without any visible signs of animation. She was very thin and pallid, and looked like a corpse that had been propped up and stiffened in that attitude. She was now taken out of bed and placed upright, and attempts were made to rouse her by calling loudly in her ears, but in vain; she stood up, indeed, but as inanimate as a statue. The slightest push put her off her balance, and she made no exertion to regain it, and would have fallen had she not been caught. She went into this state three times; the first lasted fourteen hours, the second twelve hours, and the third nine hours, with waking intervals of three days after the first fit, and of one day after the second; after this time the disease assumed the ordinary form of *melancholia*. It might be supposed that symptoms such as these were feigned; but there are cases beyond suspicion of this kind; and another very interesting case is related by Dr. George Buchanan in *The Glasgow Medical Journal* for July, 1857. It is an instance of this singular affection occurring in the male sex.

Prognosis.—The affection is generally innocent; but as it is apt to be associated with cerebral disease, which may end in *cerebritis*, *apoplexy*, or *disorders of the Intellect*, and also with serious organic disease of the viscera (REYNOLDS, WOOD), it behoves the physician to be guarded in predicting results, especially in our ignorance of the nature of this disease.

No constant line of treatment can be stated. The individual case must be judged of upon its own merits, and prescribed for according to the principles which have guided the dictates of treatment in the allied nervous affections.

NEURALGIA.

LATIN EQ., *Neuralgia;* FRENCH EQ., *Néuralgie;* GERMAN EQ., *Neuralgia;*
ITALIAN EQ., *Neuralgia.*

Definition.—*Excruciating pain, which is paroxysmal and returns with renewed violence in a part after periods of temporary remission, and which is believed to be due to some unknown morbid state of the nerves of sensation, a symptom of a local lesion, or more commonly of a general affection, or pains which are continuous but not so violent.*

Pathology.—All neuralgias are symptomatic either of an organic lesion, of which the neuralgic pain is a reflex expression, or the pain is due to a more or less grave organic lesion, involving, compressing, or otherwise irritating various branches of nerves, and so giving rise to direct pain. In some cases neuralgia is *symptomatic* of various cachexias —*e. g.*, of *chlorosis*, of *lead-poisoning*, of *anœmia*, of *malaria*, of *rheumatism*, of *syphilis;* or it is a *reflex* induction from an acute inflammation, as from a *caries tooth*, a *necrosed bone*, a *tumor*, or a *plegmon.* The form of the neuralgia from these several sources may vary, but *pain* of the characteristic kind defined in the definition is alike common to all. In most cases marked peripheral lesions are the starting-point of neuralgia, as in cases of decayed teeth, of necrosed bones, of tumors developed in the vicinity or in the substance of nerve-trunks, or of inflammation, including nerves within its area (TROUSSEAU).

The pain of neuralgia is distinct in character, and involves the minimum of organic change in the affected part, while the whole circumstances of the affection point to lowered vitality as the antecedent cause of the pain (ANSTIE). "The general bodily health is always at a low point when the attacks occur, and the nerves of the part are habitually in circumstances which must tend to lower their functional activity." Dr Anstie gives circumstantial evidence of this in his own case, consisting of the passive flow of tears, the hair of the right eyebrow becoming decidedly grey at a point exactly opposite the *supra-orbital nerve* (the one affected). These he justly regards as so many indications of *defective nervous energy.* Pain under such circumstances is generally the direct consequence of a further depression of an already feeble vitality in the nerves (ANSTIE, *op. cit.*, p. 84).

The principal varieties of this affection are—(a.) *Facial neuralgia, tic douloureux*, or *neuralgia of the trigeminus;* (b.) *Brow ague, hemicrania, megrim* or *migraine;* (c.) *Sciatica*, or *hip-gout;* (d.) *Intercostal neuralgia.* But in addition to these there may be mentioned (e.) *Crural neuralgia;* (f.) *Lumbo-abdominal neuralgia;* (g.) *Cervico-occipital neuralgia;* (h.) *Cervico-brachial neuralgia;* (i.) *Mastodynia*, or *irritable breast of Cooper.*

Symptoms.—The symptoms are similar in kind, whichever nerve is affected, modified only by the position, connections, and distribution of the nerve.

Two forms of pain are to be distinguished, namely—one continuous, increased by pressure, confined to circumscribed spots or points in the course of the nerve—usually not a very severe pain, but a very annoying and persistent source of irritation; another form of neuralgic pain occurs in paroxysms, spreading *from a point* along the course of a nerve. This kind of pain is terrible, and almost unbearable. It sometimes passes upwards and sometimes downwards from a point, and the pain is felt to be deep-seated rather than superficial.

The several varieties of neuralgia require special notice—

(*a.*) *Facial neuralgia.*—The branches of the *trifacial* nerve may be attacked separately or conjointly; most commonly, however, only one branch is affected, less frequently two; and the case must be severe in which the three branches, or the whole side of the face, is affected. Nevertheless, it sometimes so happens, extending even over the summit of the head, and over the temporal region, by the deep branch of the fifth pair, which emerges to the surface anterior to the external meatus. It is even also associated with a similar affection of the occipital branches at the same time. Next to the sciatic nerve, no nerve is so often the seat of pain as the *trifacial*—a greater liability to neuralgia arising from two circumstances, namely—(*a.*) The passage of its branches through narrow canals, or openings in bones, where they are readily compressed (HYRTL); and (*b.*) from the distributions of the nerve over a large cutaneous surface, more exposed to cold and to changes of weather than any other part of the body.

The attack of facial neuralgia is sometimes sudden, but more generally it is preceded by a dull aching pain at the points where the nerve issues from the cranium, or becomes superficial. There are especially three points of pain in facial neuralgia,—namely, the *supra-orbital foramen; the anterior opening of the suborbital canal; the mental foramen.* These points lie nearly in a vertical straight line. If the neuralgia be limited to the *first* branch of the fifth pair, the pain spreads in the branches of the supra-orbital, affecting especially the forehead, eyebrows, and upper eyelids, occasionally the eye itself. After this threatening symptom has lasted a few hours or a few days, the patient is seized with a violent darting or shooting pain in the course of the nerve, returning at intervals—phenomena which are characteristic of the disease. The paroxysm is short, lasting only a few seconds or a few minutes; but the pain is perhaps the most severe that the human frame is cabable of suffering. Some patients have compared it to an electric shock of great intensity, others to the conflagration of gunpowder, and others to the intensity and violence of a fulminating powder. The late Dr. Pemberton was known to have stamped the bottom of his carriage out during the paroxysm; and Valleix mentions a physician who, suffering from neuralgia, was induced, by excessive agony, to make deep incisions into his face, and then to apply the actual cautery to the wound; but his pain not being mitigated by these methods, he several times attempted suicide. Even in mild cases the patient often, on the instant of attack, becomes fixed like a statue, fearing to move a muscle or a limb, lest he should aggravate the pain, or reproduce the seizure. This is a condition common to many nervous affections attended by excruciating pain, such as *angina pectoris.*

In cases of ordinary intensity the effect is so completely limited to the nerve that the skin is not discoloured, while the organs immediately in connection with it are little affected—the eye, perhaps, being only watery, the nose hot, and the teeth aching. In severer cases, however, and where the disease affects the nerve generally, or the whole face and scalp, the condition of the patient is most lamentable. The mouth is spasmodically drawn, as in palsy, so that the saliva flows over the chin and neck, or, in cases where the second branch of the *trigeminus* is affected, the teeth chatter by the clonic spasms which sometimes attend the disease, and the pain is usually most severe in the parts supplied by the infra-orbital, namely, the lower eyelid, *alveoli,* upper lips, and teeth of the upper jaw. The saliva is increased in quantity and altered in quality; for in cases in which the patient is afraid to brush his teeth, lest the paroxysm should return, the whole of the teeth of the lower jaw become so incrusted with tartar, as to form one solid mass, indicating at the same time a depraved state of the digestive organs. The eye and eyelid are likewise frequently convulsed, the conjunctiva injected, from which tears freely flow, especially on remission of the paroxysms, and the nose discharges a muciform matter. The twigs of the lachrymal branch of the *fifth nerve* going to the lachrymal gland and conjunctiva, account for this increased secretion of tears and redness of conjunctiva. To touch even the hair of the head produces pain, and sometimes the affected nerve may be traced by a red line marking its course. The recurrence of the paroxysm is uncertain; in slight cases it may return only once in a few weeks, or in a few days; but in some severe cases it will return every quarter of an hour, every five minutes, or every minute, and even every few seconds. In a few cases the paroxysms occur periodically, and at stated intervals. In general, however, the times of recurrence are uncertain: sometimes the patient is attacked with great violence many times a day for many days or weeks together, so that the disease is almost continuous; at other times it intermits for a week, a month, six months, or a year. The disease is situated nearly as often on the right as on the left side of the face; and sometimes on both sides.

Neuralgia is rare in the course of the third branch of the *fifth pair* of nerves, especially of the *auriculo-temporal* and *lingual branches.* When it does occur it generally affects the *inferior alveolar*, especially the *mental*, after it escapes from the *mental foramen*, and pain is experienced along the gums and teeth of the lower jaw, the chin, and lower lip, sometimes with salivation.

When the branches, generally of the fifth cranial nerve, are affected, the most painful points are at the exit of the ophthalmic of the superior and of the inferior maxillary branches. Next to those the frontal, and next the parietal, and lastly the occipital, although its origin is independent of that of the *trigeminal;* and whether the *trigeminal* was affected by itself, or the *occipital* nerve as well, Trousseau has always observed that "pressure made on the spinous processes of the first two cervical vertebræ always caused pain, and in a certain proportion of cases immediately brought on shooting pain in the diseased nerves.

(b.) *Brow ague, hemicrania,* or *migraine, is a combination of neuralgic symptoms, with headache occurring in paroxysms, and limited to one side of the head and brow.*

It is apt to commence in childhood and go on to advanced age, occurring in both sexes, but more often in women than in men. In women the attacks are common just before the menstrual period, or during its course. In other cases mental excitement has to do with the attack. The headache is probably due to excitement of the sensory filaments from the trigeminus to the *dura mater,* or to the *sympathetic* filaments accompanying the vessels. It increases rapidly soon after waking in the morning, with chilliness, loss of appetite, and sliminess of mouth, and at last the headache is so intense as to be almost unbearable. The eyes are extremely sensitive to light, and the ears to noise; so that the darkest rooms and most retired are anxiously sought for, and absolute seclusion is desired. The pulse is usually abnormally slow, and there may be some nausea, and vomiting may occur, of a bitter greenish fluid. Generally, no relief is obtained till after a night of sound sleep—" one night through the sheets"—awaking next morning free from pain, but much depressed.

Although the disease is not believed to shorten life, it certainly embitters existence, and is rarely recovered from entirely except in cases where it is obviously connected with the menstrual periods, when it sometimes disappears, as these periods cease with age, or, as it is commonly called, at " the change of life."

The best treatment during such attacks is total abstinence, except perhaps from fluids in small quantities at a time, and to go to bed at once, and refrain from the use of any remedies.

(c.) *Sciatica is a neuralgic affection of the sensory nerves of the sciatic plexus, composed of the fourth and fifth lumbar and first and second sacral nerves.*

It has been already noticed that it is often associated with *rheumatism* —so often that rheumatism is really the most frequent cause of sciatica, with *gout,* so as to constitute a special form of these diseases—namely, *rheumatism of the hip* (*hip-rheumatism*) and *gout of the hip* (*hip-gout*). Hence, catching cold is a frequent cause of *sciatica.*

The pain may also result from caries of the vertebræ, or tumor or vascular increase, with varicose dilatations of vessels in the intervertebral foramina, through which the nerves pass. The presence of enlarged glands in the pelvis, or of pelvic tumors, especially ovarian cysts, and fæcal masses in the sigmoid flexure may also be the source of *sciatic neuralgia.* The disease is most frequent between twenty and sixty years of age, and is more common in males, and those most exposed to changes of temperature, than in females, and those who are living in ease and comfort.

The most frequent seats of pain in sciatica are along the *posterior cutaneous nerve* of the thighs, where the posterior and outer part of the skin of the leg becomes painful—the *superficial* branch of the fibular nerve, when the pain is over the outer and anterior surface of the leg and the dorsum of the foot; the *communicans tibialis* where the pain is in the outer side of the ankle and the foot.

The most frequent points of pain are behind the trochanters, some parts of the thigh, about the knee-joint, just below the head of the fibula, just above the outer ankle, the ankle-joint generally, and dorsum of the foot (VALLEIX).

The disease rarely begins suddenly, but develops itself gradually, and slowly becomes severe. The pain is constantly increasing and deeply seated, especially near the ischiatic tuberosity. Tension of the fascia usually makes the pain worse, and the leg is generally flexed, both while in bed or out of it; and from not using the limb, it may sensibly diminish in bulk in chronic cases.

Usually *sciatica* is an obstinate disease, lasting for months or even years, and liable to relapse.

The treatment of *sciatica* consists in following out, as far as possible, the removal of the causes and circumstances already noticed as producing the disease. In the rheumatic form, the warm baths are most useful, especially the systematic treatment at Neuenahr, Wildbad, Wiesbaden, Teplitz, and Bath. *Iodide of potassium,* in large doses, and *sulphur,* especially in the form of the *Chelsea pensioner,* appear to do most good. Of other specific remedies Niemeyer mentions the induced, and still more, the constant current of electricity as rarely failing to do good. *Oil of turpentine* as an electuary, is also recommended in the following—

℞. *Ol. Terebrinth* ʒi. ; *Mell.* ℥i., of which a table-spoonful is to be taken twice a day. This is very highly spoken of by Romberg.

(d.) *Intercostal neuralgia depends on the morbid excitement of one or several spinal nerves* (6*th to the* 9*th*), *especially those which pass along the upper intercostal spaces towards the sternum, and along the lower spaces to the epigastrium.*

It is very common, and is met with more frequently in women than in men, especially in the 6th, 7th, and 8th intercostal nerves, and more commonly on the left than the right side. It is not unusual after recovery from pleurisy, and may also accompany pulmonary tubercles. It is usually also associated with hysteria and anæmia, especially in women weakened by *hemorrhagia* or *leucorrhœa.*

The following points are especially noted as commonly most painful, namely—

The vertebral point in the posterior part of the intercostal space somewhat outward from the spinous process, and on the level of the point of exit of the nerve from the intervertebral foramen.

The lateral point lying in the middle of the intercostal space, corresponding to the point of division of the intercostal nerve, and from which the nerves pass to the skin.

A third point is near the sternum, between the costal cartilages in the upper intercostal nerves; in the lower ones it is in the epigastric region, somewhat outwards from the middle line (VALLEIX).

These points are generally very sensitive to pressure (hyperæsthesia). *Hard* pressure sometimes relieves the pain. An attack of intercostal neuralgia sometimes just precedes an eruption of *herpes zoster* or *shingles;*

and in the intervals of the sharper pangs of pain, numbness, coolness, and formication are occasionally felt (WALSHE).

(e.) *Crural neuralgia occurs where the sensory filaments of the lumbar plexus going to the thigh and leg are affected.*

The pain is felt along the *anterior* and *inner* surface of the thigh, leg, ankle, and dorsum of the foot, and in the great and second toes. It thus differs from *sciatica,* which is generally along the *outer* and *posterior* surface.

(f.) *Lumbo-abdominal neuralgia affects the cutaneous nerves of the lumbar plexus, going to the lower part of the back, the nates, the anterior abdominal wall, and the genitals.* The seats of pain are commonly,—to the outside of first lumbar vertebra, just above the middle of the crest of the ilium —to the inner side of the anterior superior spine, and the termination of the nerves in the *mons veneris, vulva,* or *scrotum,* towards the *nates* and the genitals generally.

(g.) *Cervico-occipital neuralgia arises from excitement of the sensory nerves, originating from the first four cervical nerves, and affects the occiput, neck, and nape of neck.*

(h.) *Cervico-brachial neuralgia* is *located among the sensory twigs of the brachial plexus, composed of the lower four cervical and the first dorsal nerves.*

When the nerves of the brachial plexus are affected, pressure made over the spinous processes of the last cervical vertebræ give pain; so also in cases of intercostal, lumbar, or sciatic neuralgia, similar pressure over corresponding regions would also produce pain. Trousseau, therefore, makes the general statement, that " in neuralgia the spinous processes of the vertebræ are tender on pressure at a spot nearly corresponding to the point of exit of the nerve from the intervertebral foramen, and that the pain pretty frequently extends a little farther up along the vertebral column." Thus neuralgia reveals itself by acute pain when pressure is made over the spinous processes which correspond to the origin or point of exit of the implicated nerves.

(i.) *Mastodynia, or "irritable breast," is a neuralgia of the intercostal nerves, or anterior supra clavicular nerves, going to the mammary gland.*

It affects women about the period of puberty, up to the thirtieth year of life. The gland is sensitive to the slightest touch at one or more points; and severe pain occasionally shoots out towards the shoulder, axilla, or hips, and is worse shortly before the menstrual period. At the height of the pain vomiting may occur. The disease causes great anxiety, besides the pain; for the patients generally believe they are to have a " cancer of the breast," especially if *neuromata* forms in the gland. It may last for months or years without any perceptible change or improvement.

A plaster worn on the breast, if composed of anodyne elements, may soothe the pain and prevent the gland being touched by the patient, as well as give support to the breast.

Cooper recommends the *soap plaster* with *extract of belladonna,* and the following pills:—

℞. *Ext. Conii, Ext. Papaver, a a* gr. ii.; Ext. Stramonii, gr. $\frac{1}{4}$ to $\frac{1}{2}$, are mentioned by Romberg as useful.

It thus appears that in all these varieties of neuralgia there are certain characteristic symptoms, and especially—

(*a.*) *Cutaneous hyperæsthesia* at the points of exit of the nerve-trunks; most marked in cases of *intercostal, lumbar,* and *crural neuralgia.* In such cases a slight scratch of the skin, or gently rubbing the skin with a blunt end of a pencil, will cause pain of a burning or pricking kind; and in many cases the track of the implicated nerve may be followed by the tip of the finger as far as its cutaneous distribution. More rarely the reverse of this condition supervenes—namely, *anæsthesia.* It is most apt to attend neuralgia apparently of a rheumatic origin, or which is due to a slight lesion of the cord. In such cases, when the *hyperæsthesia* has lasted a long time, it is followed by *anæsthesia,* and sometimes by paralysis. In *herpes zoster* this is apt to occur; also in *sciatic neuralgia.*

(*b.*) Certain *superficial tender spots* are also characteristic of neuralgia. It is generally at the point of exit of nerves from an osseous foramen that pain is most particularly felt. Thus, in neuralgia of the fifth pair, it is over the *supra orbital notch,* where the ophthalmic branch becomes superficial; over the *infra-orbital foramen,* which gives passage to the superior maxillary branch; and over the *mental foramen,* through which emerges the inferior maxillary division of the fifth pair. But when *supra-orbital neuralgia* is intense, the point over the nasal branch is also extremely tender on pressure over the point of exit of this small nerve; and pressure over the frontal eminence also causes acute pain; and another tender point is over the *zygomatic process* in front of the ear (TROUSSEAU).

The particular form of cachexia has also a remarkable influence on the seat of neuralgia. In *chlorosis, neuralgia* is apt to affect several regions, but notably the *trigeminal nerves* and nerves of the *solar plexus.* In cases of *anæmia* from *uterine hæmorrhages* or *leucorrhœa,* the neuralgia is mainly *gastric* and *intestinal.* In *malaria,* the *supra-orbital branch of the trigeminus* is mostly implicated. In *rheumatic cachexia* it is generally the *occipital* and *sciatic nerves;* and neuralgias of rheumatic origin are generally multiple in their manifestation, and frequently alternate with articular pains (TROUSSEAU).

The exceptional cases, where pain is relieved by pressure, show that the conditions causing the pain have their site to the central side of the place pressed upon. Such cases may be relieved by division or removal of a portion of the nerve-trunk at the part pressed upon.

Prognosis.—The duration of the disease is uncertain and often chronic. In some cases it terminates after a few paroxysms; in others it lasts from one to six months; and in some cases it embitters existence during the whole period of a long life. It seldom disappears suddenly, but oscillates with a decreasing intensity, the intervals between the paroxysms gradually becoming lengthened, till at last the disease subsides. Complete cure is by no means frequent except where the cause is *rheumatic* or *malarial.*

Causes.—The remote causes of neuralgia have been already indicated. Extremes of heat or cold, or sudden changes from the one to the other, often stand in the relation of cause to this disease. It is a frequent result of impaired general health. Thus women, after profuse menorrhagia, or after childbirth, or persons recovering from fever or other severe disease, are apt to suffer from neuralgic affections. Persons who have attempted to poison themselves with arsenic suffer agonizing pains along the course of the nerves of the limbs during convalescence. Blows, or wounds, or the pressure of aneurismal or other tumors, sometimes seated in the nerve itself; irritation from foreign bodies, such as bullets or gunshot material, which have become encapsulated near a nerve; the compression of contracting cicatrices; pressure on nerve trunks by *aneurisms, exostosis, cancers, neuromata;* varicosities and over distension of veins surrounding nerves as they pass through long passages like the intervertebral foramina, are also causes of neuralgia. This last I have verified by dissection in a case of severe coccygeal neuralgia, in which the veins, surrounding the nerves passing through the intervertebral spaces were extremely varicose and irregularly dilated. It is also common with intercostal and trigeminal nerve neuralgia (NIEMEYER), and rheumatic neuralgia is probably due to such hyperæmia and œdema of the *neurilemma.* A peculiar *neuralgia* often precedes attacks of *herpes,* as of the *intercostal nerves* in cases of *herpes zoster* or shingles; of the nerves over the gluteal region and femoral region, in cases of *herpes preputialis;* and of the side of the face, in cases of *herpes labialis,* or of the skin on other parts of the face or forehead.

Neuralgia is more common in men than in women; and in women it occurs rather more frequently before thirty than afterwards, especially in those whose menstruation is irregular either as to time or quantity. Among the poor and laborious classes neuralgic complaints are numerous; and, roughly speaking, they mainly present three varieties:—(1.) *Neuralgia of the head and face;* (2.) *Neuralgia intercostalis;* (3.) *Sciatica.* The great majority of the patients are anæmic, and in a fair sprinkling of cases the anæmia is obviously connected with malaria (ANSTIE, *op. cit.*, p. 86).

Diagnosis.—The disease to which *neuralgia* bears most resemblance is *rheumatism;* but it is distinguished from it by the paroxysmal nature of the attack, and by the absence of all swelling. There are certain points of the face which, upon being pressed in the interval of the paroxysm, give pain, and so far furnish phenomena which may aid in a differential diagnosis. Other diagnostic features are of importance to be noticed, such as, if pain exists—(1.) Where the nerve emerges from the bone, as at the supra-orbitary, infra-orbitary, and mental foramina, in trifacial neuralgia; (2.) Where the nerve passing through the muscle reaches the skin; (3.) Where the nerve terminates in the skin; (4.) Where the nerve becomes very superficial, as the peroneal nerves; and lastly, (5.) Where the pain affects the spinous processes corresponding to the exit of the implicated nerves.

Treatment.—A practitioner has generally some mode of treatment which, during his practice, he considers specific in this disease. *Sarsaparilla,* the *sulphate of iron,* the *carbonate of iron, arsenic, mercury,* or the *sulphate of quinia,* have all been recommended with confidence. Bleeding, either local or general, has had its advocates, while its opponents affirm this operation to be always useless and sometimes injurious.

There can be no question that the disease has often subsided under the use of all these various remedies; but the tendency in neuralgia to a spontaneous intermission is so great that it is doubtful whether in any case medicine can be said to have cured it. Opiates are unquestionably serviceable in mitigating the sufferings of the patient, and perhaps in influencing the disease, but not to the extent generally supposed. *Belladonna,* both internally and as a plaister, may sometimes relieve the pain. *Stramonium* and *opium* have a similar temporary effect. *Belladonna,* and perhaps *stramonium,* are better than opium as a habitual remedy, and they appear to have done good occasionally. *Chlorodyne* is also to be recommended.

The remedies of most value are the diffusible stimulants, such as *sal volatile,* hot tea, quinine in a full dose, alcohol in small doses, blistering, the application of ammoniacal fluid to the skin immediately over the painful nerves, and the endermic application of a fifth of a grain of morphia. These remedies act by increasing the supply of blood to the painful nerve, and, *pro tanto,* heightening its vital energy (ANSTIE, *op. cit.,* p. 84). The most speedy way of obtaining a temporary relief is certainly the application of a local stimulant, and more especially of some volatile agent, such as mustard, or, still better, *chloroform* diluted with *seven* parts of some simple liniment, such as *belladonna* (ANSTIE). A rapidly acting blistering fluid is still more effective. But the more profoundly the general health has been affected, and especially the greater the degree of anæmia, the more necessary is it to join with the use of stimulants (both local, such as above mentioned, and general, such as the *carbonate* and *muriate* of ammonia, taken in *five* and *ten* grain doses respectively), a treatment directed to improving the condition of the blood by "food tonics," such as cod-liver oil, arsenic, or steel, or a combination of some of them, joined with the use of local stimulation, by means of frictions with dilute chloroform, and the manipulations of the scientific "*shampooer*") ANSTIE, *op. cit.,* p. 87). Dr. Radcliffe reports favourably of the *hypophosphite of soda,* and believes that the *phosphorus* of this salt acts directly as a food to the nervous tissue. The subcutaneous injection of small doses ($\frac{1}{6}$ to $\frac{1}{4}$ of a grain) of morphia in solution will give temporary relief, and is especially useful to those patients who are obliged to go through an ordinary day of labour (ANSTIE, *op. cit.,* p. 87).

When these or other general remedies have proved insufficient, recourse has been had to local remedies. The most efficient of these applications is the alkaloid *aconitina,* rubbed upon the pained part in the form of an ointment, in the proportion of one or two grains to one drachm of lard. *Morphia* similarly used, and blisters, have also often exercised a beneficial effect upon the disease. Holding the head over steam, and the warm bath, are equally or even more beneficial in cases of facial neuralgia. The belladonna plaister is also a favourite application. When the neuralgia is superficial, compresses steeped in a solution of atropia have a good effect. For this remedy Trousseau recommends the following formula:—

℞. *Atropiæ sulphatis,* gr. v.; *Aquæ distillatæ,* ℥iii.; *solve.* Renew the compresses several times in the twenty-four hours. Continue them for at least an hour each time, and cover them with oilskin, to prevent evaporation.

When general and local applications are unsuccessful, the cause may be sought for in a diseased tooth or stump, and in a *very few* instances an exostosis of the stump has been discovered, removed, and the disease cured. Sometimes, however, even when the patient submits to have every tooth in his head drawn, no relief or benefit may result. The division of a nerve is sometimes had recourse to; but even this operation is very uncertain.

Where the causes of neuralgia cannot be removed, modes of treatment which greatly modify the change of tissue and the nutrition of parts are often beneficial. Chief among these is the use of electricity; and Niemeyer gives the results of his experience in the following propositions:—

"1. In treating neuralgia with the reduced current, it is best to employ the metallic electrodes known as the electric brush. While one electrode, containing a moistened sponge, is held in one of the patient's hands, or against any part of his body, we stroke the brush along the course of the affected nerve; if there are any specially painful points, we allow the brush to remain over them rather longer (*electric moxæ*)."

"2. Many cases of neuralgia, which had been previously treated without benefit by the most varied remedies, were completely and permanently cured in from twelve to twenty applications, or even sooner. In other cases no benefit or cure was effected."

"3. The first sitting shows whether the neuralgia can be cured by the induced current. We can only expect a cure where the pain is decidedly relieved, or entirely disappears, immediately after the first electrization, even if it should only be for a short time. If this temporary result do not take place, the continuation of the treatment will also prove ineffectual."

"The application of the induced current as above directed is very painful; and it is only after the patient has actually experienced benefit that he suffers it with patience, and even then he moans and whimpers during the application."

The constant current is far more effective than the induced in neuralgia. Some cases that have been treated without result by the induced current have been cured by the constant, but never by the reverse. Niemeyer places both poles along the affected nerve, and, without attending to the course of the current, holds the zinc pole on the most painful part, and on those parts where the nerve approaches nearest to the surface, as at the *supra* or *infra-orbital foramen*, or at the *zygomatico facial foramen*, or at the *sciatic notch*. If it be possible to get the nerve between the poles, as in the cheek or nose, he introduces one pole into the mouth or nose to the point whence the pains radiate, while the other is placed at the corresponding point on the skin.

"At first the application of the constant current is not particularly painful; but an unpleasant, burning, piercing pain soon commences, and gradually increases; and where the number of electric elements is large, it may become unbearable.

"The changes induced in the skin at the point of application of the constant current, if the electrodes be applied for a length of time, are far greater than those caused by the induced current. They not only consist in a lively erythema, a decided swelling of the skin, and an increase of the subjacent tissue; but papules and blebs arise on the skin, particularly at the positive pole. If the action continues long, the surface of the elevations sloughs off. These changes take place not only at the point of application, but occur equally, or at least similarly, in the deeper parts, as is shown not only by the increase in volume of the subcutaneous tissue and muscles, but also by the following experiments (ERB); which show a great deal about the mode of action of the constant current in neuralgia and other neuroses, as well as in some diseases of the muscles and joints. If we cross the fore-arms, placing the volar surfaces in contact, and apply the electrodes to their dorsal surfaces, there will be reddening not only of the parts to which the electrodes are applied, but of the corresponding points on the anterior surfaces. Generally, relief immediately follows the application of the constant current, just as it does that of the induced current; but occasionally the pain is at first increased, and that should not always induce us to stop the treatment. There seems to me no doubt that the curative action of the constant current, in most cases of neuralgia, is to be explained by the modification of the circulation, endosmosis, or change of tissue in the diseased nerve, its neurilemma, or the parts around. This "catalytic action" may result from the chemical disintegration induced through the nerves, or the attraction of the constituents of the nutrient fluids toward the pole, or it may occur in some other way" (NIEMEYER).

Narcotics, in the form of hypodermic injections of solutions of morphia, are of great value, and it is immaterial whether the injection is made near the seat of pain or at a remote distance from it. The hypodermic syringe and the morphia solution are now almost as indispensable accompaniments of the physician as the stethoscope and thermometer. Its use, however, is attended with similar bad results as the taking of opium in any other form. The dose must be gradually increased, and the remedy cannot be given up. If it is omitted the patient feels dull, weak, uncomfortable, nervous, and trembles from head to foot, as after an alcoholic debauch.

One-sixth to *one-third* of a grain of *morphine,* injected hypodermically, is the usual palliative remedy for neuralgia.

Friction of the skin with *veratrine ointment,* in the proportion of gr. iv. —gr. x. of *veratrine* to ℥i. of *lard;* or with *aconite ointment,* in the proportion of *one grain* of *aconite* to one drachm of *lard.*

ANÆSTHESIA.

LATIN EQ., *Anæsthesia;* FRENCH EQ., *Anesthésie;* GERMAN EQ., *Anæsthesie;* ITALIAN EQ., *Anestesia.*

Definition.—*The term anæsthesia implies a diminution or complete loss of* *function (i.e., of sensitiveness to external impression) in a sensory nerve, from its nervous force being reduced or destroyed—paralysis of sensation.*

Pathology.—Sensibility admits of varying degrees, being different at different parts of the cutaneous surface, as proved by the experiments of Weber (see page 137). His mode of experimenting may be adopted, as already noticed, as a practical means of diagnosis in paralysis of sensation. It occurs when there is nutritive disorder capable of destroying the irritability of the peripheral nerves, as well as when their connection with the brain and spinal cord has been interrupted mechanically (NIEMEYER).

Peripheral is distinguished by Niemeyer from *central anæsthesia* mainly by the following points:—

(1.) Extension of the anæsthesia and its complication or non-complication with motor palsy. If the anæsthesia be confined to one side, and be combined with hæmiplegia, it is probably cerebral. If, however, it extend over the lower half of the body, and be combined with paraplegia, it is probably spinal. The co-existence of palsy of the face with facial anæsthesia indicates a central cause of disease, while anæsthesia with normal motion indicates peripheral origin.

(2.) Reflex symptoms may be present in *central anæsthesia,* but they are entirely absent in *peripheral anæsthesia.*

Two kinds of sensibility have been shown to exist in the skin and mucous membrane round the natural orifices: the one is *tactile sensibility,* by which *form, temperature, consistence,* and such like qualities of a body are distinguished by touch; the other kind of sensibility appreciates the sense of pain. The first has been distinguished by the term *anæsthesia,* the latter by the term *analgesia;* and the skin may lose the sense of pain and still retain the power of touch. The finger may then be felt when rubbed over the skin, but the skin is then insensible to pricking or pinching; but when common tactile sensibility is lost, the sense of pain is also lost, except in some instances of centric reflex paralysis (BEAU).

Anæsthesia most commonly occurs as an immediate antecedent of motor palsy, or coincident with it, and also exists in various parts of the body, independent of paralysis of motion. It varies in extent from a mere numbness of the parts to a complete loss of sensation. The cutaneous nerves are those most frequently affected, and from this cause the disease most usually advances to the integuments of a portion of the trunk, or of an arm, or a leg, or some portion of the extremities, and also the whole face or parts of the face.

The following remarks by Dr. Clymer are of much practical value:—

"The function of sensation depends upon the concurrence of three sets of organs, anatomically continuous, yet to a certain extent physiologically distinct. (1.) The nerve-extremities, which receive an impression; (2.) The afferent nerve-fibres, which carry the impression to the centre; (3.) The centres, which receive the impression and transform it into a sensation. Thus the three factors of sensation are, the sensitive nervous extremities, the sensory conducting filaments, and the sentient portions of the nervous centres. If the integrity of any one of these is affected, there must result diminution or loss of sensation. If perception in the sensorium is destroyed, the nerves may transmit the impression to the sentient centres, but there is no sensation. This is one cause of anæsthesia, and occurs in certain lesions of the brain, as cerebral hemorrhage, softening, and tumors; or the function of the organ may be temporarily in abeyance, from the effect of certain agents, as chloroform or narcotics. Another way in which anæsthesia is produced is by interruption of the continuity of the nerve-trunk, by section, or compression, or atrophy, or tissue-degeneration. A third mode is, whilst both the nerve-centres and the nerve-trunks are sound, the sentient nerve-extremities have undergone some morbid change, and the impression is not received, as in insensibility of the skin from the application of intense cold, blindness from disease of the retina, loss of the sense of smell during coryza, and certain cutaneous eruptions. It sometimes happens as the result of reflex action in visceral disorders, and after mechanical injuries (BROWN-SÉQUARD, S. W. MITCHELL). This pathogeny of anæsthesia being admitted, can we make a practical application of it, and determine clinically the seat of the morbid condition, and refer it to a given lesion of the brain or medulla, or of the nerves themselves? Is the anæsthesia central or peripheral?

"(1.) *Anæsthesia resulting from cerebral disease* is usually of large extent, and may involve the whole body; generally it is unilateral, and on the side opposite to the brain lesion; voluntary motion is lessened or abolished, the reflex movements are commonly intact, intelligence is disordered, and the nutrition of the paralyzed parts impaired. (2.) *Anæsthesia consecutive to spinal disorder* is mostly less general than that caused by disease of the brain; it is as a rule bilateral and symmetrical, but when unilateral it is on the same side as the affected column of the medulla (though Dr. Brown-Séquard admits crossed spinal paralysis); voluntary motion is lessened or destroyed; reflex functions may be preserved or absent, according as the medulla is or is not sound below the site of the lesion; nutrition of the affected parts is often greatly impaired. (3.) *Anæsthesia depending on some lesion of the sensory nerves* is often very limited, its site corresponding exactly to the distribution of the nerve; voluntary motion is intact if a nerve exclusively sensory is involved, or if it be a compound nerve, the paralysis is limited to a single group of muscles, reflex movements are constantly wanting where the anæsthesia is complete, and the state of the nutrition of the affected part is variable, but often good."

The most important form in which insensibility comes under the notice of the practical physician is that known as FACIAL ANÆSTHESIA. It consists in a deficient or entire loss of sensibility in the parts supplied by the fifth pair of nerves.

Symptoms and Diagnosis of the Seat of Lesion.—(1.) The more the anæsthesia is confined to single filaments, the more peripheral is the seat of lesion. (2.) If the loss of sensation affects a portion of the facial surface, together with the corresponding cavity of the face, the disease may be presumed to involve the sensory fibres of the fifth pair before they separate, or one of the main divisions after its passage through its cranial foramen. (3.) When the entire sensific tract of the fifth nerve has lost its sensation, associated with derangements of the nutritive functions in the affected parts, the Gasserion ganglion, or the nerves in its immediate vicinity, may be the seat of the disease. (4.) If the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be presumed that the lesion is situated at the base of the brain (ROMBERG).

The symptoms may develop themselves gradually, or may come on suddenly, and are not unfrequently preceded by neuralgia; and owing to the differences in the seat of lesion, what may seem to be a permanent symptom in many is absent or less marked in others. It is frequently complicated with facial palsy of the *portio dura* of the seventh nerve. In a well-marked case the symptoms may be generally stated to be loss of tactile sensibility of the parts supplied by the nerve—namely, the integuments of the cheek and side of the head, the eyelids, conjunctiva, tongue, Schneiderian membrane—accompanied by loss of taste on the side of the tongue which is affected, frequently by loss of smell and hearing, and by inflammation of the eye, terminating in ulceration of the cornea, and by no means uncommonly in total disorganization of the globe. Paralysis and wasting of the deep muscles of mastication are also sometimes associated. The real nature of the affection may escape notice if the attention is directed solely to any one prominent symptom. The disease makes its onset in two or three ways. It is often sudden, preceded by a slight perversion of sensibility, or by a tingling sensation, frequent attacks of headache, and pains in the occipital region and side of the face. Dimness of vision is so prominent a symptom in some cases that loss of sensibility may not at first attract attention. Of twenty-four cases, vision was affected in fifteen, hearing in nine, and smell in six (Dr. J. B. Cowan in *Glasgow Med. Journal*, No. II., July, 1853).

There are two important features in facial anæsthesia worthy of special notice—namely, 1st. That the parts do not waste as in muscular palsy; 2d. That though the parts are insensible to touch, they still remain sensible to changes of temperature, and to pain arising from inflammation in the nerve itself.

Causes.—The anæsthesia may result from disease—(1.) Of the cerebrum, where the fifth nerve takes its origin; (2.) Of the nerve within the cranium; (3.) Of the nerve after it has emerged from the cranium; and according to the seat the symptoms vary.

The disease may be a consequence of some injury, such as the extraction of a tooth, as frequently happens to the submental branch, which conveys sensation to half the lower lip.

Dissections show that condensation, atrophy, softening, and the pressure of tumors are the morbid conditions out of which the anæsthesia springs. So varied are the sources whence the anæsthesia results, that its treatment can only be palliative, and directed towards mitigation of the symptoms. Spontaneous cures are said not to be unfrequent. The local applications, such as blisters, leeching, and cupping, are said to have been sometimes useful, combined with the administrations of purgatives, and the continuous or interrupted current of electricity.

Some other forms of anæsthesia are thus briefly mentioned by Dr.

Clymer in the following words :—"Those of the special senses are loss of sight from *amaurosis,* nervous deafness, *paracusis,* loss of smell, *anosmia,* and loss of taste, *aguestia. Cutaneous anæsthesia,* as has been shown, may be centric or peripheral, and vary from slight numbness to complete abolition of sensation, with no sense of pain or appreciation of heat (*thermo-anæsthesia*), and be bilateral, or unilateral, or limited to a single spot of some extent; sometimes its degree varies in different parts of the body. It is frequently associated with anæsthesia of the mucous membrane, generally the conjunctival, and with muscular anæsthesia. Besides organic lesions of the brain, medulla, and nerve-trunks, it occurs in functional disorders of the centres, as hysteria, chorea, and occasionally in insanity (AXENFELD); in visceral disease (the reflex form); after excessive fatigue (SANDRAS); after the local application of cold and certain irritating substances, as lye (ROMBERG); from the effects of certain toxic articles, as lead, arsenic, bromide of potassium, sulphuret of carbon, nitrous oxide gas, sulphuric ether, chloroform, in the course of and after septic diseases, and in asphyxia (FAURE). *Muscular anæsthesia* has been defined to be a diminution or loss of the muscular sense. There is abolition of muscular consciousness; the patient, if his eyes are withdrawn from the limb, is unable to tell its position, whether it is flexed or extended, abducted or adducted, and may try to extend a limb, which is already stretched out; there is no appreciation of the amount of force required to meet or overcome any opposing force; the weight of the limbs is not felt; and the degree of density of various bodies cannot be estimated. The perception of muscular contraction is totally or measurably gone. Muscular anæsthesia, when fully developed, is even more unequivocally shown by the absence of sensibility when the muscles are pinched, squeezed, or shampooed, or an electric current passed through them, or a sharp instrument pushed into them. It may be unaccompanied by loss of the function of motility, shown by the ability to execute all necessary movements when the eye is on the limb, and by the amount of muscular vigour that can at need be exhibited, and by the feeling of lightness, and the absence of all sense of fatigue, in the extremities, so opposite to the heaviness and weariness of muscular paralysis; or there may be motor palsy, as in hysterical paralysis; or it may be accompanied by insufficiency in the power of co-ordinating voluntary movements. It frequently coexists with cutaneous anæsthesia. Landry speaks of numbness and formication as precursory phenomena, but they probably really belong to the primary disorder of the nervous centres. It is more fixed than cutaneous anæsthesia, and rarely presents, even in hysteria, those fitful mutations of site so common in the latter. The prognosis depends upon the pressure of centric lesions" (BEAU, BROWN-SÉQUARD, SZOKALSY, LAUNDRY, BELLION, VULPIAN, YELLOWLY, BRACH, DUCHENNE (de Boulogne), BOURDON, AXENFELD).

HYPOCHONDRIASIS.

LATIN EQ., *Hypochondriasis;* FRENCH EQ., *Hypochondrie;* GERMAN EQ., *Hypochandrie;* ITALIAN EQ., *Ipocondriasi.*

Definition.—*Some disturbance of the bodily health, attended with exaggerated ideas or depressed feelings, but without actual disorder of the Intellect.*

Pathology.—It has been usual to regard hypochondriasis as a disorder of the Intellect, and the depressed feelings exist as one form of melancholia.

Regarded as a substantive disease, the predisposition to it is very slight in childhood, and is greatest between the ages of twenty and forty, and far less in females than in males. It may be congenital, or is developed as a result of such debilitating influences as sexual excesses, onanism, digestive disorders, dyspepsia, want of fresh air, and inactive modes of life.

Symptoms.—The development of the disease is gradual, and is expressed by an indefinite sense of illness; and Niemeyer gives the following very graphic word-picture of the disease, as distinguished from that form of disordered Intellect known as *hypochondriacal melancholia:*—

"The more profoundly the disease takes root, so much the more assiduously does the patient endeavour to discover the cause of his indisposition. He scrutinizes his tongue, his stools, his urine; he counts his pulse and handles his abdomen. Every trifling irregularity which he perceives, the slightest irritation, the faintest coating of his tongue, a transient colic, an insignificant cough, are all of the utmost importance in his eyes; not because he suffers more than any one else from such symptoms, but because they seem to him to afford a clew to the nature of the grave and obscure imaginary disease. To-day he may dread an apoplexy, to-morrow he may think that he has an ulcer of the stomach; at other times he may imagine his heart diseased, or that he is consumptive, or afflicted with some other serious malady of a character corresponding to his sensations of distress. He studies all the "medical advisers" and other books of "domestic medicine;" but, instead of deriving comfort and aid from them, merely finds out new diseases, by which he immediately imagines himself afflicted. As the disease gains mastery over the patient, his belief as to his condition becomes more and more biassed and incorrect. Argument is useless, as it cannot relieve him of his feelings. A few hours only may have elapsed since we last saw the patient. We then may have spared neither time nor trouble in explaining to him that his condition was perfectly free from danger, when a messenger arrives from him, begging us to come in haste, that some serious change has taken place, and that his condition has become a most critical one. In other instances, especially when the patient really has some insignificant disorder, the hypochondriac is not so apt to change his opinion as to the nature of his disease, but sticks to a belief in one, and cannot be dissuaded from it. He is not like other patients, satisfied with simply complaining of pain, oppression, or fever; partly, because he really feels worse than other people, and, in part, because he is convinced that the doctor "makes light of his sufferings." Hence he exaggerates, and often exhibits the utmost fluency in description of his infinite suffering. In spite of the severity of these imaginary symptoms, however, they by no means despair of recovery; hence hypochondriacs seldom attempt suicide, and never weary of seeking fresh medical advice and of trying new treatment. Sometimes their hopes are so much in the ascendant, and are so productive of happiness to them, that, for a while, in spite of their sense of illness, they are cheerful and in even high spirits. Such intervals, however, are usually very brief, and occur most frequently immediately after the engagement of a new medical man or the commencement of a new "cure." The old mood very soon returns. The false realizations of their sensations, and the erroneous ideas of the patients as to the condition of their own bodies, which we sometimes see in hypochondriasis, are a genuine delirium. Like other insane ideas in other forms of psychical disorder, this proceeds from morbid bias of the mind, and is to be regarded as an attempt to clear it (GRIESINGER). Hallucinations—"sensations originating inwardly"—also arise in hypochondriasis, owing to this sense of illness, and to the attempts of the patient to account for it. Thus the idea that the heart is standing still, or that a limb is withered, or that the body is putrefying, although it is not the result of a genuine sensation incorrectly interpreted, yet it is so vivid that the patients cannot distinguish it from an impression actually furnished by the senses; and they really believe they can feel that the heart does not beat, that the skin is dried up, or that they can smell the putrid emanations from their body. In spite of their mental aberration and morbid fancies, most hypochondriacs are able to transact their business, and to take care of their house and family; and this is the reason why hypochondriasis, usually, has not been regarded as a disease of the mind, being looked upon rather as a nevous disorder, a custom with which we have complied. In the worst forms of the disease, the patient loses all interest for matters which do not bear upon the state of his health. He becomes abstracted, forgetful, and negligent of his affairs, gives himself no further concern about his family, and often remains idle in his bed for years. It is often a long time before his nutritive condition begins to suffer. Gradually, however, especially in bad cases, the patient grows thin, and acquires a sickly appearance, and derangement of the secretory and digestive functions arises.

Treatment.—It is no use to dispute or attempt to reason with hypochondriacs; and the necessary therapeutic measures for his relief must vary with the nature of the individual case, while the aim of psychical treatment should be "the diversion of the attention from the sensory to the motor and intellectual spheres."

SECTION VIII.—DISORDERS OF THE INTELLECT.

The term insanity has not been recognized in the new nomenclature; and the subjects hitherto described under that popular term are now comprehended under the general heading of "Disorders of the Intellect," under which six varieties are specified, namely—*Mania* (acute and chronic), *Melancholia, Dementia, Paralysis of the Insane, Idiocy, Imbecility.*

Many theories have been propounded to explain the nature of "*Disorders of the Intellect,*" as embraced by the term "*Insanity.*" These may be resolved into two, as at present entertained, namely,—(1.) The *metaphysical, functional,* or *spiritual* theory; and (2.) The *cerebral* theory.

The "functional" or "spiritual" theory, which inculcates the belief that these disorders imply an affection of the immaterial principle, is at variance with all reasoning. Such a belief is in direct opposition to positive, well-recognized, undeniable data. It is an almost universal belief that the brain is the material instrument by which that thinking principle, the Mind, manifests itself, whether it be by the unseen phenomena of *Conception, Judgment, Reasoning,* and *Instinct,* or by the more obviously expressed phenomena of *Volition, Emotion,* and *Sensation.* "We are ignorant of any mental manifestations except through the brain. We know nothing of the actions of the *mind* except through the interposition of a material organ; and that no mental process ever emanates except from such an organism, or is received except through a similar piece of machinery. *Spirtualism* is not in vogue with us as a profession" (WILKS). To consider, then, those *subjective* phenomena which collectively, in their various manifestations, constitute "Mind"—an immaterial essence—as liable to disease apart from all derangement of the material organ, the brain—the instrument with which it is so closely and indissolubly united—is to believe in a most incongruous, unphilosophical, unphysiological doctrine.

The more consistent theory is that which is known as the *cerebral theory;* and which is now entertained by most of those eminent physicians who have made "Disorders of the Intellect" a special study and subject of treatment. Among the most able exponents of this theory may be mentioned Drs. Forbes Winslow, Bushnan, Conolly, John Charles Bucknill, Daniel H. Tuke, Morell, Schroeder van der Kolk, Skae, Griesinger, Maudsley, Laycock, and Lauder Lindsay; and from their lucid descriptions of the Insane I have drawn up the following account of "Disorders of the Intellect," sufficient for the scope of this text-book.

It has been objected that the new nomenclature discarding the term "Insanity," tends to ignore the idea of disease as associated with madness; but it does not necessarily do so. The external manifestations of disease are expressed by the "Disorders of the Intellect;" and these disorders imply *cerebral lesion* with as much distinctness as the term Insanity can do.

The belief which this so-called *cerebral* theory inculcates is, that the instrument, through which the phenomena of Mind are expressed is the part diseased;—that the encephalic nervous textures are primarily implicated. And as it is consistent with the pathology of disease in every other organ and texture of the body that the part may be diseased without our means and instruments of research being able as yet to demonstrate such morbid state to the senses, the diseased state being expressed through one or more disordered functions of the frame, so is it with the brain. It is an organ of such exquisite delicacy, both of structure and of function, that important and extensive structural changes may, and often do exist, which neither our naked eye-sight nor our touch can appreciate, and which can only sometimes be demonstrated, if at all, by various complex methods of research.

In those cases where the manifestations of the mental phenomena are simply disordered and perverted, but not abolished, it is consistent with the known pathology of disease in other parts to expect *the very slightest* structural change, such as may rather be expressed as a *tendency* to those morphological lesions which occur between the blood and the elements of texture, and which are only manifested through vital phenomena—insidious, because unseen, in their approach—and often inappreciable after death to the most experienced observers. Analogous to this morbid state in the nerve-tissue of the brain is the local morbid state which attends that complex morbid process known as *inflammation,* and to that essential part of it to which Virchow gives the name of *parenchymatous.* It is expressed in the altered vital morphological phenomena between the blood and the minute elements of tissue, and which, in some textures, has been appreciated by the microscope as a mere "cloudy swelling" of the minute elements of tissue, such as may be seen in cartilage and in some of the so-called non-vascular tissues.

The phenomena of disorders of the Intellect also furnish most conclusive data which show that the brain-tissue is impaired, especially in those cases where the manifestations of disorder obviously yield to remedies.

Those pathological doctrines are, moreover, always to be regarded with distrust, whose tendency is to hold out no hope of cure. Such is the tendency of the *metaphysical* theory of insanity.

Believing in the *cerebral doctrine,* and acting upon it, the prospects of cure are hopeful, *if the case is diagnosed early, and remedies are judiciously applied.*

"If cases of Insanity are brought within the sphere of medical treatment in the earlier stages, or even within a few months of the attack, Insanity, unless the result of severe injury to the head, or connected with a peculiar conformation of chest and cranium, and in hereditary diathesis, *is as easily curable as any other form of bodily disease for the treatment of which we apply the resources of our art*" (Winslow).

It is a serious error, on the one hand, to act upon the belief that *physic cannot make a man think otherwise,* when "one man thinks himself a king, another a cobbler, and another that he can govern the world with his little finger." On the other hand, it is equally erroneous to act upon the belief "*that no man was ever reasoned into Insanity or reasoned out of it.*" It is only by a proper combination of *medical* and *moral treatment* that the first manifestations of *Disorders of the Intellect* are to be controlled and ultimately effaced.

"The existence of so vast an amount of incurable Insanity within the wards of our national and private asylums is a fact pregnant with important truths. In the history of these unhappy persons—these lost and ruined minds—we read recorded the sad, melancholy, and lamentable results of either a total neglect of all efficient curative treatment *at a* *period when it might have arrested the onward advance of the cerebral mischief*, and maintained reason upon her seat, or of the use of injudicious and unjustifiable measures of treatment under mistaken notions of the nature and pathology of the disease. . . . Experience irresistibly leads to the conclusion that we have often in our power the means of curing insanity, even after it has been of some years' duration, *if we obtain a thorough appreciation of the physical and mental aspects of the case, and perseveringly and continuously apply remedial measures for its removal*" (Winslow in *Lettsomian Lecture*, pp. 59, 61).

The testimony from *morbid anatomy* which illustrates the pathology of *disorders of the intellect*, and which more directly supports the cerebral theory, is that which has resulted from observations made upon the *bulk* of the brain, upon its *absolute* and *specific* weight, and upon the relative bulk and weight of the grey and white substance of which it is composed. The original observations of Dr. Bucknill with regard to the *insane*, and of Dr. Sankey with regard to the *sane*, furnishing especially valuable data for comparison, are those which must be regarded as the initiative of observations from which we hope yet to learn much. Dr. David Skae, of the Morningside Asylum, near Edinburgh, and Dr. Boyd, of the Somerset County Lunatic Asylum, have more recently confirmed some of these observations, and have in some measure extended them. Such observations show generally—

(1.) That the absolute weight of the brain is slightly increased in the insane—a conclusion which is also consistent with the fact, established especially by Bucknill that in many cases of disorderly Intellect the absolute *size* of the brain is materially diminished relatively to the capacity of the cranium.

(2.) This increase in absolute weight appears to depend chiefly on an increase in the weight of the *cerebellum* compared with the *pons Varolii*, the *medulla oblongata*, and the *cerebrum*. The general result is that the *cerebellum* in the insane is heavier in relation to the *cerebrum* than it is in the sane.

(3.) Dr. Boyd records the singular fact that almost invariably the weight of the left cerebral hemisphere exceeds that of the right by at least the eighth of an ounce, (Royal Society, Feb. 28, 1861; also *Med.-Chir. Trans.*, vol. xxxix.)

(4.) On arranging the weight of the brain according to the form of insanity under which the patients laboured, the following average results were obtained:—

AVERAGE WEIGHT OF THE ENCEPHALON.

In *Mania*, ...	54 ounces	$11\frac{1}{3}$ drachms.
In *Monomania*, ...	51 ,,	$11\frac{3}{7}$,,
In *Dimentia*, ...	50 ,,	$5\frac{4}{10}$,,
In *General Paralysis*.	49 ,,	$12\frac{8}{29}$,,

The absolute weight, therefore, is greatest in *mania*, and least in the *general paralysis of the insane;* while also the cerebellum decreases similarly in weight through the same series, with the exception, that in the *general paralysis of the insane* it presents the highest average. Generally it appears that in cases of *acute mania* (which is a form of *disorder of the*

Intellect generally of comparatively short duration), there is the smallest amount of increase in the relative weight of the cerebellum; while in *general paralysis*—a disease of more prolonged duration—the greatest increase takes place.

(5.) The results of observations on the specific gravity both of the grey and white matters of the brain show an increase in the *insane* compared with the same textures in the *sane* (SANKEY, SKAE).

(6.) It is consistent also with the records of these observers that the *mode of death* has an influence upon the specific gravity; and, generally, it may be stated that when cerebral symptoms are well marked, such as by convulsions, strabismus, and the like, and when the case terminates by *coma* or by *apnœa*, the specific gravity is higher than when the symptoms are associated with exhausting disease, such as phthisis, and when the case terminates fatally by *anœmia* or *asthenia.* The high specific gravity of the grey and white matter in the former class of cases averaged 1·041, while the average specific gravity of the whole brain in similar cases, as observed by Dr. Bucknill, varied from 1·040 to 1·052; the average specific gravity of the whole healthy brain being only 1·036.

(7.) Dr. Bucknill's more recent observations show the most essential change to consist in shrinking of the substance of the brain, with degeneration of the nerve-cells, or a relative atrophy of its substance by a deposit of inert matter (*Med.-Chir. Review*, Jan. 1855).

(8.) On comparing the specific gravity of the grey substance in the different forms of mental disease, the lowest appears to occur in cases of *dementia*, where, however, it is 0·003 above the average in the *sane.* The next highest specific gravity occurs in cases of *melancholia*, the next in *general paralysis*, the next in *mania*, and the highest in *epilepsy.* In some of these, however, and probably in all, the *mode of death* appears to influence the specific gravity. For instance, in the *paralysis of the insane* terminating by *coma* or *apnœa*, the specific gravity of the whole brain has been 1·040; while in similar cases terminating by *syncope* or *asthenia*, the specific gravity of the whole brain has not exceeded the average, nor gone beyond 1·039. The average specific gravity of the white substance is lowest in cases of *mania*, next in *dementia*, higher in *general paralysis*, higher still in *monomania*, and highest in *epilepsy* (*Edin. Monthly Jour. of Med. Science*, October, 1854).

The general results of the more crude examinations of the cranium and its contents in cases of Insanity, in this and other countries, show that, in a very large proportion of cases, there are found some degree of thickening and opacity of the arachnoid, serous effusions into the subarachnoid tissue, into the arachnoid sac and ventricles of the brain, or of serum with lymph more or less gelatiniform, accompanied sometimes with increased, sometimes with diminished vascularity of the brain and its membranes, thickening and adhesion of *dura mater*, gritty state of the *pia mater*, turgescence of cerebral vessels, or of *puncta vasculosa.* Lesions are also more frequently found at the anterior and superior portions than at the base of the brain. Some cases prove fatal by a series of apoplectic seizures when the blood-vessels are extremely atheromatous. In that specific form of paralysis supposed to be peculiar to the insane there also appears to exist a peculiar softening of the grey matter, not indicated by any change appreciable to the eye, but by layers of the grey matter stripping off easily with the membranes, to which it often adheres; by the readiness with which the grey matter is broken up by a stream of water; and by changes in the contents of the brain-cells, as observed microscopically. The morbid state of the brain in the insane may extend to a considerable depth into the grey matter composing the hemispherical ganglia; but the whole of these ganglia are generally more or less implicated, in conjunction with the tubular fibres passing from the hemipsheres through the vesicular neurine. Dr. Lindsay has noticed œdematous softening of the central parts of the brain in certain of the cases he has examined. Such changes are generally associated with great vital and nervous depression. In a valuable paper "On the Blood in the Insane," Dr. Lindsay has shown that although the blood is in some cases characterized by particular morbid states, such states are nevertheless not peculiar to insanity. He is of opinion that, save in extreme and exceptional cases, there are no specific abnormalities of the *urine, blood, post-mortem appearances, craniological measurements,* or of *facial characteristics* in the *insane* as compared with the *sane.*

There is evidence to show, however, that while the whole morbid condition of the blood in disorders of the Intellect is not peculiar to such disorders, the general fact exists, that there is a diminished quantity of fibrine. A dyscrasial condition in the more severe and hopeless cases of mania seems evidenced also by the peculiar blood tumors of the ear, so often seen in that form of disorder of the Intellect.

With regard to the urine, Dr. Lindsay remarks, that his results differ widely from those published by Dr. Sutherland and other eminent authorities, especially as regards the view concerning the elimination of *phosphates,* and the connection of such elimination with Insanity (*Journal of Psychological Medicine,* July, 1856, p. 488). The general result of his experience is, that there is virtually no special or distinctive Pathology of disorders of the Intellect; nevertheless, the more general acceptation seems to be that there is some relation of phosphate elimination with its various forms. The chemical composition of the urine of the insane, as determined by Dr. Adam Addison, shows,—

(1.) That the quantities of the urine, of the *chloride of sodium, urea, phosphoric,* and *sulphuric acids* excreted during the course of a maniacal paroxysm, occurring in *acute mania, epilepsy, general paralysis, dementia,* or *melancholia,* are less than the amounts secreted in an equal time in health.

(2.) That in chronic melancholia the quantities of the same substances are reduced below the mean, and sometimes below the minimum, of health.

(3.) That in *idiocy, dementia* (*paralytic* and *common*), the same substances range above and below the normal mean of health; in some cases the amount of *phosphoric acid* is greater than the mean according to weight, but in the majority of cases it ranges between the minimum and the mean found in healthy adult men (*Brit. and Foreign Med.-Chir. Review,* April, 1865). The general conclusion seems to be that there is an excess of phosphates in *mania,* and a deficiency in *dementia.*

Drs. Tuke, Rutherford, and Skae describe a new lesion seen by them in the brain of an insane person, of which they have given a description, with drawings, in the *Edinburgh Medical Journal* for September, 1868. It consists of spots or areas of tissue, surrounded by well defined fibrous-looking walls. These spots occur chiefly in the white tissue, a single spot only having been seen in the grey. The first stage of this lesion seems to be the appearance of somewhat opaque, white, molecular material, which gives rise to a *cloudiness* of the tissue. It does not seem to have any close relation to blood-vessels, as it pushes them aside; and there was no evidence of proliferation of the nuclei of the *neuroglia*, or of the vascular walls. The molecular material, while it leaves the fibres of the connective tissue (*neuroglia*) intact, takes the place of its nuclei and of the nerve-tubes. The molecular matter of the spots seems to be the result of active growth taking place either within itself or in the periphery of the tissue. The lesion is the result of a kind of growth which leads to the formation of molecular material, which, by encroachment, causes the disappearance of nerve-tissue; and it differs from that lesion which Rindfleisch has described as the essential change in grey degeneration.

It is necessary to bear in view that crude morbid appearances occurring in the Insane are also found more or less frequently in the brains of persons who have died of other diseases, and without any manifestations of mental impairment of the nature of insanity.

Causes.—In modern times disorders of the Intellect are unhappily of frequent occurrence, and they have been supposed to be extending in proportion to the degree of civilization. It is made to appear, from an interesting paper by Dr. Winslow in *The Journal of Psychological Medicine* for July, 1857, that diseases of the brain and nervous system are not only of more frequent occurrence, but that a certain unfavourable type of cerebral disorganization tends to develope itself in the present day. This type of cerebral disorder may be characterized as being—(1.) Insidious in its approach; (2.) Tending to that form of Insanity which frequently terminates by suicide; or (3.) Leads at an early age to softening of the brain, at an age—the prime of life—when the Intellect ought to be in an active and vigorous condition of exercise and of health. This statement can be taken for what it is worth, seeing that it is based on no definite data or statistics, and is therefore a mere impression or matter of opinion.

The remote causes of disorders of the Intellect are of a Moral or Physical nature. Of the patients principally admitted into the different hospitals of France, Italy, and Belgium, about *one-tenth* have their insane state attributed to falls, blows on the head, the abuse of mercury, or other physical causes not determined. The remainder of the cases have their disorders ascribed to Moral influences—such as religion, or as having been crossed in love, jealousy, family disputes and family cares, reverses of fortune, wounded pride, disappointed ambition, anger, fright, excess of study, libertinage, and drunkenness.

It is sometimes difficult, if it is always desirable, to separate Physical from Moral causes in our speculations on the Etiology of Insanity. The action of Moral influences in producing insanity is so striking that the passing events of the day often impress upon the disease its peculiar characteristic. When magic and witchcraft were believed in, Europe was overrun with persons who supposed themselves possessed by the devil. When the Pope was at Paris, that singular event caused many religious monomaniacs—a form of disorder of the Intellect which, says Esquirol, shortly after disappeared.

The causes of disorders of the Intellect are of a nature producing in the patient, in the first instance, *emotional changes* only, either by the sudden and violent agitation of the Passions, or by the long-continued influence of circumstances operating more insidiously upon the Mind, and producing an habitual state of abnormal feeling. There is no description of disordered Intellect which, if traced to its source, may not be found either to consist in perverted Emotion, or to emanate from that origin (Bucknill in *Med.-Chir. Review* for January, 1854). Dr. Hood's statistics give the proportions as follows :—*Moral causes,* such as anxiety, grief, uncontrolled Emotions, in 40·8 *per cent.* of the males, and 32·7 *per cent.* among females. *Physical causes,* in 19·8 *per cent.* of males, and 21 *per cent.* of females. The chief of these Physical causes are *old age,* abuse of alcoholic liquors, bodily illness, and the critical period in females.

The principal predisposing causes are *age, sex, hereditary descent,* and *disease.*

(a.) *Age.*—Infancy is nearly exempted from madness, and so also is childhood, except in congenital cases. Esquirol, however, gives the case of a child between five and ten years old whose *monomania* lay in attempting to destroy both her father and mother. Disordered Intellect, however, as a general principle, seldom makes itself obvious till after puberty, when the Passions are fully developed. Dr. Thurnam's table shows that the greatest liability to such disorder exists at the ages between twenty and thirty; while the statistics of Hanwell make the period from thirty to forty to be the most liable to Insanity.

(b.) *Sex.*—It has been much disputed which sex is the more liable. Esquirol, from returns obtained from the different establishments for the insane of London and Paris, considered the numbers to be nearly equal; but Dr. Thurnam's tables show that males are more liable than females to attacks of disordered Intellect, in the proportion of 53 *per cent.* of males to 46 *per cent.* of females. An approximation as to the influence of social position on the patients shows a larger proportion among the unmarried than among the married population.

(c.) *Hereditary Origin.*—The testimony of almost universal experience establishes the fact of a very general hereditary transmission of Insanity, varying from 26 to 69 *per cent.* of the cases. This is remarkably instanced among the high nobility of France and other countries, who almost in every instance intermarry, and are allied by blood to each other, inculcating a sad lesson to those parents who consult, in the marriage of their children, their present interest rather than the health of their descendants. This hereditary tendency to disordered Intellect in the aristocracy is greatly insisted on by Esquirol, who states that only one-third of pauper female lunatics were ascertained to belong to families in which disorders of the Intellect had previously existed;

while more than one-half of the female lunatics of the higher classes were thus connected. In general, children born before the disorder of the Intellect of their parents are less liable than those born after the attack; also, children born of parents diseased in one line are less liable to it than parents diseased in both lines of descent. The condition of the mother during gestation has often a striking effect on the mental health of her future offspring. Esquirol observed that during the French Revolution many pregnant ladies, whose minds were kept constantly in a state of alarm and anxiety during that period, brought forth children who, in their infancy, were subject to convulsive or other nervous diseases; and in their youth either to *mania* or *dementia.*

The form of disordered Intellect chiefly transmitted appears to be *dementia*; the mother's Intellectual disorder being chiefly transmitted to daughters, and that of the father's to sons.

Legislative enactments regarding the intermarriage of persons tainted by disordered Intellect are greatly to be desired; and certainly the concealment of such disorder, with a vew to marriage, ought to render marriages null and void which are concluded under such circumstances.

(d.) *Disease.*—Certain diseases, also, are powerful predisposing, or even exciting causes of disordered Intellect, such as *epilepsy,* which gives rise to a large number of the most incurable cases. Derangement of function or structure of the uterus is another powerful predisposing cause; and many persons become deranged after severe fevers or attacks of "*sunstroke,*" in climates like that of India. Dyspepsia is also a frequent forerunner of Intellectual disorder.

Symptoms and Forms of Disorders of the Intellect.—No unobjectionable classification of these forms can be propounded; and, as Dr. Daniel H. Tuke justly states, any classification must be regarded merely as a chart by which we may shape our course, having only the prominent points marked or partially delineated upon it.

So far, also, as cause can be ascertained, the same cause does not always produce the same result; for in different individuals it will occasionally produce very opposite forms of disorders of the Intellect. Dr. Lindsay justly observes, as a result of his prolonged experience, that "a very large proportion—perhaps a majority—of hospital cases of Insanity cannot be referred *simpliciter* to the heading of any *nosology.*" Changes of type in the disease are also not uncommon; such that a woman, for example, previously suffering from *melancholia* may become *erotomaniacal* or *maniacal:* and *dementia* is a frequent sequel of all the other forms of Intellectual disorder. Such changes of type are of constant occurrence; and it is by no means unusual to find the same case of disordered intellect, at different stages of its progress, presenting the characters successively of *mania, monomania, melancholia,* and *dementia.* The differences between *forms* of Intellectual disorder cannot therefore be regarded as *essentially* very great. The existing manifestation of disorder may be very partial, but the disease is the same.

Besides the simple classification given by the College of Physicians, and already stated, it is of interest to recognize other methods of classification, especially the classifications of Insanity by those men who have had most practical experience with the insane, and who are the greatest authorities on the subject. Two only of these classifications will be given, namely,—

(1.) The following classification (a modification of Heinroth's), suggested by Dr. D. H. Tuke in his and Dr. Bucknill's *Manual of Psychological Medicine*, first edition, p. 89.

CLASSIFICATION OF DISORDERS OF THE MIND, INVOLVING—

CLASS I.—*The Intellect.*

	FORMS OR PHASES OF INSANITY.
Order 1. Development incomplete.	IDIOCY. IMBECILITY.
Order 2. Invasion of Disease after development.	DEMENTIA. MONOMANIA (Intellectual). INCLUDING Delusions. Illusions. Hallucinations.

CLASS II.—*The Moral Sentiments.*

	FORMS OF INSANITY.
Order 1. Development incomplete.	MORAL IDIOCY (?). MORAL IMBECILITY.
Order 2. Invasion of Disease after development.	MORAL INSANITY. MELANCHOLIA— 1. Religious. 2. Hypochondriacal. 3. Nostalgic. EXALTATION, regarding 1. Religion. 2. Pride. 3. Vanity. 4. Ambition.

CLASS III.—*The Propensities.*

Order 1. General.	MANIA (usually a disorder of all the faculties).
Order 2. Partial.	HOMICIDAL MANIA. SUICIDAL MANIA. KLEPTOMANIA. EROTOMANIA. PYROMANIA, DIPSOMANIA.

(2.) The following classification and synopsis by Dr. Lauder Lindsay of the dominant symptoms, principal phases, or general conditions of Disorders of the Intellect, may be useful for the student, as being at once comprehensive and general, accurate (so far as it goes) and practical. It makes, however, no pretence to rigid philosophical or scientific exactitude, or to exhaustive completeness—an exactitude and completeness which its author believes to be unattainable. But it may claim to be more simple and less technical, and hence more *likely to be easily remembered,* than the majority of the many and varied classifications or

Nosologies of Insanity that have been offered during the last half century :—

I. Insanity may be—
  - a. *General* [Examples, Mania, Dementia]; or
  - b. *Partial* [Example, Monomania].

  It may—*apparently,* though perhaps not *really*—affect
  - a. *All* the faculties or powers of the mind [Examples, Mania, Dementia];
  - b. Or any *one* or *two* of their groups, viz. :—
    - 1. Intellect proper [Example, Delusional Insanity];
    - 2. Moral sentiments [Example, Moral Insanity];
    - 3. Propensities or passions [Examples, Dipsomania, Kleptomania, Erotomania].

II. It may be—
  - a. *Congenital* [Example, Idiocy]; or
  - b. *Acquired* [subsequent to birth and generally to puberty].

III. Its characteristic may be—
  - a. *Excitement* [Example, Acute Mania];
  - b. *Depression* [Example, Melancholia];
  - c. *Neither excitement nor depression*—No departure from normal character or conduct [Examples, Some cases of Delusional and Moral Insanity].

IV. It may or may not be accompanied by *Delusions,* which may be either—
  - a. *Partial and uniform* comparatively [Example, Monomania]; or
  - b. *Numerous and variable* [Examples, Some cases of Delusional Insanity.

V. The following *Propensities* or tendencies may be exhibited singly or in combination :—
  - a. *Destructiveness*—
    - 1. *To life*—Suicide, homicide;
    - 2. *To property*—Clothing, furniture, glass, &c. [Example, Pyromania];
  - b. *Alcoholic intoxication* [Example, Dipsomania];
  - c. *Theft* [Example, Kleptomania];
  - d. *Sexual aberration* [Example, Erotomania];*
  - e. Abstinence from food.

VI. Insanity may be—
  - a. *Simple;* or
  - b. *Complicated* [with other diseases].

  The chief complications are—
    - 1. *Epilepsy;*

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* *Satyrasis* and *nymphomania* ought to be distinguished from *erotomania*—the former being an insatiable desire in man to have frequent connection with females, and with the physical power of doing so without exhaustion, with constant erection and desire for venery. It corresponds with *nymphomania* in the female.

Erotomania is a disorder of the Intellect (love melancholy), associated with, if not caused by, the more refined passion of love.

2. *Paralysis*, which may be—
    a. *Partial;* or
    b. *General* [Example, General Paralysis or Paresis];
3. Various—
    a. Functional; or
    b. *Organic* affections of the—
        1. *Lungs* [Example, Phthisis];
        2. *Stomach* and intestines [Example, Dyspepsia];
        3. *Uterus*, &c.

VII. Each of the main forms of Insanity—
    a. Mania,
    b. Melancholia,
    c. Dementia,*
    d. Amentia—

may be developed in *different degrees of intensity* [Example, Dementia is represented by mere facility of character—by silliness, imbecility, or weak-mindedness of every grade, onwards to utter fatuity.

MANIA.

LATIN EQ., *Mania;* FRENCH EQ., *Manie;* GERMAN EQ., *Mania*—Syn., *Tobsucht;* ITALIAN EQ., *Pazzia.*

Definition.—*Disorder of the Intellect (usually of all the faculties) with excitement.*

Pathology.—Mania, having its origin in disordered Emotions, is essentially a disorder of the Impulses or Propensities in the first instance, tending to more or less "*disorder of the Intellect with excitement.*" One or more of the Passions is almost always exalted in *mania,* of which there are two forms—namely, *acute mania* and *chronic mania;* and furious expressions of passion, of prolonged duration, are very generally present in the acute form of this disease. It has in almost all instances its stages of incubation. At first there may be only apparently trifling irregularities in the Affections. The maniac may be at the outset of his disease either sad or gay, active or indolent, indifferent or eager, but he soon becomes impatient and irritable (ESQUIROL). He neglects his family, forsakes his business and household affairs, deserts his home, and yields himself to acts which strikingly contrast with his ordinary mode of life. Delirium and Reason begin to alternate with each other. Periods of composure and agitation succeed each other, and so do acts the most strange and extravagant. The kindest love and tenderness of domestic life serve but to irritate and provoke, so that to remain in the bosom of his family excites the patient by slow degrees to the highest pitch of fury.

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* *Dementia* ought always to be distinguished from *imbecility,* which is a minor degree of *amentia.* *Dementia* is represented by deterioration of Intellectual functions, extending from failing Memory and slight confusion of Thought, onwards to utter fatuity.

In is seldom in *mania*, as in *monomania*, that the patient is insane on one subject only. His mind, says Esquirol, is a perfect chaos; all is violence, effort, perturbation, and disorder. He confounds time and space, associates persons and things the most unnatural, creates images the most unreal, and lives isolated in feelings and reasoning from all the rest of the world. He hates all whom he was wont to love, and wishes to overthrow and to destroy everything. The female maniac, perhaps in health the model of candour and virtue, gentle and modest, an affectionate daughter, a devoted wife, and a good mother, becomes in this disease bold and furious, exposes her person unmoved to the gaze of every eye, is blasphemous and obscene, respects no law either of decency or humanity, threatens her father, strikes her husband, or perhaps murders her children.

In another class of cases the premonitory symptoms are characterized by gloom and despondency, upon which the maniacal excitement supervenes. There is generally a marked departure from the patient's former state of health. *Insomnia* is one of the most important and earliest symptoms. The functions of the body are more or less deranged, and fever may prevail, sometimes severe.

Special or partial forms of mania may be noticed under the following heads:—

(*a.*) **Homicidal Mania** in some cases is the result apparently of delusions, of suspicion, or of implacable enmities against supposed foes. A plausible reason is generally assigned for the attempt in such cases; the victim is represented as having systematically annoyed, or irritated, or conspired against the lunatic. In other cases the attacks are the offspring of momentary, uncontrollable impulse, without cause *quoad* the persons assaulted. The pretext for assault is then frivolous in the extreme—*e. g.*, " he could not help it," or, he did it for " fun." Such homicidal impulse and attempts of the most persistent and dangerous kind may co-exist with a perfect knowledge of right and wrong, and their bearings on human actions—with perfect ability also to manage business affairs, though of a complex pecuniary character—with perfect propriety in maintaining most of the relationships, or of discharging most of the social or public duties of life—with deportment often the most polished and gentlemanly, the most considerate and kind (Dr. Lindsay, *Rep.* xxxvii., p. 42). Gall gives the case of a man at Vienna who, after witnessing a public execution, was seized with an uncontrollable propensity to kill, although he had a clear consciousness of his situation, expressed the greatest aversion to commit such a crime, shook his head, wrung his hands, and cried out to his friends to keep away. Pinel mentions the case of a person who exhibited no other unsoundness of mind than this propensity to murder; so that his wife, notwithstanding his tenderness for her, was nearly being destroyed, he having only time to warn her to flee. In the intervals of the paroxysm he expressed the greatest remorse, was disgusted with life, and attempted several times to put an end to his own existence. Esquirol mentions a woman seized with sudden paroxysmal impulses to destroy her children, and only saved them by locking the bed-room door and throwing the key away. Metzyer relates a similar case of a nurse who requested to be discharged, giving as a reason that every time she undressed the child, struck by the whiteness of its skin, she had an irresistible desire to rip open its belly. The deadly purpose is accomplished in many different manners and times. Sometimes the murder is long premeditated and the victim marked out, the patient concealing a knife about his person till an opportunity for effecting his object presents itself, though that period be remote. In other cases the destructive propensity seems the result of a sudden impulse. Esquirol mentions the case of a patient who was sitting by the fire with other patients, when he suddenly seized a chamber-pot and broke it over his neighbour's head: fortunately he was immediately secured. In a lucid interval he stated that he had made this homicidal attempt in consequence of his brother having appeared to him at that moment, crying out, "Kill him! kill him!" Others, again, are so aware of the approach of the attack that they entreat to be confined, in order that they may not commit the mischief to which they seem irresistibly impelled.

(*b*.) **In Suicidal Mania** there is an irresistible propensity on the part of the patients to destroy themselves. Some of these unfortunate persons, not having resolution to put themselves to death, have killed others, in order to suffer a judicial death. One woman reasoned, "In order that I may die I must kill some one," and accordingly she attempted to kill both her mother and her children. Some of these tragedies are most terrific. A man in a paroxysm of disorder of the Intellect is related to have killed his wife and three children, and would have killed the fourth had the child not escaped. After these horrible sacrifices he ripped open his own belly; but the wound not being mortal, he again drew out the instrument, and pierced himself through and through. This man had enjoyed a good education, and was of a mild disposition. In a large number of cases the suicidal propensity is developed in connection with *religious melancholia*—a form most difficult to eradicate or conquer; and, from its inveteracy, the forerunner often of incurable forms of disordered Intellect.

The propensity to commit suicide is in some persons so great that many destroy themselves although in possession of fortune, of station, of objects of affection, and apparently in every other respect in the fullest enjoyment of happiness. The ingenuity of the patient in providing means for his own destruction is often singular. Some have thrown themselves under the wheels of a waggon; and recently it has not been uncommon for them to cast themselves before the locomotive of a railway train in full speed; others have drowned themselves in an incredibly small quantity of water; others have most ingeniously strangled themselves; and others, more closely watched, have swallowed all sorts of heterogeneous articles—pins, needles, bits of broken glass, nails, buckles—in short, any and every hard substance they could force down their throats. Pinel gives the case of a man who had cut off one of his hands with a hatchet before his arrival at Bicêtre, and afterwards, in spite of his bonds, attempted to tear the flesh off his thigh with his teeth.

(*c*.) **Pyromania.**—The derangements of the Emotions and of Reason may take other forms than murder; and arson is one of the more common. Some seem impelled to this criminal act by the mere sensual gratification of the excitement, confusion, noise, and bustle consequent on the conflagration; delight in the blaze, the ringing of the bells, and in the thronging of the people. It may also result from a process of reasoning, or from acting upon some delusion, as in the destruction of York Cathedral by Martin, effected under a feeling of Divine impulsion, and of his being commissioned thus to purify the house of the Lord.

(*d.*) **Kleptomania** is an irresistible desire to steal. Gall mentions that the first King of Sweden was always stealing trifles; and a countess at Frankfort had the same propensity. Esquirol gives the case of a lady of an exactly opposite character. Her Insanity consisted in a ceaseless dread of appropriating what did not belong to her; she therefore combed her hair an endless number of times in the day, examined her dress minutely every time she put it on or took it off, felt in her shoes, turned up the chairs, looked under her plate, and thus consumed many hours in the day in endless cares lest something of value might have adhered to her dress.

Such are some of the forms or phases of a malady whose varieties are endless. It may be objected by some to the account here given of Insanity, that whereas book descriptions of the disease generally refer to what are regarded as typical cases, which are selected, exceptional, and extreme, yet in the great bulk of the insane the phenomena or symptoms of the malady may be either so complicated and intermixed, or so obscure and ill-marked, that it is not only impossible but futile to attempt a scientific enumeration or classification of all.

(*e.*) **Monomania*** is a term which comprehends various forms or phases of Intellectual disorder, attended with *delusions.* There is generally an undue intensity and exaltation of the Conceptive and Perceptive Faculties. The disorder of Intellect is more or less partial, in such a way that some one Passion or idea so entirely possesses the patient so as often to lead to dangerous conduct. The modes by which the monomaniac gives expression to his particular *delusion* are endless; and the mental affliction is especially indicated by *delusions.* A fixed idea overcomes the Will; and the brain, from its diseased condition, fails to correct by the normal exercise of the Faculties the delusions under which the patient labours. Reason is unable to dissipate them; the exercise of observation fails to discredit them; and *delusions,* more or less persistent, are the result.

It is hardly possible to understand the nature of this phase of disordered Intellect without being well aware that every sense is liable to express, by the mode in which it performs its function, the existence of cerebral disease; as when light things feel heavy, small things seem large, hot things feel cold; or when the senses are liable, from the irritation of the brain or other cause, to become morbidly *active,* the patient seeing persons or hearing discourses when no such person is present, and no such discourse is being related.

Much objection has been taken to the term "*delusion;*" yet the

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* It is directed by the College of Physicians that cases of so-called *monomania* are to be classed under *chronic mania,* or *melancholia,* according to their character.

"symptoms of delusion are still accepted in our courts of justice as the most authentic mark of insanity, and as the essence of cerebro-mental disease." The term may be thus defined, from the gist of the writings of Drs. Bucknill and Tuke:—*A delusion is a belief in the existence of things which have no existence in reality, or an erroneous perception of the nature of things, or of their relation to each other, occasioned by cerebro-mental disease*; or again (as Dr. Bucknill defines it), *An intellectual error caused by the pathological condition of the mind, and displaying itself in false Sensation, Perception, or Conception* (*op. cit.*, 317). A few instances of such morbid sensations, perceptions, or conceptions, are of the following nature:—

Dr. Falconer mentions a case in which cold bodies felt intensely hot to the patient, who could not move without believing he was burnt. Esquirol mentions a lady who, being recommended a lavement, was desirous of administering it herself. No sooner, however, was the syringe put in her hands than she threw it away with an expression of horror, stating that it felt so heavy that she believed it to be filled with mercury, and that they wanted to make a barometer of her body. A gentleman whose Mind was in every other respect perfect had constantly the sensation of his mouth being full of pieces of broken glass: while another, curious in his table and choice in his wines, believed everything tasted of oatmeal porridge. The sight is often the medium of morbid perception. Dr. O'Connor met with a patient recovering from measles, to whom every object appeared diminished to the smallest possible size. Baron Larrey mentions a person who saw men as big as giants; and another patient, on recovering from typhus fever, who felt himself to be ten feet high, his bed eight feet from the floor, and the opening of the chimney as large as the arch of a bridge. The celebrated Pascal always believed he saw a precipice on his left hand, and had a chair placed on that side to prevent his falling into it. The ear, also, is likewise often affected. It hears "the airy tongues that syllable men's names." A gentleman riding by a barracks at evening-call never got the sound of the bugle out of his ears for nine months; and everybody knows that Dr. Johnson always entertained a deep impression that, while opening the door of his college chambers, he heard the voice of his mother, then many miles distant, calling him by his name, "Sam! Sam!"

It is remarkable, also, that *hallucinations* sometimes occur when the organ is itself destroyed through which they would be *objectively* expressed, thus showing their *subjective* nature. Esquirol, for example, attended an insane merchant, who, though labouring under *gutta serena*, not only heard persons talking to him, but saw visions that perfectly enchanted him. He had also under his care a Jewess who was blind, and yet saw things the most strange. She died, and the optic nerve, from its commissure to its entrance into the sclerotic of the eyeball, was found atrophied, so that the transmission of any objective impression was impossible. The optic nerve was, therefore, never called into action; the *hallucination* was a false thought, a conception, which the patient could not distinguish from a true perception. He mentions also two other women absolutely deaf, who had no other delusion than that of hearing every night invisible persons addressing them. Such are instances of *hallucinations*, and the images thus excited are described to be as vivid as those produced by objective causes; so that the patient, when insane, entirely believes the empty and false forms he sees, the ideal sounds he hears, to be real and substantial. Nothing can persuade him of the non-reality of any one of them. Like Macbeth, he insists, "If I stand here, I saw him." It is only by the occurrence of a temporary *hallucination* that we can explain the apparition of the ghost of Cæsar to Brutus, promising to meet him at Philippi; or of the familiar spirit which conversed with Tasso; or of the demon of Socrates, and such like.

Illusions differ from *hallucinations* in this, that the *optic nerve*, or the *auditory* nerve has conveyed an impression which Thought or Idea perverts. They are "false appreciations of real sensation" (BRIERRE DE BOISMONT). They are the misapprehension or false interpretation of external objects which really do exist, so that the most harmless beings may become objects of great terror to patients, and especially to children. Hence the danger of disordered Intellect arising from fright.

Dizziness is a familiar instance of a simple *hallucination*. It consists in a vivid representation of movement either of the body of the patient himself or of surrounding objects which are actually at rest.

In cases of disordered Intellect, if a part of the body be diseased, the Imagination often personifies the local lesion into some strange reality. There are constantly patients in hospitals for the insane who, suffering pains in the crown of the head, believe they are caused by worms gnawing the brain; or, suffering pains in the stomach, believe that organ to be filled with serpents, frogs, or mice. A woman, for many years a patient at Saltpêtrière, who suffered severely from abdominal pains, believed she had a whole regiment of soldiers in her abdomen, and when the pains were severe, that they fought with each other. Another woman, called by the patients "Mere de l'Eglise," believed she had in her entrails all the personages of the New Testament, and occasionally those of the whole Bible. At other times she believed the Popes held their councils in her abdomen. She died, and the abdominal viscera were found adherent to each other and to the peritoneum.

MELANCHOLIA.

LATIN EQ., *Melancholia;* FRENCH EQ., *Mélancholie;* GERMAN EQ., *Melancholie;* ITALIAN EQ., *Malinconia.*

Definition.—*A "disorder of the Intellect with depression, often with suicidal tendency."*

Pathology and Symptoms.—A state of melancholy is often the first indication of Mental disease. It may precede *mania*, and it is associated with or supervenes upon other forms of disorder of the Intellect. It may supervene suddenly, as when it is the immediate consequence of grief; or it may come on gradually, as the mere exaggeration of a naturally melancholy frame of mind. Sudden melancholia is rare. Premonitory symptoms generally indicate a period of incubation more or less prolonged and sufficiently obvious. A state of depression often follows upon a state of *mental elation*, or on prolonged mental exertion and occupation, which suddenly ceases. It also succeeds the mental exaltation produced by inebriating drinks. These effects, however, are generally slight and transient, and the cases of this kind ought to be separated from those cases in which the depression becomes persistent. In *Persistent melancholia* the "relish for existence" becomes less and less, the spirits become depressed, and the man feels unequal to the ordinary duties which call him into public life. In the domestic circle he becomes silent, and seeks entire solitude (TUKE, *op. cit.*, p. 148).

> "He makes his heart a prey to black despair;
> He eats not, drinks not, sleeps not, has no use
> Of anything but thought; or, if he talks,
> 'Tis to himself."—DRYDEN.

And even although he is "cheerless, moody, and taciturn," as Dr. Tuke observes, "he appears to be unceasingly revolving in his mind his unfortunate condition, which, while he regards it as worse than that of any other person, he still believes to be good enough for a miserable wretch like himself." His Propensities are to indolence and general indifference. He reads nothing, writes to nobody, shuns all exertion. One dominant Propensity alone is too often active, namely, self-destruction (CONOLLY); while obstinate abstinence from food and drink is a common feature. There is greatly increased susceptibility of the Emotions; and the subjects of *melancholia*, in its early stage, are easily moved to tears by trivial circumstances. All consolation is disregarded by such patients. They are beyond persuasion. Nothing can subdue their Will, convince them of their error, quiet their alarms, or allay their fears. Nothing can remove their prejudices, overcome their repugnances, or conciliate their aversions. Nothing can divert them from the engrossing thoughts that occupy their mental energies, and take possession of their Emotions. Occasional remissions of the affection may deceive the patient's friends for a time; but the disease progresses till the patient is either placed under the care of competent guardians, or he voluntarily seeks the tranquillity of an asylum. But, fortunately for the chances of cure, a love for and appreciation of the ridiculous is often associated with the tendency to *melancholia*. Cowper, who penned "John Gilpin," is a familiar example. Carlini, a French comic actor of reputation, consulted a physician to whom he was unknown, on account of attacks of profound *melancholia* to which he was subject. Amongst other remedies, the doctor recommended the diversion of the Italian Comedy. "Your distemper," said he, "must be rooted indeed if the acting of the *lively Carlini* does not remove it." "Alas!" said the miserable patient, "I am the *very Carlini* whom you recommend me to see; and while I am capable of filling Paris with mirth and laughter, I am myself the dejected victim of melancholy and chagrin (Prefect's *Annals of Insanity*, p. 404, quoted by Dr. Tuke, *op. cit.*, p. 150).

The ultimate course of cases of *melancholia* varies with the constitution of the patient, his age, the degree in which the disorder is complicated, and the existence of hereditary predisposition to mental disease. It tends to pass into *dementia;* but the tendency thereto is much less marked than in *mania.* The prognosis is more favourable in simple *melancholia* than when complicated with disorder of the Intellect.

Among the earliest mental phenomena *are forgetfulness, abstraction, simple depression of spirits, alterations of the affections towards children or other near and dear relations, restlessness, religious dreads, delusions, alterations of the Instincts, such as hunger.*

"*Among the earliest* physical symptoms *of melancholia are loss of sleep, and disturbed dreams. The digestive organs are frequently deranged, the tongue is unnaturally red or loaded, there is fulness at the epigastrium, and the alvine evacuations are deficient in bile. The tongue is sometimes flabby, pale, indented at the edges ; a fixed dull pain, or an ill-defined sense of oppression is often experienced in the head. The pulse is generally slow and compressible. The urine is often pale, sometimes high coloured, depositing lithates. The skin varies; it is usually harsh, but not unfrequently it is moist and clammy. The uterine functions are more or less disordered, and in a large majority of cases are suspended. In men the reproductive instinct is in abeyance*" (TUKE, *op. cit.*, pp. 152, 153).

The attitude is characteristic. The head is bowed on the chest; answers are given to questions with effort and in monosyllables, or after a considerable pause. The patient is apathetic, taciturn, or absolutely silent.

Constipation, long-continued and habitual, associated with solicitude and family cares in persons formerly healthy, and of excitable and lively dispositions, appear in some cases to have acted as an exciting cause of *melancholia* (Dr. W. H. O. Sankey, *Medical Times and Gazette,* September 19, 1863).

Melancholia comes next in frequency of occurrence to *mania.* It is often hereditary; and all its varieties are disposed to be remittent. "It is generally observed that the remission takes place in the latter part of the day, the patient suffering most acutely in the early morning, and for some hours subsequently." In those cases which have a suicidal tendency this remittent character should be borne in mind (TUKE, *op. cit.*, p. 171).

The principal forms which *melancholia* assumes are—(1.) *Religious;* (2.) *Hypochondriacal;* (3.) *Nostalgic.*

Religious Melancholy.—In the *first* form of melancholy the impress is given to its character by the religious tendencies of the patient, the exciting cause being not rarely traceable to the fiery denunciations of well-meaning but injudicious clergymen or preachers, whose great power in the pulpit is mainly attributable to the excitement and alarm they are able to produce upon susceptible listeners. They delight to suspend such persons over a bottomless pit, in order that they may drag them up again when they fancy they have sufficiently impressed them with most unchristian terror. Such preachers were wont to be banished from the country even in Pagan times by a law of Marcus Aurelius.

Hypochondriacal Melancholy.—In the *second* form of melancholy, that of *hypochondriasis,* the morbid mental state is expressed by the *exaggeration or increase to a morbid degree of intensity,* of that property which every one possesses more or less, by virtue of his physiological and psychological endowments, of creating around him, or within himself, sensations which are not the result of external impressions or corporeal conditions; but which, having their origin in the Mind (subjective), are represented and appreciated by the material organs of the body. It consists essentially in the transference of a phenomenon (subjective or mental in its origin and essence) into what appears to be a real material change, appreciable sometimes by others (REYNOLDS). It is often expressed by the sense of touch, combined with a morbid imagining, so that the patient believes himself to be strangely metamorphosed, changed into some inanimate thing, or he loses all knowledge of his personal identity; and this form of disease is sometimes combined with other *delusions.* The odd conceptions of the patients under these circumstances are singular enough. Men have imagined themselves to be so much butter or putty, and in the one case to be unable to bear heat for fear of melting, and in the other have forborne to walk lest their legs should be crushed by the weight of their body. One man keeps the house, imagining he is too large to pass through a given door-way; and when he is pushed through he screams, and will affirm that his flesh is being torn from his bones. Another imagines he is a pump; that his arm, which is in perpetual motion, is the handle; and bitterly complains that the inhabitants will let him have no rest, morning, noon, or night. A third goes round to his neighbours, believing that he is a seven-shilling piece, and hopes, if his wife should bring him to their shops, they will neither take him in payment nor give change for him. A fourth supposes himself transformed into a beer-barrel rolled along the streets. A fifth, that he is a mutton-chop, and insists that he shall be taken daily to the butcher to be trimmed. Bishop Warburton speaks of a person who thought himself a goose-pie, a circumstance referred to by Pope in his sketch of hypochondriasis—

> "A pipkin there, like Homer's tripod, walks;
> Here sighs a jar, and there a goose-pie talks."

Among other singular forms of *hypochondriacal Insanity* is a belief in an absolute change of sex. Dr. Arnold saw a man who fancied himself in the "family way;" and Esquirol speaks of a male patient who fancied himself a woman, and felt insulted if the slightest liberty was taken with his dress. Some have thought themselves converted, like Nebuchadnezzar, into wild beasts. Some patients imagine they have no soul, others no body, others that they are absolutely dead. One gentleman, approaching his ninetieth year, so far lost his mind that he assembled his family around him, and announced to them that he was dead; begged, in communicating the sad intelligence to his absent friends, they would say he went off easily, and expressed himself a little scandalized that the windows were not closed on the occasion, and entreated, as a last favour, for one pinch more out of his favourite snuff-box before he was finally screwed down. A soldier, who received a severe wound at the battle of Austerlitz, believed he had died, and that the body he had now got was not his own. Another, that he was guillotined during the French Revolution, and had not only lost his own head, but, somehow or other, had got a new one. A third, that his head had been put on his shoulders with the face towards his back; and, lastly, some believe they have not only lost their heads, but can see them rolling on the ground.

It is seldom, however, that *hypochondriasis* is of so simple and harmless a nature. More commonly the Affections are subverted, and those who ought to be most dear to the patient by the ties of relationship become most hateful. The mind, too, is more commonly swayed by some destructive Passion to effect some object criminal in itself. *Delusions* are neither necessary nor essential symptoms. *Hypochondriasis* is often one of the worst concomitants or sequelæ of dyspepsia (WATSON); and when disorders of the Intellect assume the form of *hypochondriasis*, and are at the same time associated with real lesions of the body, the complication is often very embarrasing to the physician, from the distorted, exaggerated, absurd, and false statements of the patient.

Nostalgic Melancholia is sometimes expressed by the term *nostalgia*, and makes itself obvious by an inordinate desire to return to one's native country when far away from home, and to which is added the apprehension, on the part of the patient, that he may never be able to return. The prophecy of the inspired writer seems ever ringing in his ears,—"Weep ye not for the dead, neither bemoan him; but weep sore for him who goeth away: for he shall return no more, nor see his native country" (Jeremiah, chap. xxii., ver. 10). Army surgeons often witness such cases of home-sickness. Ninety-seven soldiers in the French army fell a sacrifice to this disease between 1820-26; and the celebrated Baron Larrey came to the conclusion that the mental faculties in nostalgic patients were the first to undergo a change. Decided aberration of Mind was present in all the cases which he has recorded, expressed by exaltation of Imagination, especially in extravagant delusions respecting their homes. This mental excitement was accompanied by increased heat of the head and acceleration of the pulse. There was redness of the conjunctivæ, and unusual movements of the patient were frequently observed. Uncertain pains occurred in various parts of the body. The bowels were constipated. There was a general feeling of oppression and weariness; an inability to fix the attention; and conversation was apt to be unconnected. A sense of weight and pain pervaded the viscera. There was also sometimes partial paralysis of the stomach and diaphragm, and symptoms of *gastritis* or *gastro-enteritis* would supervene. Under these circumstances prostration of strength ultimately becomes extreme, mental depression keeps pace with the decline of the body, the patient lies weeping, sighing, or groaning, and a propensity to suicide is not unfrequent when the debility becomes extreme. General paralysis is common; but death is the result of a gradual exhaustion of the vital powers (TUKE, *op. cit.*, p. 156).

The Dutch, the Swiss troops, the Highlanders, and the Irish, are those soldiers amongst whom this form of insanity has been mostly noticed; and the disorder is apt to be prevalent during extreme height of the barometer.

DEMENTIA.

LATIN EQ., *Dementia;* FRENCH EQ., *Démence;* GERMAN EQ., *Verrücktheit;* ITALIAN EQ., *Demenza.*

Definition.—*"A disorder of the Intellect characterized by loss or feebleness of the mental faculties."*

Pathology.—The condition is arrived at in several ways, capable of being traced in the mental history of the patient, and is represented by deterioration of mental function, extending from failing Memory and slight confusion of Thought onwards to utter fatuity. Dr. Tuke has pictured these cases in the clearest language, from which the following outline is taken. Some have previously afforded examples of *melancholia,* and were perfectly conscious of all that passed around them. Some have been *maniacal;* others have suffered from the severe delirium of fever or sunstroke; but after a partial recovery from these immediate affections, by slow gradations the mental faculties become dulled, confused, and finally obliterated. Others, again, lose their faculties by reason of extreme old age—*senile dementia*—" the last infirmity of noble minds" (TUKE, *op. cit.,* p. 113).

All such cases are remarkable by their conversation and acts, which greatly resemble infancy; and the mental alienation is indicated by the disorder of ideas, affections, and determinations. Feebleness is the essential characteristic of this form of intellectual disorder; and there is abolition, more or less marked, of all the sensitive, intellectual, and voluntary faculties. In its various stages dementia passes from its slightest and most incipient form to that in which the patient has no longer any just perception of the objects around him, can no longer reason, has completely lost the comparing faculty, and has left to him little more than the functions of vegetable and animal life. There is no judgement, either true or false. On the slightest excitement, some Dements are liable to maniacal outbursts. " If dementia is long continued, its outward signs become well pronounced in the face of the patient. The vacant and puzzled look, the lack-lustre eye, the weak smile, the meaningless laugh, betray the vacuity of Mind " (TUKE, *op. cit.,* p. 115).

Its varieties are *acute and chronic dementia.* Dements may become paralytic, and a thickness of speech may be the first symptom of its approach. After a time the speech is more manifestly affected, followed by a loss of power in the limbs of one side, more marked in the lower extremity, so that the step is feeble and straggling. In the last stage they are completely paralytic, and only able to utter a few unintelligible sounds,—presenting then the phenomena of—

PARALYSIS OF THE INSANE—Syn., GENERAL PARALYSIS.

LATIN EQ., *Paralysis insanorum*—Idem. valet, *Paralysis ex toto;* FRENCH EQ., *Paralysie des aliénés*—Syn., *Paralysie générale;* GERMAN EQ., *Algemeine Lähmung der Wahnsinnigen;* ITALIAN EQ., *Paralisi generale de' pazzi.*

Definition.—*A form of general paralysis in which the cineritious substance* *of the brain is the seat of cloudy swelling of its minute elements,—the brain-cells, with lesion of minute blood-vessels and increase of connective tissue, tending to peculiar disorder of the Intellect, general failure of nerve power, muscular debility, frequent blood extravasation (hœmatoma), convulsions of the nature of apoplexy and epilepsy combined, and to involvement of the whole brain in degeneration and atrophy, so that general and complete paralysis of body and mind results.*

Pathology and Morbid Anatomy.—Muscular debility from general failure of nervous power, rather than motor paralysis, characterizes the cases of this disease; and so far muscular power in this and some other disorder of the Intellect may be regarded as the "*pulse* of insanity." In this affection delusions of grandeur, extreme wealth, prowess, or personal excellence prevail, and a form of convulsions between apoplexy and epilepsy are common, but not constant phenomena.

The lesion seen in the brain after death consists mainly in a parenchymatous inflammation of the cortical substance of the hemispheres, beginning in the inner layer and spreading from thence, causing slow but progressive destruction of the brain-cells, as shown by Dr. Franz Meschede, whose paper, in Virchow's *Archives* for 1865, has been translated by Dr. G. F. Blandford in the *Journal of Mental Science* for October 1866. In acute cases running a rapid course there are hyperæmia and parenchymatous swelling of the inner layer—a congestive turgescence and succulence—causing it to appear darker and wetter on section, such an appearance as attends the "*cloudy swelling* of parenchymatous inflammation. (See vol. i., p. 37). The alteration of the cells is found in different degrees, from mere parenchymatous swelling down to their reduction to molecular detritus (*Biennial Retrospect*, 1865-66, p. 86). The inner layer of the cortical substance has a bright red look, sometimes extending to the surface, chiefly expressed in the anterior lobes, on the convexity along the longitudinal fissure, and tolerably constant in the convolution of the temperal lobes, much less on the basilar surface, and least of all on the posterior lobes. The capillary network is greatly developed, filled to excess with blood-corpuscles, sometimes with points of extravasations here and there, the vessels being tortuous and elongated. In chronic cases this inner layer appears dark, dull, and yellowish, with hardening, so that its consistence is firmer than in the normal state (*sclerosis*). The brain cells of this layer are in different stages of degeneration, at first turgid and swollen, they become cloudy from aggregation of fatty molecules, which sometimes obliterate the form of the cells till they seem mere heaps of granules. It thus really seems to be a diffuse chronic inflammation of the peripheral cortical layer of the brain ("*periencéphalitis chronica diffusa*" of Calmeil).

In vertical sections of the cerebral convolutions, Lockhart Clarke has found a series of streaks or lines radiating through the white and grey substance towards the surface; and in the white substance there appears to be, after hardening in chromic acid, a number of vertical fissures or slits containing blood-vessels, surrounded by secondary sheaths, thicker, darker, and more conspicuous than in healthy brains (Robin). He also finds pigmental degeneration (*Lancet*, vol. ii., 1866). Dr. Sankey is of opinion that the capillaries of the cortical substance are more or less diseased, and there is probably an excess of connective tissue.

The morbid anatomy, as shown by several independent observers, seems to point to that form of "*cloudy swelling*" of the minute elements of tissue—the brain-cells of the cortical substance—to which Virchow gives the name of parenchymatous inflammation, the tendency of which is directly to degenerate and degrade texture; and the minute cerebral capillaries are also implicated in this change.

But, as Dr. Wilks points out, the nomenclature of the disease—"*General Paralysis of the Insane*"—is unfortunate. "An *acute cerebro-meningitis* is found in the ordinary category of diseases treated by the general physician; whereas a *chronic cerebro-meningitis*, attended by a slower derangement of the bodily and intellectual faculties, is styled "*general paralysis of the insane*," and is ignored by the same physician and handed over to the alienist. It is known as *dementia paralytica*, or *paralysie* or *folie ambitieuse;* and the whole brain no doubt eventually suffers, the powers both of body and mind being completely lost. This result is due, in the first instance, to a peculiar inflammatory process in the cineritious matter; and Dr. Wilks considers it has been most satisfactorily determined, that in this disease a very definite morbid process takes place in the cineritious substance. Salaman, Wedl, Robin, Rokitansky, Clarke, and Sankey have also drawn attention to the increased production of connective tissue, and to alterations in the blood-vessels, which have become thickened and varicose; and in a case of general paralysis, an account of which was published by Dr. Wilks, he found that "the cineritious substance had undergone an important degenerative change, and the ganglionic cells were much altered in form and colour, and apparently contained earthy matter. The superficial grey matter was also full of large amyloid bodies, and the blood-vessels (although the patient was comparatively young) had undergone a most remarkable degeneration. The smaller ones were more rigid than any Dr. Wilks had ever seen, standing out from the cut surface of the brain like so many bristles from a brush. Were they in the condition of lardaceous disease? In this disease Dr. Wilks considers the *pia mater* is involved—indeed, the membranes are often incorporated—with the cineritious substance; and such adhesions are in themselves a sufficient proof that the brain-structure has suffered. He here differs somewhat from the account given by Meschede, in this, that Dr. Wilks describes the change as beginning with the *pia mater*, and on the layer of brain-substance; whereas Meschede describes the lesion as commencing with the innermost layer—a description which corresponds with the observations of Baillarger and Regnard as to the changes in the white substance, by its prolongation, in hard yellowish ridges, into the grey matter, as prolification of connective tissue, an appearance well marked in eight out of twelve *post-mortem* examinations, in five of which cases the disease had lasted only nine months. "Hence the term *peri-encephalitis* or *meningo-encephalitis* has been used to express the pathological condition which exists in these cases; and the disease has been regarded as standing in the same relation to *acute meningo-encephalitis* or *cerebritis* as other chronic disease to the corresponding acute affections." "The general *paralysis of the insane* is, then, a disease which has an appreciable morbid anatomy; and in consequence, when cerebral diseases are classed on a pathological basis, it comes under the domain of the ordinary physician; but since the mind suffers in a more chronic manner than in most of the other affections which are seen by such practitioners, this complaint is in practice treated chiefly by the alienist." There are, however, many reasons for drawing a line between it and other mental affections. Thus, it sometimes arises from a definite cause, such as an injury, in a person not predisposed to insanity; it runs a certain course of not many years' duration, and it may attack a brain previously sound. Dr. Wilks gives cases of the disease where the general paralysis followed —(1.) injury; (2.) dementia; (3.) where it was not attended with any exalted ideas; (4.) where it existed without mental symptoms; (5.) where maniacal symptoms accompanied the general paralysis. On the other hand, purely mental diseases are generally dynamic or functional: they depend upon some natural and inherent failing, and they show themselves by peculiarities of manner, habits, and feelings. Then he goes on to inquire whether the peculiar symptoms are sufficient to characterize the disease—many of the symptoms belong simply to an atrophy of the brain, being present even when this atrophy is from *alcoholismus*, also chronic lead and mercury poisoning, and simple old age. Hence, it must be asked, "Whether the peculiar form of delusion, which is certainly one of the most striking features in these cases, is sufficient to characterize the complaint, and whether its absence is enough to negative the diagnosis of general paralysis" (Wilks, in *Guy's Hospital Reports*, vol. xii., 1866, p. 207).

The evidence, *clinical* and *post-mortem*, tends to prove that *general paralysis* of the insane differs essentially from all other forms of disorders of the Intellect, whether the symptoms during life or the appearances after death are regarded—the cases have many well-marked characters peculiar to themselves. The symptoms connected with the Intellect are distinct, as are those also of the bodily functions; the mode of attack is peculiar, while in persons liable, the cause and duration of the malady, all differ from other disorders of the Intellect. The lesions seen after death are also peculiar. It seems to me to be a distinct species of paralysis, rather than a mere variety of disorder of the Intellect.

Symptoms.—The invasion is generally gradual, mental feebleness and muscular weakness being then so slight as scarcely to attract notice; in a few cases, however, the attack is sudden, and preceded by intense energy of body and mind, violence of manner and increased bodily temperature, rapidly followed by commencing *imbecility*, advancing to *dementia*, with incipient paralysis of speech and limb. The impairment of speech very much resembles that which indicates incipient alcoholic intoxication; and the mental condition, that of the effects of Indian hemp (*cannabis sativa*), as taken in the form of *hashish*, "a placid, self-complacent vanity is developed which makes the subject of it feel himself the greatest being, physically and mentally, in the universe." There is an inordinate expansion of *self-feeling*, comparable by Meschede to the pleasing mental sensations induced by wine—believed to be a characteristic result of *turgescence of the cortical substance* or *mind organ*.

Whether the disease commences suddenly or gradually, one of the earliest phenomena is a feeling of weariness of the lower extremities—

wearіedness after little exertion. The gait also soon becomes peculiar and characteristic, as the patient walks "to and fro"—"to and fro"—without any definite object, but an expression of restlessness, which indicates the desire to continue the movements. As the disease advances, more attention is paid to the walk, so that the centre of gravity is maintained with great care, and the patient moves with caution and studied attention, looking neither to the right hand nor to the left. The step is characteristic. The foot has no elasticity of motion, but comes down flat upon the ground. The steps are shuffling and short, while the legs are thrown outwards and apart, and the patient straddles rather than walks, with a shambling and tottering gait.

Perfect co-ordination of the muscles of the lips first begins to fail, indicated by slight tremulousness; and a feeling of stiffness in the lips may be complained of. As a consequence there is hesitation in articulating words, and particularly of words with labial letters. Afterwards, as the disease progresses, any attempt at speech induces convulsive movements of the corners of the lips, twitches or quivering of the upper lip, and contractions of the chin. The head at the same time is nodded shortly and sharply, as if to aid expression. The speech soon progresses in difficulty: it becomes thick, like that of a drunken man, until articulation is impossible. The face becomes devoid of all expression. It is mask-like, or like a curtain—a perfect blank of thought and feeling.

If the patient is asked to put out the tongue, he involuntarily raises his hand to his head, as if to aid the effort; but the mouth is then merely opened, and if the tongue is protruded, it is done in a jerking way, and it trembles greatly. When the patient is made to stand erect, he appears to balance his weight on both legs as equally as possible. He cannot "stand at ease," and the position of the arms and hands is constrained. In sitting, the attitude is square, squat, and graceless, the head droops slightly, the thighs are held parallel, and the knees bent at a right angle, each hand resting on a knee or on the elbows of a chair (SANKEY).

It sometimes happens that muscular feebleness exists for some time before mental imbecility betrays itself; but when the disorder of Intellect manifests itself, it is characterized by delusions of a peculiar kind, such as the possession of good fortune, great wealth, high birth. The animal spirits are exuberant—there is general contentment and good humour, except when thwarted or contradicted, when an outburst of ungovernable passion is apt to be induced. The temper is extremely irritable and uncertain, while the views held for the time and expressed, vary from day to day. The delusions also are apt to be characterized by suspicion, with continual fear of assassination or injury. When the invasion has been gradual, the Intellect becomes slowly weaker and weaker; the Will becomes feeble and purposeless; the Memory becomes treacherous and fails, so that words are omitted in speaking and writing, or sentences are repeated. The pupils may be contracted at first, with subsequent irrgularity, and the *irides* variously susceptible to light. Anæsthesia may exist, with impairment of tactile sensibility. (See example in Dr. Wilks' excellent paper in *Guy's Hospital Reports*, vol. xvi.)

The handwriting is greatly altered, needlework is clumsily done, and with much fumbling, the thread is got and held with difficulty, and often dropt; and all acts of delicate manipulation requiring a keen sense of touch become impossible. The patients are apt to fall and to be much knocked about without feeling pain, and sometimes clumsily pull the features of their face with their hands (SANKEY).

The morbid change on which the disease depends progresses slowly through the brain, and as it does not affect it all at once, the symptoms vary in different cases. Since it begins in the cineritious substance, the mental powers often fail, while much bodily power remains. Thus every lunatic asylum contains many patients suffering from this disorder who can walk well, who can deal a vigorous blow with their fist, and who enter into the games of the institution, or are engaged in labour. That the guiding Will may be almost absent, although the motive powers connected with the central ganglia are perfect, is sometimes remarkably shown by cases of patients who have considerable difficulty in starting to walk, but who, when once the machinery is set agoing, will continue to walk until exhausted, having scarce any power to stop. Such a case is the very opposite of one in which, from disease of the centres or spine, all power is lost, though the mind and will are intact (WILKS). The mental symptoms alone are believed by Dr. Blandford to be sufficient to denote that general paralysis exists; and he believes the defect connected with articulate speech to be in the nerve-centres, which supply volitional power, because the patient, by a strong effort, in which he forces all his nervous energy in the direction, can correct it. This, he considers, would account for the absence of unilateral symptoms which are often absent throughout (*Journal of Mental Science*, Oct., 1866).

At first the patient has the appearance of being well nourished; the appetite and digestion are good.

The body-temperature at the beginning does not vary from that of health, but tends rather to be under the normal, with a difference of about 7° Fahr. between morning and evening temperatures. It is not till after the disorder has existed for some time, and when these peculiar convulsions set in—something of the nature of epilepsy and apoplexy combined—that the temperature is materially increased. At the same time observations as to temperature in the insane must be taken with caution, and with the knowledge that the patient may at the same time be suffering from deposit of tubercle in the lungs and other parts, to which they are greatly liable, and which would very materially elevate the temperature.

This general paralysis of the insane steadily advances from bad to worse, but with occasional remissions in the symptoms; and hence it has also been named "*progressive general paresis.*"

The earliest evidence of impaired excito-motory functions consists in imperfect deglutition, the mouth being filled and the food kept there, or rolled from side to side. There is danger also of its becoming impacted in the pharynx, and so choking the patient. That reflex action is also weakened, is shown by the fact that the soles of the feet may be tickled without causing reflex muscular movements. The sensitive irritability of muscles to electricity is sensibly impaired or altogether absent. In the last stage of the disease there is a constant tendency to gather up the bed-clothes and roll them over, and all instinct of decency is lost. Death generally takes place suddenly, as from meningeal apoplexy, during or after one of the convulsive attacks; or by asphyxia, when the respiratory muscles are invaded. If he survives such an apoplectic attack—and, indeed, as these attacks of convulsions between epilepsy and apoplexy are frequent—layers of effused blood become organized; and *hematoma* of the dura mater are often met with after death in this disease (see *Hæmatoma*, page 199, *ante*), and hence the *cystic hæmatoma* seen in such cases is most probably an accidental complication.

The average duration of the disease is about thirteen months (CALMEIL); few survive three years (ESQUIROL); and it is seldom protracted beyond four or five (WILKS).

Causes.—It may result from an injury to a previously perfectly healthy brain. Men in the prime of life are chiefly attacked, whose activity of brain-life and brain-circulation is in excess; who take a strong flesh diet, much meat and drink; who fully taste life's troubles and joys, excitements and delights; whose brain is much irritated, somatically and physically, and whose powers of resistance are weakened by hereditary taint, illness, or alcoholism (MESCHEDE). It is rarely met with under twenty-five, most frequently about thirty-five to forty-one and a half years, the age which just precedes the fullest brain development—a time at which there appears to be always great nutritive excitement. It rarely occurs after sixty, and never after seventy. Males are more liable than females in the proportion of fifty to fifteen (BLANDFORD). When it does not result from injury, the direct causes, on the authority of Sankey, seem to be prolonged over brain-work, probably combined with sexual excesses, in those who lead "a fast life." Dr. Sankey found hereditary predisposition only in 14·5 *per cent.* of the cases; and Dr. Wilks considers that the evidence of its being hereditary fails in this disease more often than in any other class of disorders of the intellect. This he justly considers is in favour of the view that general paralysis may affect a previously good and healthy brain. Its immediate cause is often an accident or injury; hence, probably, its greater frequency in men than in women, and also its extreme rarity in ladies of the upper class of life. The final phenomena of the disease appear of a passive kind, essentially resulting from wasting or atrophy of the brain; and this recognition, as Dr. Wilks justly remarks, is the basis of correct diagnosis and judicious treatment. The degeneration and brain atrophy, on the other hand, are *results* and not a *cause* of the morbid process in the nerve-cells of the hemispheres of the cerebrum, more especially of its cortical layer.

Prognosis.—Death is the usual end; although some cases of recovery are recorded, as if by crisis, expressed by the formation of boils or abscesses (SANKEY). An authentic case is also fully reported by Dr. Domett Stone, in the *Lancet* of February 2, 1867. The patient got well, under abstinence from mental work, good diet, constant exercise, varied amusement, diversion of the mind, and a restorative drug treatment by *iron, cod-liver oil, phosphorus, nux vomica,* and the like. As a rule, however, general paralysis is intractable. It is continuously progressive to a fatal end, and may almost be regarded as a malignant disease of the grey matter of the hemispheres (BLANDFORD).

Diagnosis.—The disease has symptoms in common with—(1.) Wasting palsy or wasting muscular atrophy; (2.) Chronic lead and mercurial paralysis; (3.) Forms of palsy from cerebral or spinal disease; (4.) Chronic alcoholism,—all of which lead to the final production of an atrophy essentially the same as that which occurs in the later stages of *general paralysis.* But the special form of disorder of the Intellect, which has been regarded by some as essential and characteristic, distinguishing the disease from all these morbid states, are delusions regarding grandeur especially, elation of spirits, a joyous and happy state of mind, extravagant notions (" *Paralysie,*" or "*folie ambitieuse*"), constant mental exaltation; and these delusions are quite different from those which obtain in chronic alcoholism, and which are just the opposite, especially constant mental depression, dread of bodily harm and tendency to suicide, a sense of shame, degradation, and timidity. Alcoholic tremor in the upper extremities is wanting in the general paralysis of the insane, and so are the usual hepatic and other visceral disturbances that attend on chronic alcoholism. The pupils are nearly always dilated from chronic alcoholism; in the insane paralytic they are irregular. But the broad distinction has to be made between this disease and other forms of Disorder of the Intellect. It can only be distinguished from them and from the other diseases already mentioned by the surroundings of the individual case. It cannot be identified generally by the symptoms nor by any group of them, independent of *post-mortem* proof of change in the grey matter. Wilks gives authentic cases of tumors of the brain causing similar symptoms; and the mental exaltation usually met with, and considered by some as pathognomonic, is not absolutely constant, neither are the condition of the pupils, so that the points of distinctive *history* are sometimes wanting or indecisive. Under the term "*general paralysis of the insane,*" a very characteristic form of disease, when seen in its typical form, is no doubt recognizable; but if modified forms and early stages of the disease be included, the term is equally applicable to other affections. The disease is as tangible as Bright's disease of the kidneys, and probably much resembles it (WILKS).

IDIOCY (CONGENITAL).

LATIN EQ., *Amentia* (*ingenita*); FRENCH EQ., *Idiotisme;* GERMAN EQ., *Idiotismus* (*angeborener*); ITALIAN EQ., *Idiotismo* (*innato*).

Definition.—*An abortive condition of the intellect from defective developement of the brain.*

Pathology.—Subjective *phenomena are not capable of being exhibited; and therefore cannot be inquired into. The condition is congenital* (ESQUIROL, COKE, PRICHARD, BUCKNILL, TUKE).

The characters of the idiot, as portrayed by Dr. Tuke, vary according to the degree in which the cerebro-spinal system is involved. In the lower or most abject forms of idiocy the functions of *organic* or *vegetative* life are ill-performed; nutrition is most imperfect; the power of reproduction is small; and the patient would perish were it not for the assistance of others. The functions of animal life are likewise impaired, and he may be scarcely alive to external impressions, nor possess the power of executing spontaneous acts. In the most abject state of idiocy he is blind, deaf, and dumb, while the excretions are passed involuntarily (*op. cit.*, p. 95). From this abject state there are grades of Intellectual and Moral capacity among idiots; and generally it may be stated that the greater the organic deformities the more marked will be the imperfections of Sensibility and Intelligence. Although the smallest heads appertain to the most degraded class of idiots, yet a small head is not a necessary accompaniment of the condition. On the contrary, many idiots have very large heads. The abject condition of the idiot is obvious in his vacant stare, and the thick everted lips, the slavering mouth, the irregular teeth, the gums often swollen, the frequent strabismus, the general want of symmetry, the absence or defect of the senses of sight, hearing, and speech, taste and smell. Notwithstanding the staggering gait, he is constantly moving, if on his feet; and if seated, he has a difficulty in balancing himself, (TUKE, *loc. cit.*)

Moral Idiocy, or congenital defect of the Moral Sense, while the Intellectual Powers are not deficient, but sometimes superior, is a morbid condition not easily demonstrated. There are cases, however, which now and then, throughout the periods of childhood, boyhood, and youth, exhibit all the evidence one can have of an inert Moral nature, associated with an activity of the animal Propensities. Over such a mental condition, threats, rewards, or punishments exercise little or no control. There is said to have been at one time in the Richmond Lunatic Asylum, in Dublin, a man who exhibited a total want of Moral feeling and principle, but who possesssd considerable intelligence, ingenuity, and plausibility. Dr. Crawford wrote of him that he had never been different; that he had never evinced the slightest mental incoherence on any one point, nor any kind of delusion. He appeared, however, so totally callous with regard to every Moral principle and feeling; so thoroughly unconscious of ever having done anything wrong; so completely destitute of all sense of shame or remorse, when reproved for vices or crimes; and he proved himself so utterly incorrigible throughout life, that Dr. Crawford expresses himself as almost certain that any jury before whom he might be brought would satisfy their doubts by returning him insane. The "exact counterpart" of this case was admitted into the New York State Asylum (Benedicts' *Annual Report*, 1850). Dr. Tuke gives many other instances besides these (*op. cit.*, p. 181); but it is still very doubtful if *Moral Idiocy* can be recognized as apart from impaired Intellect; although feeble Moral Powers and Volition, or dangerous peculiarities of temper, may be occasionally associated with good intellectual abilities.

In the so-called *Moral Insanity as developed in adult life*, a standard of *mental health* is to be sought for in the natural and habitual character of the patient; who being thus compared with himself, it will be seen that a change in his feelings and conduct sets in gradually. Moral

Insanity has been more or less clearly defined by Drs. Prichard, Tuke, and Bucknill. Dr. Tuke observes that the adult about to become insane in this respect is "more absorbed or reserved, and on any provocation, however slight, is unreasonably irritated. He becomes suspicious, liable to attribute false motives to his friends or to others, and to cast ungenerous reflections upon his nearest relatives." He is observed to be morose. The alteration of the man from his former nature is complete; and some act of an outrageous character is at last committed. In other cases, an individual has been subjected to over-exertion of mind, his powers overtasked, or his feelings put upon the stretch in consequence of anxiety or unaccustomed responsibility. He then finds himself susceptible to the slightest Emotion. Sleep and rest are lost; he is conscious of more or less uneasiness about the head—a sense of tension and dull aching pain; and at last he is unequal to the discharge of his usual duties. Certain impulses and tendencies begin to distress his Mind, because he knows they are alike repugnant to his Reason and to the dictates of his Moral nature. Often the impulse is to do violence to himself or others; or it is simply to break glass or articles of furniture (*op. cit.*, pp. 185, 186).

Moral Insanity thus expresses itself simply as a tendency to disordered emotional excitement, which affects the course of Thought and Action, without destroying the reasoning process in any other way than by supplying wrong materials to it. There may be no disorder of the Intellectual Powers, or any delusion whatever (CARPENTER). *Reason* does not reign supreme: it is simply retained possession of, while the *Feelings and Emotions* are beyond control. Circumstances in life create feelings and prejudices which prevent the morally insane patient from passing through life smoothly. As regards the *Intellect*, the patient is not insane; but he cannot control his Feelings or Emotions.

Among patients morally insane, physical health, as Dr. Tuke observes, is almost as frequently deranged as it is among those whose Intellect is manifestly disordered; and the cases not unfrequently terminate in some unmistakable physical disease, such as the general paralysis previously described. In a large number of cases the patient has been subject to epilepsy in childhood, or is at the time so suffering; and there can be no doubt that convulsions in infancy are, in relation to their ultimate effects on the Mind, not sufficiently recognized. They often pass away and are forgotten; but some portion of the cerebral tissue has doubtless received an injury which, in any other tissue of the body, would be easily recovered from, but which permanently injures the delicate tissue of the brain of the child, so that his Moral or Intellectual Powers are impaired; and the result may become painfully perceptible as the child grows up to manhood (*op. cit.*, p. 187).

The diagnosis is to be made between this form of disorder of the Feelings and Emotions and mere vicious propensities; and the only ground of diagnosis, pointed out by Dr. Bucknill, lies in the mode of causation. "Moral insanity is always preceded by an efficient cause of mental disease, and there has always been a notable change in the emotions and the propensities following" (l. c., pp. 328, 329).

SECTION IX.—GENERAL DIAGNOSIS OF THE DISORDERS OF INTELLECT.

There are two especially weighty reasons why it is important to obtain a correct diagnosis—namely, *first*, with reference to the necessity of an early application of remedies; and *second*, with reference to the question as to whether or not the person presumed to be insane is or is not legally responsible for his acts.

Dr. Winslow has shown with what inexcusable neglect affections of the brain are generally treated by the public, and the lamentable amount of ignorance that unhappily exists in the non-professional mind respecting these disorders,—a neglect and ignorance which, by sins of omission, often suffer the sacrifice of valuable human life to occur. The overwrought brain meets with but little attention and consideration when in a state of incipient disorder. While medical advice and remedies are eagerly sought for trivial organic or functional disorder in other parts of the body, serious well-marked symptoms of brain disorder are often entirely overlooked and neglected. Such symptoms are not unfrequently permitted to exist for months without causing the faintest shadow of uneasiness or apprehension in the mind of the patient or his friends. These premonitory indications of cerebral mischief, or *prodromata*, as they are technically called, consist of *morbid alterations of temper, depression of spirits, amounting sometimes to melancholia; headache, severe giddiness, inaptitude for business, loss of Memory, confusion of Mind, defective power of mental concentration, the feeling of brain lassitude and fatigue, excessive* ennui, *a longing for death, a want of interest in pursuits that formerly were a source of gratification and pleasure, restlessness by day and sleeplessness by night.* Any one or more of these symptoms obviously indicate an unhealthy state of the functions of the brain and nervous system; but their insidious mode of approach, and the unwillingness of friends to believe that anything is wrong with their relative, rarely if ever permit the symptoms to attract attention till some phase of insanity becomes unmistakably developed. If a person previously in a state of bodily and mental health is conscious that abnormal changes are taking place in his Mind—that trifles worry and irritate him, that he feels his brain unfit for work, that his spirits flag, that he tends to magnify all the evils of life; if, moreover, he is observed to be fanciful, if he imagines things to exist which have no existence apart from himself, if he believes that kind friends ill-use and slight him; if, besides, symptoms like these, or analogous to these, are associated with headache, derangement of the digestive organs, want of continuous sleep, the friends of such a sufferer may rest assured, and the patient may perhaps be convinced, that the state of his brain is abnormal, and he may be induced to commit his case to the careful consideration of a physician. Symptoms of severe bodily fatigue, associated with extreme depression of spirits, mental exhaustion, reverie, paroxysms of melancholy, partial somnambulism, or hallucinations manifesting themselves at an early period of life, must be regarded as important psychical phenomena—deviations from the state of health, requiring the most careful and cautious Moral and Intellectual training, combined with medical and hygienic treatment; more especially to be persevered in if despondency become more marked, or if gloomy thoughts and apprehension of an early death lay hold of the Mind. Of the numerous and seemingly increasing cases of suicide which occupy a place in our daily newspapers, in most of them well-marked symptoms of physical ill-health, disorder of the brain and nervous system, may be traced to exist before the act of self-destruction. In upwards of a hundred recent cases Dr. Winslow has shown this connection to have existed. These cases are full of interest, and demand the most attentive study by the student who would make himself acquainted with the earliest symptoms and most distressing results of Insanity. They are recorded in *The Psychological Journal* for July, 1857, already referred to.

To obtain a correct and early diagnosis, with a view to attain either or both of these objects, one "*only safe rule*" is to be observed; for in the existing state of legal and medical science there is no uniform test of Insanity, either of a legal or of a medical kind, which can be safely or certainly applied. This rule consists in a close and thorough appreciation of the physical and mental aspects of the existing condition of the presumed lunatic, at the period of his supposed Insanity, compared with his prior physical and mental manifestations, which were regarded as his natural and healthy state, and which had not been observed to be different from those of other men—"*a comparison of the individual with his former self.*"

This point was originally insisted on by Dr. Combe; and the necessity of making the Mind of the individual patient, and not that of the physician, the standard of comparison by which to determine the sanity or insanity of the patient cannot be too strongly urged. The man must be the measure of himself; and this principle is found to be of universal application in all physiological and pathological investigations. For example, before the Physician can judge of the condition of the urine passed by a man in disease, he must know the conditions of that man's urinary excretions when they are in a normal state. Scarcely two men are alike in this or in any other respect regarding their excreta,—age, weight, height, and many other circumstances materially modifying the result. So, also, before the physician can judge as to the impairment or disorder of Intellect in a given individual, he ought to acquire or possess some knowledge as to his Intellectual powers previously.

In judging, therefore, of all cases of presumed Insanity, the Intellect must be considered in relation to itself,—the manifestations of Mind *now* must be compared with those which have been *heretofore* expressed by the same individual; and if mental phenomena are ascertained to exist of a morbid kind, compared with those which have been expressed before, and especially if there be any characteristic symptom of cerebral lesion, the individual may be fairly deemed insane, and, if so, he is legally irresponsible for his acts. Between the criminal and the insane mind there are important relations, and it can be shown—as there is reason to believe valuable evidence and strong testimony exist to prove—that a large amount of crime is connected with minds diseased by hereditary predisposition and descent. A large field of usefulness is here opened to the politician, the lawgiver, and the physician, from which future generations can alone hope to reap the benefits—when crime and lunacy may perhaps diminish together.

The following rules, compiled from Drs. Bucknill and Tuke's admirable work on *Psychological Medicine*, ought to be adhered to in diagnosis:—

(1.) *Learn as thoroughly as possible the antecedents and history of the patient.* One of the great difficulties to overcome in the diagnosis of Insanity, especially in the endeavour to ascertain the antecedents of an attack, is the great risk the physician runs in being misled by the interested statements of friends and relatives. They too frequently act on the principle that "what they wish to be, that they believe." They may wish their relative to be considered sane, or the contrary; and they may believe him to be so when he is not, or the reverse; and they will at first invariably disguise or deny circumstances which might be thought discreditable to the presumed patient or themselves. The physician will therefore often find himself surrounded by relatives of the patient from whom he can derive little information which is unbiassed and trustworthy. He will find the household divided against itself; and seeing that such is the case, his best policy is to become a good and patient listener; and if he has a good memory, and is quick in perception, he will arrive at conclusions the more readily that he avoids all cross-examination. With prudence and caution he ought then to seek out people who have known the patient, but who are neither friends nor neighbours, whose evidence will often be more truthful and useful to him. One great aim of such inquiry ought to be to ascertain the existence or not of hereditary predisposition, and of previous attacks of Insanity.

(2.) *Estimate the value of the hereditary tendency, upon the following principles:—*

(*a.*) The Insanity of one parent indicates a less degree of predisposition than that of a parent and an uncle; and still less than that of a parent and a grandparent, or of two parents.

(*b.*) The Insanity of a parent and a grandparent, with an uncle or aunt in the same line, may be held to indicate even stronger predisposition than the Insanity of both parents.

(*c.*) The Insanity of a parent occurring after the birth of a child, without predisposition, is of no value in the formation of a hereditary tendency.

(*d.*) If several brothers or sisters, older and younger than the patient, have become insane, the fact tells strongly in favour of predisposition, although neither parent nor grandparent may have been so.

(*e.*) The Insanity of cousins cannot yet be determined as worth anything in favour of predisposition, except in corroboration of other and weightier facts (BUCKNILL, *op. cit.*, p. 272).

(3.) *Ascertain if there has been any change of habits or disposition.* The physician will thus learn what kind of a man the patient has been when in health. The over-susceptible rather than the eccentric man is the more likely to become insane.

(4.) *Exercise the greatest tact and discretion in the personal examination of probably insane patients.* Obtain an introduction to the patient in as natural a way as possible; and, above all, avoid commencing any conversation which will tend to divulge the object of the visit. In the incipient stage of the disease, the patient is generally suspicious and hostile, contrivance and great tact being required to open up a conversation. It may even be necessary for the physician to be a party to some deception; but he must bear in mind that the discovery of even the slightest deception by the patient may have a most prejudicial influence on future management. As a general rule, it is best to engage the attention, and obtain at least the good-will of the patient by sympathizing inquiries respecting bodily ailments, or concerning such things as he knows the patient takes a lively interest in; and the first aim of the physician must be directed to placing himself on good terms with his patient. "For this no general directions can be given. He must employ that tact, derived from good sense and knowledge of mankind, without which he will find himself lame and impotent in the field of medical practice amongst the insane. The most difficult cases to be inquired into (*de lunatico inquirendo*) are those in which differences of opinion and of interest exist among the members of the patient's family; and when the patient has quietly been told by some one of the family that it is wished to prove him insane, and to place him under confinement, and that a doctor is coming to examine him for that purpose. The physician, under such circumstances, must then do the best he can; and if this is but indifferently well done, he may content himself with the reflection that the fault is not his" (BUCKNILL, *op. cit.*, p. 279).

(5.) *Observe any peculiarities of residence or of dress.* Many circumstances testify to a want of order and direction in household affairs where the head of the family is insane; in the room occupied by the patient things are liable to be out of place, especially as regards the decoration of the walls and the arrangement of the furniture. A love of order is rarely seen among the insane.

(6.) *Study the appearance, demeanour, and general conduct of the patient.*

(7.) *Notice any peculiarities of bodily condition.* This is necessary, especially as regards plumpness or emaciation, the state of the skin, the pulse, the tongue, and the temperature, and condition of the eye, as indicating impaired bodily health. The bodily temperature in the insane is often higher than normal, especially so in *mania,* associated with deposit of tubercle, and the temperature gradually diminishes in the following order,—namely, *general paralysis, acute mania, melancholia, mania, mild and complete dementia.*

In *dementia,* the temperature is often indeed below the standard of health. The difference between morning and evening temperature is often much less than in health, mainly owing to rise in the evening temperature, and not to lowering of the morning temperature, as compared with the healthy standard. This rise in the evening temperature, as compared with that of the morning, is in the exact ratio of the death-rate among the various forms of insanity, finding its acmé in *general paralysis,* in which disease the average evening temperature is higher in every case than in the morning temperature. The evening temperature in every form of disorders of the Intellect is higher than the evening temperature of health. Excitement in a patient is almost always attended with a rise of temperature, as compared with a state of depression or of repose. The differences average 2·2° Fahr. in periodic *mania,* with long intervals, and 1·1° Fahr. with short intervals.

In *general paralysis* there may be a difference of 5·8° Fahr. in the same person at various stages of the disorder. The average temperature falls as the patients get older, but the fall is chiefly in the morning temperature.

The average frequency of the pulse in the various forms of Disorders of Intellect corresponds with the mean temperature, but the rise in the evening temperature has no corresponding increase in the rate of the evening pulse (Clouston, in *Journal of Mental Science*, April, 1868).

The state of the *optic nerve* and *retina* in the insane, as seen by the opthalmoscope, has been made the subject of study. Dr. T. C. Allbutt has noted fifty-one cases of *mania*, in twenty-five cases of which symptomatic changes were found. These symptoms occurred most commonly in cases where other symptoms of organic disease existed, and seemed not unfrequently to depend on meningitis. After a paroxysm of *mania* there remained a paralysis of blood vessels in and about the discs, causing obvious hyperæmia. During such paroxysms there is probably a spasm of those vessels; but the permanent changes are those of stasis, consecutive atrophy, or of simple atrophy.

He noted thirty-eight cases of *dementia*, of which twenty-three showed marked disease of the *optic nerve* or *retina*.

He noted seventeen cases of *melancholia* and *monomania*, in three of whom disease of the eye existed. *Anœmia of the retina* was commonly found in *melancholia*. In forty-three cases of insanity, with epilepsy, there was disease of the *optic nerve* or *retina* in fifteen, and organic disease was known to exist in them from other symptoms. In twelve cases of idiocy there was marked atrophy of the discs in five (*Brit. Med.-Ch. Rev.*, vol. i., 1868).

(8.) *Observe any peculiarities of gesture*, and the expression of the countenance of the patient. One of the great difficulties of diagnosis, also, is to distinguish cases of *monomania* from *sanity*. With the exception of some given *delusions*, turning on a small number of fixed ideas, the patient may be rational on all other subjects; and in some instances even the powers of his mind may be superior, and they often are so. One celebrated instance of this kind occurred to the late Lord Erskine. The patient had indicted a most affectionate brother, together with the superintendent of the asylum, for false imprisonment. He was placed in the witness-box, and the learned Lord, not instructed in the *delusion* of the monomaniac, consumed the whole day in fruitless attempts to expose it. At length Dr. Sims came into court, and suggested to the learned counsel that the patient believed himself to be the Lord and Saviour of mankind. Lord Erskine then adroitly addressed him in that character, lamenting the indecency of his ignorant examination. The patient at once expressed his forgiveness, and with the utmost gravity and emphasis, in the face of the whole court, said—" I am the Christ." In a similar case, tried before Lord Mansfield, the patient evaded the questions of the court the whole day, till his physician arriving, asked him what had become of the princess with whom he corresponded in cherry-juice. Instantly the man forgot himself, and said it was true he had been confined in a castle, where, for want of pen and ink, he had written his letters in cherry-juice, and thrown them into the stream below, and that the princess had received them in a boat. Such answers, of course, immediately terminated the cases.

(9.) *In medico-legal cases of all kinds, including questions regarding disorders of the Intellect, let the physician avoid becoming a partizan.* He ought never to permit his evidence *to be led on matters of opinion*, either directly or indirectly, by counsel on either side. "Facts observed by himself" are the elements on which alone his reputation can be safe in a witness box. On all other points let him steadily refuse to give forth the expression of a mere opinion in a court of law. If he does so express an opinion, it is sure to be made the most of, either for or against contending interests, and so he becomes a mere partizan, derogatory to the dignity of the Physician and degrading to the Science of Medicine.

The student will also find some valuable "*hints for certifying in cases of lunacy*," by Dr. J. S. Bushnan, in the *Medical Times and Gazette* for August, 1862.

SECTION X.—PROGNOSIS IN DISORDERS OF THE INTELLECT.

As a general rule, the younger the patient the greater are the chances of recovery; but above the age of fifty few are cured. The comparative curability of Insanity in its earlier phases is also fully proven by the records of asylums for the insane; while the advantages of early treatment, and the superiority of hospitals for the management and cure of the insane is now fully shown by the increasing annual admissions to these useful public institutions. Of those that recover, the exciting cause often greatly influences the result. Many cases recover when the Insanity proceeds from drunkenness, provided the patient can be restrained from drinking alcoholic fluids; and also if the Insanity arises from slight moral or physical causes. When, however, the shock is severe, the recovery is less certain, and if combined with epilepsy, recovery is almost impossible. The form of the disease also greatly influences the result. When the patient suffers from *delusions*, the chances of recovery are much diminished.

Taking Insanity generally, *monomania* and *dementia* are least frequently cured; *mania* is most frequently cured or improved; and *melancholia* holds an intermediate place. In the Murray Lunatic Asylum, over a period of thirty years, the recoveries from *mania* amounted to 55·02 *per cent.;* from *melancholia,* 31·38 *per cent.; monomania* and *dementia,* 6·90 and 6·70 *per cent.* respectively (*Thirty-Third Report*, p. 11).

Thus *mania* is the most, and *dementia* the least hopeful form of Insanity; and the chances of recovery are greater in *melancholia* than in *monomania.*

If, when labouring under Insanity, the patient be seized with paralysis, it is rare that he survives beyond a twelvemonth after the first symptom—the affection of the speech. The patient may even appear comparatively strong, but great excitement may be followed suddenly by collapse, and a fatal issue supervene sooner than inexperienced persons would anticipate.

In both sexes the recoveries average 51·5 *per cent.* when the cause of the Insanity is of a moral character, and 33·8 *per cent.* when the cause of the disease is of physical origin; and the recoveries are at the rate of 14·6 *per cent.* when the only cause that can be assigned is hereditary predisposition.

The influence of sex on recovery is peculiar. In males the disease terminates at a much earlier period than in females, and its *early* termination in the male is more frequently caused by death. The cases of Insanity in the male sex are not therefore to be considered as more curable, but rather as more fatal; and in the female, also, the cases cannot be said comparatively to be more curable, for although they may not die so readily as the males, the disease may continue in them, passing into a chronic and more permanent state (Hood's *Statistics of Insanity*). On the whole, however, the probability of recovery is greater among women than in men.

From the statistics of Hanwell, by Dr. Thurnam, from 1831 to 1841, the recoveries *per annum of the cases under treatment* have been 5½ *per cent.*, and the average time required to effect the cure has been between four and five years. The following general result is stated by Dr. Thurnam, and is most important to be held in remembrance regarding prognosis:—namely, That in round numbers, of ten persons attacked by Insanity, five recover, and five die sooner or later during the attack. Of the five who recover, not more than two remain well during the rest of their lives; the other three sustain subsequent attacks, during which at least two of them die. All recoveries cannot, therefore, be regarded as permanent or stable; and the broad rule may be laid down, that when Insanity has once exhibited itself, there is ever afterwards a tendency to relapse; and of the insane in public asylums for the reception of all classes, about 70 *per cent.* may be reckoned as incurable. The intervals of recurrence or relapse are very uncertain, varying from a month to thirty or forty years.

Intercurrent bodily diseases in the insane are of great importance in forming a prognosis, and demand the careful study of the physician; inasmuch as they are exceedingly insidious and exceptional in the insane as compared with the sane. In the insane the characteristics of bodily disease are masked or obscured by an inertia or torpor of the nervous system. Their febrile type is generally typhoid or asthenic. There is an absence frequently of symptomatic fever. No complaint may be made, and no external evidence of pain or suffering may be given in cases even of *acute phthisis*, where subsequent *necropsy* shows the lungs riddled with vomicæ and full of pus (vol. i., p. 248); or in cases of *phlegmonous erysipelas* going on to the formation of pus in the limbs; or in *pneumonia*, where the lungs are solidified, and normal respiration impossible; or in *organic diseases of the heart*, in *gastritis*, and in other painful diseases, such as *cancer*, *enteritis*, or *peritonitis*. The possibility of Bright's disease should not be forgotten. Surgical operations may be submitted to without a murmur, as if feeling were completely obsolete. The most serious chest diseases may run their course without cough or expectoration; the excito-motor nervous system would seem to be nearly inert or torpid, and concentration of thought or attention in the insane is in a great measure or quite lost. Sooner or later progressive emaciation and debility, languor, lassitude, and indolence, perhaps anorexia or sleeplessness, direct attention to the state of the patient, in whom the physical signs then show the extent of the apparently latent disease, perhaps rapidly advancing for some time before towards a fatal termination (Dr. Lindsay). The usefulness of the thermometer in detecting such latent disease ought not to be forgotten (see vol. i., under "Fever"). Dr. Lindsay further notices how the type or aspect of Insanity may be quite changed by fatal bodily disease. A patient, from having been passionate, fretful, and abusive, may become affable, mild, and docile. The dying patient sometimes becomes sane towards his last moments, death being preceded by a bright though transient flicker of the light of reason; and the sufferer has even expressed himself serenely, contentedly, happily, as to his latter end, and his transition from life, with all its troubles and diseases, to eternity, with all its joys and comforts (*Thirty-first Report* of the Murray Royal Institution, near Perth, p. 14). The mind may even then be soothed by the hopes and consolations of religion. "The cloud, though not wholly removed, may yet be tinged with a silver lining, and the music of the Gospel truth may help, like the harp of David, to charm the troubled spirit to repose" (Rev. W. D. Knowles, *Thirty-seventh Report*, l. c., p. 297). Such *euthanasia* for the insane is surely to be desired. That insanity also is sometimes vicarious, or alternates with other diseases, must be remembered in prognosis. It is so sometimes with phthisis, one of the most common complications of Insanity. When the phthisis becomes acutely developed, the patient may become temporarily sane, and *vice versâ*.

In the period of convalescence the return of the correct exercise of Judgment is an uncertain and fallacious indication of cure, so long as the Emotions are perverted even in a slight degree from their normal conditions; but immediately the Emotions are controllable and proper, the cure may be considered complete, (Bucknill, *op. cit.*)

The mortality among the insane (based on the average population of a mixed county asylum, according to Dr. Thurnam) is considered decidedly unfavourable if it exceeds 9 or 10 *per cent.;* a mortality less than 7 *per cent.* is highly favourable. The largest mortality is from *dementia,* the least from *monomania;* in the latter, indeed, where there is no tendency to suicide, the duration of life is little abridged; so that premature death is almost in all cases owing to accidental and often preventable causes.

Expectancy of life in the insane, in relation to Life Insurance, thus involves questions of great pecuniary importance. Popular as well as professional errors exist on the subject. The insane are separable into classes for the purposes of Life Insurance, each of which within certain limits is characterized by a different chance or expectancy of life; but statistics on the subject are greatly required (see Dr. Lindsay's *Thirty-Second Report*, p. 17).

But apart from the question of life or death—comparative mortality merely—the curability or incurability of the various forms of disorder of the Intellect is of the utmost importance. What are the chances of recovery after a long or short period is a question on which an opinion is often anxiously looked for. Must the patient be sent to an asylum? Dr. G. F. Blandford has given an interesting paper on these points in the 2nd vol. of *St. George's Hospital Reports* for 1867. The authorities of Bethlehem and St. Luke's Hospital profess to admit only curable patients; and their rule as to curability is, "that the patient must not have been insane more than twelve months." Dr. Blandford gives the history of three cases of recovery from *melancholia*, after illnesses of seven, six, and five years' duration. Such recoveries are rare in those affected with *monomania*, characterized by hallucinations and delusions not melancholic. But when great depression is the prominent feature, it appears that the delusions attending it will vanish if the feeling itself passes away; and we learn from such cases once more the lesson, that the greater the emotional disturbance in any insane person, the more favourable is the prognosis.

Dr. Blandford also notices cases of a *transient* variety of insanity to which the name of *mania transitoria* has been given. Of this there are two varieties; one is connected with epilepsy, and proceeds directly from an epileptic attack, or may take the place of it. Under the influence of it great crimes have been committed. In the other form of transient mania the attack, with the usual violence, delirium and delusions, runs a rapid course, terminating in recovery in a week or less. It is of importance to be able to decide whether the attack will be of this transient kind or run the usual course of *acute* mania of four or five weeks' duration. Time is an element in solution, and in two or three days will solve the difficulty. The attack is likely to be transient if the invasion is very sudden, and if there is a definite and sufficient mental cause, such as a shock or fright. It is likely to be prolonged if its approach has been very insidious and gradual, and if there is no assignable cause. If the bodily condition is much affected; if the tongue is brown and dry, the urine scanty and high coloured; and if the bowels can hardly be moved by the strongest purgatives, it is *not likely* to pass off in a few days. If, on the other hand, the bowels are easily and freely opened, if the urine is copious and pale, and the tongue pale and moist, we may hope that the attack will be soon over, especially if there is extreme violence, bearing no proportion to this slight bodily disturbance. If sleep occurs in a day or two the attack soon subsides. Sound and long sleep is not to be expected so soon in *acute mania.* Removal to an asylum is not to be carried out till it is certain that the illness is *not* transient, and such a measure inevitable (BLANDFORD).

SECTION XI.—MANAGEMENT OF DISORDERS OF THE INTELLECT.

The treatment of Insanity resolves itself into the *medical* and the *moral* management of the case. Medicine indirectly acts upon the brain, as it does upon other organs, so as favourably to influence the course of the disease. It regulates the different actions and secretions of the viscera of the body, and thus improves the general health, so that the happiest results are often obtained by the early and judicious use of medicinal agents.

"Cases of severe mental despondency and distress—instances of alienation of mind associated with hallucinations, and with apparently chronic and fixed delusions, accompanied by strong suicidal and homicidal feelings—have all yielded to medical treatment; and thus persons in all grades of life, who, if those conditions had not been fully appreciated, would have fallen victims to their own insane impulses, have been restored to society in a state of mental health. The symptoms which so generally precede the act of suicide—such as depressed spirits, distress of mind, needless alarms and apprehensions as to some foreboding evil, great irritability of temper, and inability to attend to the ordinary occupations of life, excitability, headache, disturbed or sleepless nights, morbidly exaggerated views of the actual ills and circumstances of life—are in many cases certain signs of acute disorder of the brain, requiring medicinal relief, and being manifestly and rapidly benefited by prompt and energetic medical treatment" (WINSLOW).

In the British military service the following instructions regarding the care and treatment of officers and men suffering from mental diseases are laid down in the *Medical Regulations*, published by Parker & Son, 1859, p. 114:—

"Especial attention should be given to all indications of insanity arising among soldiers; the earliest symptoms of mental disorder should be accurately observed, and suitable means of alleviation should be promptly adopted and carefully carried out.

"Any obvious or marked change in temper, disposition, habits, or conduct, or any peculiar expression of countenance, state of sleeplessness, excitement, or depression, which cannot be traced to an assignable or special cause, should be noticed and duly watched, with a view of ascertaining whether such states are or are not the first manifestations of mental aberration; and should this prove to be the case, some precautionary steps or means of alleviation should be at once taken.

"Whenever the incipient symptoms of the disorder have become more fully developed, the patient should be placed under judicious care, in cheerful, airy apartments, well warmed, lighted, and ventilated, and supplied with all articles needful for comfort, and also for the proper treatment of the case.

"The medical officers being left unfettered as to the precise mode of treatment to be pursued in each individual case, it appears only necessary to urge on them the importance of generally adopting a liberal and soothing system, including good diet, warm clothing and bedding, free exercise, kind and cheerful demeanour of attendants, and all the various medical and moral influences found so beneficial in the treatment of insanity.

"If a propensity to suicide be manifested by the patient, all means of effecting this object should be removed; he should be carefully watched, and during the night time an attendant should be placed to sleep in the same room with him.

"In every case of insanity it is essential to bear in mind that the patient is not accountable for his actions, and consequently that a more than ordinary degree of responsibility attaches to the medical officer and others intrusted with his care and treatment.

"The symptoms, progress, and the remedies employed should be carefully recorded; and in the event of a transfer to another establishment, a full statement of the past history and of the existing condition (mental and bodily) of the patient should be drawn out and sent with him. In order to preserve a full record of all cases of insanity occurring in the army, the particulars in regard to such cases required by the instructions for invaliding must be carefully recorded.

"The previous diseases to which the patient has been subjected for at least the two preceding years (if the man has been with his regiment for that period) must also be noted. In the event of a fatal termination of the case, a careful record should be made of the morbid appearances of the brain, spinal marrow, thoracic and abdominal viscera" (page 113, *Medical Regulations*).*

No uniform method of treatment can be taught. Generally it may be stated that the more the symptoms approach those of *cerebritis, encephalitis,* or *meningitis*—in other words, in proportion as they indicate active inflammatory action—general and local blood-letting may be advisable, *but only in cases otherwise suited for such methods of cure.* Local bleeding is not to be confined to the head, for it not unfrequently happens that it may be adopted with reference to a distant viscus. Leeches to the *vulva* and thighs are beneficial in cases of *mania, monomania,* or *melancholia,* concurrent with the menstrual period; and to the *sphincter ani* in those cases obviously connected with suppressed hæmorrhoids or hepatic congestion. In some instances leeches may be applied with benefit to the Schneiderian membrane, particularly in those cases occurring in early life, and in persons of plethoric constitution and of sanguine temperament. Illusions of hearing or of vision, which had embittered the patient's life, have been removed by leeching behind the ears or over the superciliary ridges. The utility, in *acute mania,* of prolonged hot baths is much insisted upon by Dr. Winslow. The patients remain from eight to fifteen hours in them, at 82° to 86° Fahr., whilst a current of water at 60° is continually poured over the head. Various details of what is now known as hydropathic treatment have recently been introduced, with great advantage, into English asylums, such as the Russian or vapour bath, the wet sheet, wet pack, and the like. Packing in the wet sheet, warm baths, with cold to the head, will often procure sleep more certainly than medicines of the sedative or hypnotic class.

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* MEDICAL ATTENDANCE IN CASES OF INSANITY IN THE ARMY.

An officer or soldier, whether at home or abroad, should, under ordinary circumstances, be attended by the medical officers of the corps to which he belongs, for one month at least., as those officers, from knowing the probable origin and causes of the complaint, may be most competent to its treatment in its earliest stages. But if, after a reasonable time, the patient shall not recover, he should be sent to any general hospital, where temporary lunatic wards may have been provided; and if, after a further reasonable period, there be no prospect of his early recovery, a detailed history of the case is to be transmitted to the director-general, in order that the permission of the Secretary of State for War may be obtained for the removal of the patient to a lunatic asylum, if such removal shall be deemed advisable. In every such case of removal the patient should be sent in charge of a careful non-commissioned officer, and at the same time a minute history of the disorder, its origin, causes, and treatment must be transmitted to the asylum, not only from the regimental medical officer, but from any detachment, garrison, or general hospital where the patient may have been under treatment. A complete statement of the service of any soldier so transferred from his corps to a general hospital should be sent to the governor or commandant, together with the fullest information that can be obtained relative to his place of settlement and nearest of kin. In cases of men invalided and sent to the invalids depot, on account of mental disabilities, the medical history should furnish the following details:—

1st. Name. Regiment. Regimental No.

2nd. Age. Length and places of service.

3rd. Place of birth. Names and Residences of nearest surviving relations.

4th. Social state—married or single.

5th. Temperament.

6th. Character; especial regard being paid as to whether temperate or otherwise.

7th. Form of mental disorder.

8th. Whether a first attack.

9th. Duration of present attack.

10th. Whether the attack was sudden or insidious? If the latter, mention any peculiarity of behaviour or change in habits which preceded it.

11th. Whether insanity was preceded or accompanied by any particular illness, as fever, rheumatism, syphilis, &c. Whether mercury to a large extent has been used for the treatment of any one of them?

12th. What are its supposed causes (moral or physical). Whether the patient has suffered from sun-stroke, concussion, or injury of the head?

13th. Whether any hereditary predisposition exists?

14th. What are the particular ideas or actions which have induced the belief of insanity?

15th, Whether the disease is complicated with epilepsy or paralysis, with homicidal or suicidal impulses? If suicidal tendency exists, mention the way in which self-destruction has been attempted.

16th. Whether the patient is noisy, dangerous, or mischievous, or given to steal? Whether his habits are cleanly or dirty?

17th. What treatment has been adopted since invasion of disease?

To obviate amongst medical officers the confusion so common in the use of terms designating the various forms of mental unsoundness, it is requested that cases may be named in accordance with the terms and definitions as already given by the College of Physicians, and adhered to in this article.

Sedatives, or agents which modify directly the condition of the cerebral tissue, constitute very valuable remedies. In recent acute cases they are generally admissible; but it is in the various chronic forms of *melancholia* that they are most useful. Dr. Winslow observes:—

"In suicidal Insanity, when local cerebral congestion is absent, and the general health and secretions are in good condition, the *meconate* and *hydrochlorate of morphia* often act like a charm, if *uninterruptedly and perseveringly given* until the nervous system is completely under their influence."

Radical cures have been effected by the occasional local abstraction of blood from the head, the administration of alteratives, the warm bath, and sedatives. Success from the use of sedatives often depends upon a *ready adaptation of the form of sedative to the description of case in which it may be deemed admissible, and a judicious admixture of various kinds of sedatives.* With respect to *opiates,* "that medicine which will allay watchfulness in one will not in another, but, on the contrary, increase it. This is particularly the case with opium, which is rarely found admissible in Insanity in its crude state. It more frequently creates heat and general febrile action than sleep" (Sir William Ellis). In cases, however, of recent excitement, *morphia* in considerable doses has been found most beneficial. So also will *chlorodyne* and *chloromorphine* be of use in some cases.

Indian hemp is an extremely useful sedative, not hitherto appreciated sufficiently, for many reasons. (See an excellent paper on its uses by Professor Russell Reynolds, in Beale's *Archives,* vol. ii., p. 154.) It relieves pain, is *soporific, anodyne, antispasmodic;* and while conducing to sleep, promotes at the same time *diaphoresis* and *diuresis,* without producing headache, vertigo, constipation, or impairing the appetite. The dose varies from *one-sixth to one-half grain for a child,* and from *one-third to one grain and a half for an adult.* In cases of mental or emotional disturbance it will be found extremely useful, especially where there is deranged cerebral circulation, with pain and delirium; in cases of incipient Insanity after fever or *sun-stroke;* and in cases of *senile ramollissement.* Recently *digitalis, conium, belladonna,* have been extensively employed as calmatives.

Endermic medication in Insanity offers numerous advantages, but, in the opinion of Drs. Winslow and Laycock, is too little practised. "In some chronic forms of Insanity—in *dementia* and persistent *monomania,* connected, as was supposed, with morbid thickening of the dura mater, and with interstitial infiltration of the membrane, as well as with exudations upon its surface—the head having been shaved, a strong ointment of the *iodide of potassium* combined with *strychnine* has been perseveringly rubbed over the scalp. In other instances the shaved head has been painted with the mixture of iodine; and both modes of treatment have been attended with benefit. When the mental symptoms are supposed to be associated with effusions of serum, *iodine* applied externally at the same time with minute doses internally of *calomel* or *mercury with chalk* (so as slightly to affect the system) are recommended. This treatment, conjoined with occasional tonics, diuretics, and stimuli, to support the vital powers, is occasionally productive of considerable benefit in cases apparently placed quite beyond the reach of improvement or cure." A *solution of iodide of potassium* constantly applied to the shaven scalp has been followed by improvement in the mental state. Cases of *dementia,* the consequence of scrofula, are those in which endermic medication will yield the most satisfactory results (Laycock, in *Med.-Chir. Review* for Jan., 1857). *Hypodermic injections,* especially of calmatives and soporifics, have also recently been employed. Dr. Reissner's experience leads him to recommend *morphia, codeia* and *narceia,* the former being much the most useful, and the others only to be used in special cases when morphia fails. *Narceia* he recommends in those cases where the injection of morphia produces unpleasant symptoms of an uncomfortable kind—not to say of a poisonous tendency. *Narceia* he considers a special remedy in such cases. He has also employed a 6 *per cent.* solution of *Indian hemp* in strong rectified spirits, but without effect.

Preparations of opium introduced into the system by the hypodermic method, it must ever be remembered, are more speedily manifested by the results than when administered by any other mode. The *acetate of morphia* is the best form to use, with a minimum of *acetic acid* in hot distilled water, in the proportion of *five* grains of *acetate* of *morphia* to one fluid drachm of water or of glycerine. *One minim* of this will represent $\frac{1}{12}$th of a grain—a safe and useful minimum dose. *Two minims,* equal to $\frac{1}{6}$th of a grain, is the best commencing dose for relief of severe pain, and as a hypnotic in states of nervous irritability, whether connected with disorder of Intellect or other diseases. *Three minims,* or $\frac{1}{4}$th of a grain, is *an unsafe dose* TO COMMENCE WITH; dangerous and even fatal results have resulted from such a dose. It should not be given till smaller doses have been tried.

Used endermically, the salts of opium are reckoned to be three times as powerful as when swallowed (WARING).

Hydrate of chloral is of use in subduing the delirium of mania, and sometimes in obtaining sleep.

Purgatives may be regularly required. When the bowels are constipated, the form is best determined by the state of the tongue, and sometimes by the idiosyncrasies or proclivities of the patient in regard to medicine-taking. Supposing the tongue to be white and coated, the *sulphate of magnesia*, or other neutral salt, combined with *tincture of hyoscyamus*, in the proportion of ʒj. of the former to ♏ xv. to ♏ xxx. of the latter, in *camphor mixture*, is a formula to be recommended. If, on the contrary, the tongue be clean, the cathartic should be given with some slight bitter, as the *infusion* of *orange peel* or of *gentian*. In some cases the bowels are not only exceedingly obstinate, but the patient may be greatly averse to all medicines. In such cases one or two drops of croton oil placed on the tongue or introduced in food produces free evacuations.

Mild purgative treatment formed the basis of cure in the school of Pinel and of Esquirol; and they usually combined it, in cases of violence, with the application of cold to the head, and of warmth to the lower parts of the body, such as placing the patient in the warm bath and giving him the cold douche—a remedy since more particularly insisted upon by Dr. Brierre de Boismont of Paris, and Dr. Winslow of London. The further treatment consists in restoring any other functions that may be in defect or in excess, as the functions of the uterus in the female, and of the liver or heart in both sexes, by the usual remedies applicable for these purposes.

The Moral management of Disorders of the Intellet is by many supposed to constitute the more efficient mode of cure in Insanity; and it must be admitted to be a most important adjunct. The first important rule is to remove the patient at once from his family: in slight cases, in order that he may be induced to exercise such command over himself as he possesses, and to remove him from influences which may have been aggravating his morbid state; and in severe cases, in order to prevent his doing mischief either to himself or others,—are the reasons for this practice. The main feature in the Moral management of the insane in this country is the abolition and absence of mechanical coercion or restraint. The beneficial action of this system, generally known as "*the non-restraint system*," is now thoroughly recognized in England and Scotland, where it has been gradually established in every asylum since 1847-48. But there are certain exceptional phases of Insanity in which some mechanical restraint is the most humane mode of treatment—the only mode, indeed, of avoiding certain catastrophes of too common occurrence. In certain conditions of excitement, however, it is proper to place the patient at once in a darkened room, remote from noise and the means of injury to himself or others, so that as few objects as possible may irritate him, just as a patient with his eyes affected is kept in a darkened room. The effect of such seclusion is generally of a soothing character; and in not a few cases of periodic mania it is eagerly sought by the patients themselves.

As convalescence advances the patient should be induced to undertake some manual labour, or some office in the asylum or household, which, by amusing his mind, will invigorate his body, and greatly tend to restore the healthy working of his brain. There are no more powerful moral medicines than "Occupation," "Recreation," and "Education." Occupation should be such that no time is left for idleness, or for sitting brooding over morbid fancies. The curative results of well-chosen means of Recreation cannot be over-estimated. When the circumstances of the patient admit of it, travelling, which embraces change of air and change of scene as well as exercise, is often highly salutary in incipient cases; and much has of late been done by the judicious introduction of music and other amusements into asylums. Thus, concerts, balls, conversaziones, evening entertainments, pic-nics, excursions, *fêtes champêtres*, athletic games, pedestrian excursions, public amusements in towns, and carriage drives, are all legitimate and well-approved means of maintaining a constant and varied succession of recreation adapted for all classes of the insane. When Reason is restored, and the Affections again fix themselves on their natural objects, *and when the Emotions are under control*, the patient may be allowed to see his friends, and have his attention directed to the affairs and interests of his family; but it should be remembered that the mind remains weak and enfeebled for some time after apparent recovery: and consequently the patient's restoration to society should be gradual.

With regard to diet, it only requires to be stated that it is often necessary to have recourse to artificial alimentation by the stomach pump in exceptional cases, when food is persistently refused.

An Hospital for the Insane, or Lunatic Asylum, is the most fit and proper place for a "person of unsound mind;" and every asylum ought to be governed by one superintendent, who should be a medical man—an officer of health to the community over which he presides. He ought to have the means of controlling all sanitary arrangements in whole and in detail,—of avoiding overcrowding,—of preventing and destroying effluvia,—and of examining the quality of the food, the water, and the drugs furnished to the establishment. Chemical and pathological appliances ought therefore to be at his disposal for his use. The number of patients who may be thus under the supervision of one medical head in an asylum ought not to exceed 200 (ESQUIROL, AMERICAN COMMISSIONERS IN LUNACY). Premature removal from asylum treatment in opposition to medical advice is greatly to be deprecated; and its baneful results are frequently to be seen, especially in the sad endings of cases of *suicidal melancholia*. In such cases an acknowledgement should be required of the recipient of the patient, or his friends, that he is removed notwithstanding the assurance given by the medical superintendent that the patient is not recovered, and is unfit for removal (Dr. Lindsay, *Thirty-second Report*, p. 14). Such removals not unfrequently induce a change of type from acute and curable to chronic and incurable mental disease.

The subject of Insanity has been treated of in this text-book because it is a subject which medical men are expected to study, and to be called upon to treat and to deal with in a medical and also in a medico-legal point of view. They have, however, few opportunities at our schools of medicine of studying Insanity as they would any other disease in a general hospital. They are, therefore, often called upon to give certificates of Insanity, without perhaps having ever seen or studied a case of disorder of the Intellect; and many recent legal decisions have shown that it is not always safe for a medical man to incur the responsibility of signing such certificates, in the present state of the law relating to lunacy.

Seeing, also, that the legal and medical views of Insanity are not in unison, nor in accordance with what is known as the correct pathology of the disease, the subject has been treated of as fully as time and space would permit. To learn this important subject in all its bearings, the student is recommended to study the comprehensive work so often quoted and referred to in these pages—namely, the *Manual of Psychological Medicine* of Drs. Bucknill and Tuke, of which a second edition has been recently published; and the valuable reports of the Murray Royal Institution for the Insane, near Perth, prepared by Dr. Lauder Lindsay. Writing of "Reports," I heartily concur with Dr. Wilks, when he writes:—"I regret to find that in the reports which are abundantly heaped upon us from lunatic asylums, the work of the mere secretary or superintendent so much overshadows that of the physician, and that the scientific value of these pamphlets is altogether sacrificed to their business character." To this expression of opinion I know of no exception than that of Dr. Lauder Lindsay's, from whose reports I have profited so much.

CHAPTER XVI.

DISEASES OF THE EYE.

SECTION I.—GENERAL PATHOLOGY OF DISEASES OF THE EYE; THEIR RELATION TO DISORDERS OF THE NERVOUS SYSTEM AND TO GENERAL DISEASES.

THE numerous morbid states to which the eye is liable, as well as the peculiar and varied structures of which it is composed, enable us to see almost all its diseases in miniature, as if through a glass, and "to learn many of the little wonderful details in nature of the morbid processes, which, but for the observation of them in the eye, would not have been known at all." So wrote Dr. Latham, many years ago, relative chiefly to the phenomena of inflammation, and before the ophthalmoscope was devised by Helmholtz. Sir Thomas Watson subsequently endorsed this opinion, and commenced his course of lectures on Medicine, with disease of the Eye; and for this reason,—"that we find in the Eye more satisfactory and plain illustrations of the *general* facts and doctrines of pathology than in any other single organ of the body," (*Lect.* xvii.)

Now, since the ophthalmoscope has enabled us to look into the interior and very innermost depths of the eye, it is found that visible indications of disorders of the nervous system may be seen, the great importance of which, to the physician, is only yet beginning to be appreciated, as first pointed out in England by Dr. John Ogle.

Certain changes in the eye are known to accompany certain diseases of the brain and spinal cord, and also certain lesions of organs more remote, which are also the expression of *general* and *constitutional disease.* Various and most important indications of disease are therefore to be seen in the eye. But, like the subject of syphilis (see page 858, vol .i.), the subject of diseases of the Eye have suffered from the unscientific division of the field of medical practice into surgery and medicine; and, at the same time, the uncombined and partial study of Eye diseases by the physician and the surgeon has been a loss to each, preventing each from taking the comprehensive view of the whole facts which belong to each particular case. One-half the facts are overlooked on the one side or on the other; for the ground has been neutral at one time, at another claimed as the exclusive province of the surgeon or the physician. Although, therefore, the diseases of the Eye form now almost a distinct speciality, and are claimed by the ophthalmic surgeon, nevertheless pathology recognizes no such artificial distinctions. The student of medicine must study diseases of the Eye with the aid of the ophthalmoscope as a general practitioner of the healing art. Physicians are learning every day to appreciate the advantages of this instrument as a valuable aid to diagnosis and prognosis, especially in cerebral affections, and the medical schools have recognized the necessity of teaching its use.

The innermost depths of the eye of patients suffering from any acute or chronic affection of the nervous system ought to be examined, whether they complain of defect of sight or not; and practical experience in the use of the ophthalmoscope is absolutely essential. To the works of Bader, Hart, Soelberg Wells, Jonathan Hutchinson, Hulke, Hughlings Jackson, and Zachariah Laurence, the reader is referred for guidance and directions in acquiring a practical knowledge of this instrument.

One of the first and most important observations, in a pathological point of view, made by the aid of the ophthalmoscope, consisted in the recognition of the close relations which exist between the cerebral and intra-ocular circulation, as pointed out by Dr. Ogle; and since then the changes in the *optic disc,* the *retina,* and the *choroid,* happening in the course of cerebro-spinal disease, have been made the subject of special study by Bouchut, Bader, Galezowski, Hughlings Jackson, Hulke, Allbutt, and Laurence. The following account of these changes has been compiled mainly from an able article by Dr. Allbutt on *Medical Ophthalmiscopy,* in the *Brit. and For. Medico-Chir. Review,* vol. xii., 1868, p. 127; from papers in the *Medical Times and Gazette,* for 1868, by the same author; from the works of Hughlings Jackson, Bader, Hutchinson, and Hulke.

The *optic disc* may be *seen* to be the seat of simple congestion, and of congestion with effusion within or around it, of inflammation of its sheath, of inflammation in its substance, of anæmia, and lastly of atrophy.

The circulation of the optic nerve is thus judged of from the condition of its disc and of the retina, so that any change of vascularity evident in those parts of the *fundus* of the eye may be to some extent an index of changes in the cerebral circulation. The circulation in the optic nerve is essentially part of the cerebral circulation. The *optic tract, chiasma,* and *nerve* receive their blood-supply chiefly from the *choroid plexus* and *pia mater;* the *optic tract* generally receives also a branch from the middle cerebral artery, and close to the sclerotic the *optic nerve* receives the short *ciliary arteries* given off from the *ophthalmic* soon after the *arteria centralis retina.* Thus the blood-supply influences the appearance of lesions as they proceed from *optic disc* or *retina.*

The *retina* may be *seen* to be the seat of fibrous and fatty exudations or patches, more especially in the course of the vessels, also of hæmorrhages.

The *choroïd* may be *seen* to be the seat of disturbance, or loss of its pigment, and of hæmorrhages.

These affections of the nerve-vascular parts of the eye may be embraced under the following heads (ALLBUTT) :—

(1.) *Simple hyperœmia* of the *discs* and *retinal blood-vessels;* (2.) *Anœmia of the same parts;* (3.) *Ischœmia* of the *discs and its consequences;* (4.) *Acute interstitial neuritis and consecutive atrophy;* (5.) *Neuro-retinitis;* (6.) *Peri-neuritis, chronic neuritis;* (7.) *Primary or simple progressive atrophy.*

The Blood-vessels within the eye may be *seen* to undergo many characteristic changes, such as diminutions or obliterations, dilatations, tortuosities, pulsations, varicosities, blood stasis, embolism, thrombosis, or rupture. Examples and descriptions of these various morbid lesions are to be seen in the valuable Atlas of Liebriech and in the cromo-lithographs of Bader, Galezowski, and Bouchut.

In severe congestions within the eye there is generally a decided difference between the conditions of the two eyes. The *papilla* is scarcely distinguishable in colour from the *retina,* and perhaps is to be traced only by the convergence of the vessels. The veins tend to become swollen and tortuous, sometimes varicose. When the centre of the disc remains white, this form of congestion has been named *peri-papillary* (BOUCHUT); and is often seen to be confined wholly or partially to one part of the circumference of the *disc* with the corresponding district of the *retina.*

The retardation of blood in the veins produces in them every degree of change, from simple dilatation to varicosity, elongation, and even rupture; and ruptures are most frequent in cases of *albuminuria.* The effused blood degenerates more or less quickly, so that these hæmorrhages appear also as whitish blotches or streaks in the course of the vessels, and are very characteristic of that state of the system in which the small rough kidney is found.

Simple *hyperœmia* may be due to orbital disease, to *choroiditis,* or to Bright's degeneration and *alcoholismus;* but in by far the greater number of cases it is due to encephalitic disease—to tumors, acute or chronic meningitis, or to changes in cerebral vascularity, attended with convulsions.

The Optic Disc.—The lesions which attract most attention here, as connected with central disease, are :—(1.) *Optic neuritis,* with its *consecutive atrophy;* and (2.) *Primary* or *progressive atrophy.*

Optic neuritis is very generally co-existent with *meningitis* of the base of the brain, with *tumors* and with large *hæmorrhages;* and hence it is a valuable symptom. It is to be distinguished, however, as such, from the *retino-neuritis* of albuminuric patients, and from the *retino-choroiditis* of syphilis, mainly by the history of the case, and the limitation of the affection for the most part to the papilla and converging vessels. *It is a condition marked by serous infiltration and prominence of the papilla, and is most commonly due to extra ocular causes, whether orbitar or cerebral* (GRÄFE). The disc becomes larger than usual, its edges indistinct, irregular, and puffy; the infiltration casting a veil over it so as to change its colour into a lilac-grey, and more or less to conceal the vessels as they pass within its margin. The veins increase in size, become tortuous, or even varicose; they darken in colour, and are seen to be gorged with blood; the capillaries also, which, in their normal state, ought not to be seen, become evident, and give a mossy or woolly appearance to the disc. The walls of the vessels are mostly healthy, so that the extravasations of blood, often seen in albuminuric cases, do not generally occur in *optic neuritis.* The lesion is generally seen in both eyes, unless it depend upon orbitar disease. The pupils are generally dilated; in simple atrophy they are for the most part contracted.

During the congestive stage of *optic neuritis,* the *optic nerve* for more or less of its length is of diminished consistence, and of a red or yellowish-grey colour. The sheath is thickened—inflammatory thickening (VIRCHOW); and the contents of the sheath may be pultaceous, when the softening generally extends to the *chiasma,* or far beyond the *tracts,* and even as far as the *central peduncles, corpora geniculata* and *quadregemina* (GALEZOWSKI, TURCK).

The next stage in the process of *optic neuritis* is the commencement of the *consecutive atrophy.* The intense vascularity in and about the disc subsides—the infiltrations are absorbed—the nerve whitens, and the capillaries slowly shrivel and vanish. The edges of the disc become distinct, but are deformed; and patches of organized lymph are to be seen upon and about them. Nevertheless vision is very little interfered with; and therefore this disorder of the eye is apt to be overlooked; and sometimes when patients were more or less blind in the acute stage, sight has been recovered to some degree in the subsequent stage, when the consecutive atrophy commences. Tumefaction disappears in the injured nerve, the nerve-fibres are mostly broken up, and there is considerable hypertrophy of the connective tissue. The nutrition of the nerve has been interfered with, but the nerve, though crushed, may not be quite killed. It generally is not, and if relieved from congestion, it has a chance of recovery.

In *optic neuritis,* Mr. Hutchinson considers that "the neurilemma is first involved in the *neuritis,* and that its nuclei proliferate. Into its meshes the effusions, solid or fluid, of inflammation take place; and it is owing to the continuity of their structure that the inflammatory process travels from one end of the nerve to the other. Thus, also, the nerve tubules are subjected to pressure, and their function is suspended." This view is concurred in by Drs. H. Jackson, Virchow, and most continental pathologists. On the other hand, the able writer on the subject of *Medical ophthalmoscopy*, in the *British and Foreign Med.-Chir. Review* for 1868, does not consider that the structure of the nerve is *primarily* in fault, but concludes that *optic neuritis* depends not at all upon the nature of the intra-cranial disease, but upon the amount of resistance which it offers to the circulation. M. Bouchut likewise concludes that meningitis does not disturb the optic nerve by exciting inflammation in the course of the *neurilemma*, but by throwing up a dam in the way of the venous blood. The *choroïdo-retinal* branches of the ophthalmic vein conveys the blood to the *cavernous sinus*, and thence into the *petrous* or lateral sinus, to gain the jugular vein. But there are lesions which act as obstacles to the flow, and tend to dam up the blood in the sinuses for more or less of their extent. Such obstacles frequently occur from inflammation of the brain or of its membranes, tumors, phlebitis, hæmorrhages, chronic hydrocephalus, cerebral congestions, and which sooner or later, by obstruction to the circulation, cause degenerations of the *retina* and *choroid*, and atrophy of the *optic nerve* and *disc*.

Simple primary or *progressive atrophy* of the *optic disc* is the most hopeless of lesions. It is a process of degeneration from within, mostly ending in utter blindness. It must be distinguished from atrophy with *glaucomatous excavation* or *posterior staphyloma*, and from the changes in the disc which may accompany pigmentary retinitis and choroïditis.

The appearance of *proyressive atrophy* is characteristic. The fine capillaries which give the rosy tint of the healthy disc slowly wane, and a dead white or pearl-like white is left. Vision is lost, passing steadily and surely away. The border of the disc is sharp, clearly defined, flat and even, and it strongly contrasts, by its whiteness and definition, with the red tissues surrounding it. The lesion depends nearly always on some disease of the cerebrum, cerebellum, or spinal cord. In many the atrophy is of a mixed kind, and there may be slight effusion.

Hemiopia (lateral) is always due to cerebral disease.

The *optic nerves* are almost always involved in the process of *meningitis* at the base, and in *tuberculous meningitis*, *optic neuritis* almost always exists. Not so meningitis confined to the *fissure of Sylvius*, or upon the convex surface of the brain. Bouchut, on the other hand, gives a table of fifty-seven cases of *meningitis*, some tuberculous and some not, where obvious changes were seen in the eye, in all except two.

In the first period of *meningitis*, he finds dilatation of the veins of the *retina*, peri-papillary congestion, and often effusion. In the second period, tortuous veins, stasis, thrombosis, and even rupture of the vessels —lesions which appear more or less quickly, according to the amount of obstruction to the circulation in the sinuses occasioned by the meningitis. It always tends mechanically to oppress the venous circulation. Lesions in the back of the eye are also to be seen in Bright's disease and syphilis.

In *Bright's disease* the nutrition of the *optic nerve* as well as of the *retina* is profoundly altered; and atrophic changes of the nerves have been found, even at the corpora quadrigemina, with fatty patches in and about the *chiasma* (GALEZOWSKI). Upon the *retina*, extravasations are found in the course of the vessels. These extravasations are slowly effused, and pass into degenerative states, forming white patches or striations along the margins of the veins, while other patches may be due to degeneration of the retina itself. These *retinal* degenerations are the first in order in *Bright's disease;* while in *cerebral disease* the *optic disc* is generally the first part to show signs of change.

In *syphilis* the *choroid* is the chief seat of lesion; and patches of many colours are to be seen at the back of the eye, some of a brilliant white, others of darker tints, such as red or brown.

The most important indications of cerebro-spinal organic lesions capable of recognition by ophthalmoscopic examination of the eye have been summed up as follows by Dr. E. Bouchut, and are here given together with the observations of Dr. Clymer on lesions in the fundus of the eye, significant of general diseases:—

(1.) *Optic neuritis* and *neuro-retinitis, choroïditis,* and *papillary atrophy* accompany the greater number of acute and chronic affections of the brain and spinal-cord.

(2.) The law of coincidence of *optic neuritis* and organic lesions of the nervous system may be explained by the anatomical and physiological relations of the eye with the brain and spinal cord.

(3.) Whenever any positive hindrance occurs in the cerebral circulation, in consequence of a lesion of the brain or spinal cord, there is *papillary* and *retinal hyperæmia.*

(4.) When there is acute or chronic inflammation of the brain, it may extend to the eye by following directly the course of the optic nerve.

(5.) Affections of the anterior spinal cord may, on account of the anastomosis of its nerves with the sympathetic, at a level with the two first dorsal pairs of nerves, produce the phenomena of *papillary hypertrophy* in the eye, followed later by *atrophy* of the *optic nerve.*

(6.) *Optic neuritis* and *neuro-retinitis,* caused by acute and chronic diseases of the nervous system, are generally found in both eyes.

(7.) In affections of the brain and its meninges, *optic neuritis* is most often more marked in the eye corresponding to the hemisphere most severely affected.

(8.) Changes in the *optic nerve* and the *retina,* accompanying disorders of sensibility, of Intellect, and of motility, always indicate organic diseases of the brain.

(9.) These changes in the *optic nerve* and the *retina* should not be separated from other symptoms of the existing disease; their presence, however, is an element of diagnosis of positive certainty. (Communicated to the French Academy of Sciences, June 8, 1868).

Ischæmia of the discs occurs in all intra-cranial affections which more or less directly distend the ophthalmic veins, as *meningitis, hydrocephalus,* and *intra-cranial tumors.* In *meningitis,* the exudation at the base of the brain may press upon, or the inflammation may involve, the cavernous sinus, in which case there is only *ischæmiæ.* Or the inflammation may creep down the nerve and cause *neuritis optici,* or it may mainly follow the sheath of the nerve and cause *peri-neuritis;* or it may both affect the sinus and the optic vein, and may creep down the nerve; in which case there will be both *ischæmia papillæ* and *neuritis.* Bouchut thinks that the changes in the eye often occur early enough to be the first certain signs of the existing disease. He relates a case of *tuberculous meningitis* of the convexity of the brain, whose diagnosis by the general symptoms was at first doubtful, but which was immediately made clear by the ophthalmoscopical signs,—œdematous redness of the retinæ, and of the papillæ, stasis in the retinal veins, and depigmentation of the choroid. The diagnosis was confirmed at the autopsy (*Gaz. Méd.*, Juillet, 1868). Dr. Clifford Allbutt has observed that the mirror shows the presence, or the traces, of meningitis in a large number of children, and of adults, who survive, and in whom the disease may or may not have been suspected. Such persons who have survived the disorder, tubercular or not, may wholly recover from a state of obscure and protracted ill-health; or a want of full mental power, or a capriciousness of temper may remain; or the reason or the affections may be changed to the degree of insanity. Hydrocephalus is very destructive to the optic nerves. If it be extreme, the discs and retinæ become wholly disorganized, and the optic nerves atrophied by pressure. *Ischæmia papillæ* is the earliest change.

Intra-cranial tumors, including not only malignant growths but cysts, aneurisms, and all local enlargements and thickenings, are, after meningitis of the base, the most frequent cause of *ischæmia* and *neuritis.*

Disease of the cerebellum—tumors, inflammatory changes, and softening—cause mischief to the optic discs.

Acute and chronic cerebritic softening alone never produces *ischæmia,* the changes in the optic nerve being *neuritis,* acute or chronic, and atrophy.

In cerebral hæmorrhage, when the effusion is large, it causes stasis and infiltration in and about the optic discs by obstruction, and this is more marked in the eye corresponding to the site of the clot.

In symptomatic epilepsy, with coarse cerebral lesion, optic atrophy is common, being connected with the organic affection, but it is never associated with essential epilepsy; some circulatory troubles occur in the latter, and venous dilatations are said to have been seen in the central ganglia in severe cases. Dr. Hughlings Jackson has recorded many instances of convulsions beginning unilaterally in which there was double *optic neuritis* (*Royal Lond. Ophthal. Hosp. Rep.*, vol. v.; *Lond. Hosp. Rep.*, vol. iv.; *The Med. Times and Gazette,* May 16, 1868).

In general paralysis, atrophy of the discs is almost constant; but it is not an early symptom, appearing generally about the end of the first stage or the beginning of the second.

In disease of the spinal cord, simple progressive atrophy of the optic nerve is not uncommon. It is frequent in locomoter ataxy—sclerosis of the posterior cord—and has been observed in some cases of chronic myelitis, not attended with shooting pains. It probably depends upon an affection of the great sympathetic, through its communication with the anterior roots of the spinal nerves.

Coloured vision has been seen in epilepsy (JACKSON), and in sunstroke (SWIFT, p. 207, *ante*).

Colour-sensibility is of great importance to be recognized at the commencement of nerve atrophy, and in some forms of syphilitic disease.

In *retinal apoplexies* the *chromatic* faculty is liable to alteration when the patches are very large, or when they affect the central parts of the *retina*. In *syphilitic retinitis* or *neuritis*, with or without choroiditis, the general impairment of sight is attended by loss of perception of green, and sometimes of red. In the *retinitis* of *albuminuria* or of *diabetes*, there is no colour-blindness until the disease has reached an advanced stage, so as to involve the external layers of the *retina* and *macula lutea*. In *atrophy* of the nerve the chromatic function suffers from the outset, especially with regard to red and green (GALEZOWSKI).

In the following sections concerning diseases of the eye, those only will be noticed which are most frequently brought under the notice of the Physician, either as diseases of the Eye, or as morbid states of that organ associated with other internal lesions, often of a general or constitutional kind.

The descriptions given of the following diseases are compiled mainly from the works of Bader, of Stellwag von Carion, and Mackenzie, on diseases of the eye.

SECTION II.—DISEASES OF THE CONJUNCTIVA.

CONJUNCTIVITIS—*Syn.*, OPHTHALMIA.

LATIN EQ., *Inflammatio conjunctivæ*—Idem valet, *Ophthalmia;* FRENCH EQ., *Conjonctivite;* GERMAN EQ., *Entzündung der Bindehaut*—Syn., *Augenentzündung;* ITALIAN EQ., *Conjunctivitide.*

Definition.—*Inflammation of the conjunctiva.*

Pathology.—The conjunctiva is the mucous membrane of the eye, and begins as the immediate continuation of the skin of the face at the edges of the eyelids. Like other mucous membranes, it thus forms a surface, communicating with, and to a very considerable degree exposed to, the external air. An *ocular portion* of conjunctiva covering the eyeball, and a *palpebral portion* covering the eyelids, are to be distinguished, with the *fornix* and *semilunar fold* at the inner canthus, uniting these two portions. Near the cornea the conjunctiva is in closest contact with the sclerotic. Its epithelium is strongly developed, and is continued over the surface of the cornea. In childhood the *ocular conjunctiva* is almost transparent; but with advancing age it becomes opaque, and blood-vessels appear in it, anastomosing freely with those of the *palpebral portion.* The vessels are most abundant on the *tarsal* parts. The vessels of the *sub-conjunctival tissue* are supplied by the ophthalmic artery, and anastomose freely with those of the interior of the eye, so that changes within the orbit, or within the eyeball, may cause over-fulness of the conjunctival vessels. The *fifth* (*tri-facial*) nerve supplies the conjunctiva very richly with nerves, and especially the *palpebral portion.* The structure generally is that of mucous membrane, the component parts being mainly connective tissue corpuscles, and the loosely shreddy bundles of fibres of intercellular substance lying between.

These connective tissue corpuscles are generally found to be the centres where the morbid processes commence in the conjunctiva, especially those of *proliferation,* which may be so luxuriant, and the morbid products so great, that the interspaces of tissue may be completely filled up with new growth, which sometimes undergoes fatty degeneration. The products of growth are always greater in the superficial than in the deep layers.

The outermost layers thus become constantly loosened; and the excretion of new material the more extensive, the more rapidly the process runs its course, and the more luxuriant the *proliferation* of tissue. As the process increases in severity, the elements become more and more removed from the characters of epithelium, and are gradually changed into *mucus* or *pus-corpuscles.* The characters of the secretion also changes. It no longer mixes with the tears, but is a thick and transparent material, which rolls up into balls. As the process becomes more severe the secretion becomes opaque from pus, and may be a whitish-yellow or green-grey colour (*catarrhal*).

Conjuntivitis, or *ophthalmia,* has been distinguished into—(1.) *Catarrhal;* (2.) *Pustular;* (3.) *Purulent;* (4.) *Gonorrhœal;* (5.) *Strumus,* and (6.) *Chronic;* but the distinctions which theoretically cause the forms of conjunctival inflammation to be separated from each, are in reality such that in practice they run into each other by so many numerous intermediate forms and combinations, that diagnosis may altogether depend upon the view taken by the physician of the case.

Symptoms.—The most striking, prominent, and common to several or to all, is the "*redness of the eye,*" caused by the various kinds of increased vascularity. It varies as to tint and as to the arrangement of the blood-vessels which appear on the surface. It is usually of a *bright scarlet* colour in simple conjunctivitis, may affect the *ocular* or *palpebral* conjunctiva alone, and may involve the sub-conjunctival tissue, or the sclerotic also. It varies in degree, is usually irregularly distributed or diffused in patches, some fasciculi of vessels being more distended than others; but when the inflammation is intense, the vascularity may be so great as to obscure completely the colour of the "*white of the eye*" (the *sclerotic*), so that the whole surface, except that of the *cornea,* becomes of a scarlet red. As a rule, the redness is most considerable over the palpebral conjunctiva and at the *fornix,* whence it advances gradually towards the *cornea.*

The blood-vessels of the ocular conjunctiva and of the sub-conjunctival tissue, thus rendered visible by inflammation, anastomose freely with each other, and so form a net-work over the conjunctiva, which can be made to glide to and fro on gently rubbing the conjunctiva against the sclerotic, or the net-work of vessels "can be slipped and dragged about over the adjacent surface by moving the eyelids with the finger" (WATSON). In this way can be recognized the share which the conjunctival vascularity takes in the general "*redness of the eye.*" Varicose conditions of the capillaries may occur, and they may spontaneously rupture, as during coughing, when a red non-vascular patch will denote the site of extravasated blood. It is then commonly called a "*blood-shot eye,*" meaning thereby *ecchymosis.*

Such *ecchymosis,* or blood-shot condition, occurs spontaneously in some persons; and then, though harmless in itself, it ought to be taken as a sign or hint that there may be morbid changes of blood-vessels in other parts.

In contradistinction to *conjunctival* redness, the redness of the *sclerotic*, as seen *through* the *conjunctiva*, must also be recognized by its appearance and by the arrangement of its vessels. The *tint* of the redness is different from the vascularity of the conjunctiva, and two kinds of "*sclerotic redness*" are to be distinguished, namely:—

(1.) Delicate pink or red patches—indicating circumscribed *sclerotitis* or morbid changes in the iris (*iritis*) or *ciliary* processes. If the redness surrounds the margin of the *cornea* like a halo, over which the conjunctiva is easily made to slide, forming a zone of *sclerotic* redness, it is known as a "*sclerotic zone*" or "*ciliary redness*," and indicates undue vascularity of the parts within, especially of the *ciliary processes.* The vessels are small and fine, like hairs, radiating and straight. They cannot be made to shift their place by any dragging of the lids.

(2.) Large blood-vessels emerging from the interior of the eye through the sclerotic, in the ciliary region, especially in front of the sclerotic insertion of the *recti* muscles, one generally from the *rectus externus,* and two from each of the other *recti.* These vary in number, and may be of hair-like thinness, or large and varicose. Such forms of vascularity are met with most frequently in the chronic stage of *arthritic ophthalmia;* and is seen in *chronic glancoma, hypermetropic,* and *presbyopic* patients, where the eye is subjected to much fatigue.

Conjunctival and sclerotic redness may occur simultaneously in *iritis* and *catarrhal ophthalmia,* and may be distinguished by the characters already indicated.

When the vascular ocular conjunctiva and sub-conjunctival tissue next the cornea becomes swollen, the lesion is termed "*chemosis*" (from χημα, *hiatus*—a gap or hollow), and the swelling may be so great that the swollen vascular conjunctiva overlaps the cornea, which thus lies in a hollow or pit, and may be quite hidden from view—the swollen vascular tissue protruding from between the lids, and preventing their being completely closed or opened. The condition is accompanied with pain and heat. The material causing the swelling of *chemosis* may be blood, serum, or both, escaped from an incision, or it may be a fibrinous solid *chemosis*, by infiltration of lymph or fibrine, as in the acute stage of *diphtheritic ophthalmia.* It always indicates a high degree of inflammation.

Œdema of the conjunctiva, or infiltration of its non-vascular tissue with fluid, is observed in the course of *Bright's disease,* and in simple catarrhal ophthalmia in weak persons.

A *discharge* of fluid flows from the inflamed *conjunctiva.* It consists of decomposed intercellular substance, with different kinds of cells, *epithelium* (old or young), *mucous*-cells, (with turbid contents—*cloudy swelling*—and small nuclei), *pus-cells,* incompletely developed *nuclei*—fatty degenerated pus-cells and nuclei. If the discharge contains pus, it is generally contagious.

The discharge varies in consistence: it may be viscid and transparent, as in chronic catarrhal ophthalmia. In the acute form, it is viscid, transparent, and streaked with grey at the commencement; it is opaque and streaked with yellow grey when inflammation has reached its height. The more acute and severe the inflammation, the more abundant, thinner, and less viscid is the discharge.

The more viscid it is the less will it mix with the tears, and will only do so when it becomes greenish, yellow, opaque, and fluid. The proliferation of material is most active close to the surface of the conjunctiva.

Intolerance of light (*photophobia*) and spasmodic *closure of the eye-lids* is generally most expressed when the *cornea* becomes implicated; and very slight morbid changes will often produce the most intense *photophobia*—the instinctive or reflex desire to exclude the light causing the spasmodic closure. This spasmodic closure may be subdued by the application of *tincture of iodine* to the skin of the eyelids twice a week—taking special care that none runs into the eye; also by the insertion of a seton in the skin of the corresponding temple (BADER).

Watering of the eye (*epiphora*), *profuse flow of tears* (*lachrymation*), are also most expressed when the *cornea* is implicated, as by a minute superficial ulcer, inflammation, or pustular corneitis. The forcible opening of the eyelids will then be followed by a gush of hot and scalding tears, discharged from the outer and upper part of the fornix, where most of the lachrymal ducts open on the conjunctiva.

Pain during conjunctivitis, if severe, is a sign of implication of the cornea, by ulceration or suppuration; and its sudden appearance and sudden subsidence, after six to twelve hours in *diphtheritic ophthalmia* suggests perforation of the *cornea*.

One form of ophthalmia may pass into another by numerous intermediate forms and combinations—catarrhal into gonorrhœal, and granular into diphtheretic. Implication of the cornea and changes in the eyelids may happen at any stage.

Treatment must be directed to the purulent discharge, whatever may be the condition of other parts, by caustic and astringent remedies.

Dr. Bader recommends the following lotion for catarrhal and other mild forms of purulent ophthalmia.

℞. *Aluminis*, gr. xxx.*; Spr. vin rect., f* ℥ ss.*; aquæ Rosar., ad.* ℥ viii.*; misce.*

In simple ophthalmia, whenever undue vascularity of the conjunctiva exists, the state of the eyelashes and the tear ducts, the puncta, and surface of the conjunctiva, especially the fornix and the portions along the inner edges of the palpebral margins, should be particularly examined. Small white particles of chalk may be working their way towards the surface, an eyelash may be turned inwards on the eyeball, or lodged in one of the cunaliculi.

CATARRHAL OPHTHALMIA.

LATIN EQ., *Opthalmia cum catarrho;* FRENCH EQ., *Ophthalmie catarrhale;* GERMAN EQ., *Catarrhalische Augenentzundung;* ITALIAN EQ., *Oftalmia catarrale.*

Definition.—*An inflammation of the conjunctiva and the meibomian follicles, characterized by the secretion of a varying amount of turbid, mucous,* *or of muco-purulent material, the discharge of which is considerable, with some hyperœmia and swelling* (Von Carion).

Pathology.—If with the *conjunctiva* the *sclerotic* seems unduly vascular, a rheumatic condition may be associated with the ophthalmia, and has been named catarrho-rheumatic ophthalmia, to indicate the combinations.

Symptoms.—The conjunctiva becomes vascular, generally first at the inner canthus, and in slight cases the vascularity confines itself to the *palpebral conjunctiva,* or to the *semi-lunar fold* and *caruncle.* In severe cases a uniform redness of the *ocular* and *palpebral conjunctiva,* obscuring its large vessels, is observed. There is a reticular injection of the ocular conjunctiva; and in the severest forms the whole conjunctiva is reddened of a light hue, so long as the symptoms of irritation only predominate. Numerous small blood-spots may appear among the enlarged vessels of the ocular conjunctiva. The swelling rarely amounts to *chemosis,* and in weak persons *œdema* of the lids may appear; but there is no intolerance of light unless the *cornea* becomes implicated. Swelling is chiefly seen in the *semi-lunar fold* and *caruncle.*

The discharge at first is viscid and clear, with a few opaque flocculi, which causes the discharge to be streaked with grey. It is composed of fully but ill-developed epithelial cells, with one, rarely with two nuclei, and mucous cells; and during the acute stage mucous and pus cells with ill-developed epithelial cells. It then becomes opaque and yellow. At first the pus is not abundant, and it may generally be seen lying in the angle between the eye and the lower lid upon pulling the lids apart; or it makes itself visible at the corner of the eye, or between the eyelashes, along the edges of the lids. In chronic catarrhal ophthalmia it becomes transparent and viscid. It does not mix with the tears, the flow of which is not increased unless the cornea is implicated.

"Stiffness of the eyelids," and a sensation as if "sand had got into the eye," are the statements usually made by patients at the commencement of catarrhal ophthalmia, and commonly called and considered to be a "cold in the eye." During sleep there is very little sectretion from the conjunctiva, so that in the morning after sleep a sensation of "dryness of the eye" is complained of; and the slight discharge that has occurred is so altered in character that, accumulating round the eyelashes, it causes them and the eyelids to "stick together"—a condition which at once suggests the use of astringent lotions. The leading symptoms are then *redness* of the conjunctiva, some pain and uneasiness of the eye, an increased *discharge* from the conjunctival membrane and meibomian follicles, and *sticking together of the eyelashes and eyelids.*

Vision is generally more or less impaired, by flocculi suspended in the tears being diffused over the cornea by the motion of the lids. They render objects cloudy, as if a smoked glass were held before the eye.

Prognosis.—If left without treatment acute catarrhal ophthalmia may get well in about six weeks; otherwise it goes on to *purulent ophthalmia,* or it may become chronic, or pass on into *granular ophthalmia,* thus proving a source of permanent irritation. Under judicious treatment it ought to subside in from two to six weeks. It is one of the most common forms of ophthalmia or diseases of the eye.

Causes.—Sudden atmospheric changes, as from crowded rooms to the open air; the contact of secretions from other mucous membranes, such as the vagina or the lachrymal sac, are the most frequent causes of catarrhal ophthalmia. It may also occur during the course of erysipelas, or after *measles, small-pox, or scarlet fever* (*exanthematic ophthalmia*), *eczema* or herpes of the face, or after injuries to the eye.

Treatment.—Exposure to drafts or currents of air are to be avoided; and the patient ought to remain in a room of uniform temperature, and have the bowels freely moved by a searching purgative, *such as calomel* and *jalap.* Astringents or caustics applied to the conjunctiva usually arrest the inflammation. Local applications to the affected membrane are more useful than general treatment. "General remedies in this disease are inferior to local ones. *Violent* general remedies are worse than useless, but a local stimulant treatment may almost entirely be relied on" (Mackenzie, p. 441, 4th edition). This disease is one to which the use of powerful astringents is more particularly applicable. The object of astringents and caustics is to cause irritation, with destruction of the surface with which they come in contact, and to relieve the loaded blood-vessels by the "serous secretion" they induce. The pain caused by caustics or astringents should not continue longer than from ten to fifteen minutes. The best astringent lotion is the "*lotio aluminis,*" already given at page 421, if there is no intolerance of light or pain. But before using either lotion or caustics the discharge should be washed quite away from the lids with warm water, or warm milk and water, after which the margins of the eyelids and "inner corner of the eye" are wiped frequently with a piece of lint dipped into the lotion; and while this is being done the patient ought to be made to open and close the eyelids, so as to allow some of the lotion to get "into the eye." This process is to be repeated from three to ten times daily, for about five minutes each time, according to the amount of the discharge. The same piece of lint is not to be used a second time, but must be at once burnt, and a fresh piece used for each application.

The late Dr. Mackenzie, of Glasgow, recommended a *solution of the nitrate of silver* in distilled water, in the proportion of four grains to the ounce. A large drop of this solution is to be applied to the membrane once or twice, or three times, in the course of the day, by means of a pretty thick camel-hair pencil. If the patient recline his head backwards the drop is to be placed in the hollow formed in the internal angle of the eye. The drop will then be diffused over the globe upon the separation and subsequent winking of the lids. After a minute or two a pricking smarting sensation is felt, which ought to subside in from ten to twenty minutes; after which the feeling of "sand in the eye" is entirely removed and the inflammation abated. The eye continues easy for five or six hours, when another drop must be let into it, and so on till the remedy is found to give less and less pain, and at last is scarcely felt, and the cure is complete.

As a caustic, "green stone" (or *lapis divinus*) may be used. It is composed as follows :—

℞. *Cupri Sulphatis; Nitri puri; Aluminis,* āā ʒi.; *Camphoræ rasæ,* ʒss. (BADER). These ingredients are to to be dissolved, and moulded into sticks, to be used for gently "touching" the palpebral conjunctiva every other day, as long as the discharge is abundant.

Common salt, sesquioxide of iron, are also used; and it is usual to write for a grain of *sulphate of zinc, sulphate of copper, sulphate of cadmium,* or *crude alum,* to be dissolved in one ounce of water; and half a grain of *nitrate of silver,* a quarter of a grain of *corrosive sublimate,* four grains of *acetate of lead,* two to four grains of *salt,* or *sesquioxide of iron,* to be dissolved in one ounce of water; and of *tincture of opium,* half a drachm to the ounce. These solutions are all now considered equally efficacious. They all act irritatingly on the conjunctiva, causing an abundant nuclear formation or pus, according to the strength of the solution.

To prevent the eyelids sticking together in the morning, after sleep, *glycerine ointment* is to be applied at bed-time along their margins.

It is composed as follows:—Mix one part of *amylum* (starch) with five parts of *pure glycerine,* having previously heated the *glycerine* to about 90° Fahr., in which the starch is to be soaked (Von Carion).

This ointment is soluble in water, always retains the same consistence, and mixes freely with the moisture on the conjunctiva. It is therefore a useful agent for keeping the edges of the lids moist, and serves also as a vehicle for other astringent remedies, such as *sulphate of copper, acetate of lead, nitrate of silver,* or *red oxide of mercury.* Two grains of either of these substances, well mixed with one drachm of the *glycerine* ointment, are equal in strength to four grains of the same substances when mixed with one drachm of fat.

PUSTULAR OPHTHALMIA.

Latin Eq., *Ophthalmia pustulosa;* French Eq., *Ophthalmie pustuleuse;* German Eq., *Pustulöse Augenentzündung;* Italian Eq., *Oftalmia pustulosa.*

Definition.—*Inflammation of the conjunctiva, accompanied by small, round, circumscribed, grey, opaque, and more or less vascular elevations, which finally become pustular, or nodules situated in the conjunctiva, at or near the outer and inner margin of the cornea, and rarely on the palpebral conjunctiva.*

Pathology.—The lesion is generally associated with scrofula, and hence sometimes considered as a form of "*strumous* or *scrofulous ophthalmia.*" It has also been called *herpes* of the conjunctiva, and *phlyctænular ophthalmia.* It is a disease to which children are so liable, that out of one hundred cases of inflammation of the eyes of young subjects, ninety are of this kind. It is very often the first and earliest manifestation of a scrofulous constitution; and, if neglected or mistreated, may become the cause of permanently impaired vision, or even of entire loss of sight. It is most prevalent from the time of weaning till about eight years of age. "When asked to prescribe for children having bad eyes, you will find in nineteen cases out of twenty that you have to deal with *purulent ophthalmia,* if the child be still at the breast, and with *strumous* (*pustular ophthalmia*), if it has been weaned" (Watson). It is rare in adults, unless they have already suffered in early life. Sometimes one and sometimes both eyes are affected from the first; or the disease may pass from one eye to another; and when both eyes are inflamed at once, the one is generally much worse than the other (MACKENZIE). The so-called "pustules" are really small "*pimples*" at first, varying in size; and round their bases the conjunctival and sub-conjunctival vessels are congested. They are exudations among the sub-conjunctival blood-vessels in the first instance, and, passing through various stages, may finally change into ulcers before they disappear. Or, becoming deeper seated, an abscess forms, accompanied by much swelling of the surrounding sclerotic. Groups of the so-called pustules may sometimes be seen at the margin of the cornea, close to each other, causing a flat, circumscribed, grey-red swelling, abruptly defined by the margin of the cornea, and surrounded by large blood-vessels.

Symptoms.—The appearance of the pustules is preceded by a burning, stinging pain, vascularity of the conjunctiva, with some mucous and watery discharge, and by intolerance of light, even in cases where scarcely any redness is present. Dr. Mackenzie suggests the following anatomical explanation as sufficiently accounting for this intolerance of light—namely, that the lachrymal nerve, after supplying the lachrymal gland, goes to the *conjunctiva* and *orbicularis palpebrarum*, and may serve to establish a strong nervous sympathy of a reflex kind between these parts. There is an evening remission of the disease, suggestive of a retina in a state irritable to light.

This lesion of the eyes is generally associated with other symptoms of scrofula, such as eruptions, sore ears, swollen upper lip, nasal catarrh, enlarged lymphatic glands, swollen joints, *tabes mesenterica*, dyspepsia, tumid and hard abdomen, and general debility.

Prognosis.—The pimples or nodules appear two or three days after commencing vascularity of the conjunctiva, which begins to diminish about the seventh day, assuming a dark-red hue. Pus then begins to appear at the apices of the pimples, and thus a pustule forms.

This process may go on repeating itself, and so protract the ophthalmia until a better state of the general health is brought about. A very cautious opinion must be given as to recovery. No disease is so apt to relapse; and in after life *amaurosis*, with hardness of the eyeball and *glaucoma*, are apt to occur in those who have suffered from this disease in youth (MACKENZIE).

Treatment requires that the general health should be improved by the management of the diet, and by every other hygienic arrangement that the case may suggest. In children especially, the diet must be regulated and restricted to the most digestible forms of food and cooking, with a warm bath every day. Wine may be required in some cases.

With regard to medicinal treatment, such remedies as are of a tonic nature—improving the general health—are to be at once commenced.

There are certain indications for the use of such remedies, of which the following may be stated:—

If the lips and nostrils are red and swollen, with crusts round the nasal orifices, the preparations of *arsenic* with *iron* will be found most useful. If the sclerotic is implicated, as indicated by the swelling and characteristic vascularity, the *bi-chloride of mercury* (corrosive sublimate) ought to be given in doses of *one-sixteenth* of a grain in half an ounce (a table-spoonful) of water twice daily. In young children, the *Hydrag. C. creta* (Bader), or a combination of *rhubarb, soda,* and *calumba* twice daily will be found most useful, where mercurial preparations may be objectionable.

Such medicinal treatment is required to be continued in the constitutional management of such cases for at least two, three, or four weeks, even after the immediate lesions are subdued. In many cases, however, it proves most rebellious; and Dr. Mackenzie was wont to put patients with considerable quickness of pulse and heat of skin on a course of *nauseants* or *emeto-cathartics,* when suffering from this disease. To an adult he would prescribe from one to four grains of *tartar emetic,* with from one to two ounces of *sulphate of magnesia,* dissolved in twenty ounces of water, of which two or three table-spoonfuls may be taken every half hour till vomiting is excited, after which the dose may be repeated at intervals of three, four, or six hours, according to circumstances. From one-half to one-sixth or one-eighth of a grain of tartar emetic may be given to a child.

As to local applications, the *lotio aluminis* (p. 421) is to be used to wash the margins of the lids night and morning, if they are "gummed up," and may be discontinued as soon as this condition has subsided. If the discharge is only watery, the lids may be kept clean with warm water only. If the edges of the eyelids are red or excoriated, after washing with the *lotio aluminis*—warm—particles about the size of a hemp seed of the *unguentum, hydrag. nitratis mitius* may be rubbed upon the lids at bedtime.

If vascularity should persist and continue great, with pains and heat—suggestive of corneal implication—one or two leeches may be applied at bedtime to the corresponding temple of the morbid eye. *Quinia* to the extent of one grain three times a day is of great use, using at the same time the following collyrium:—

℞. *Corrosive sublimate,* gr. i.; Sal. ammonica, gr. vi.; Vin. opii., ʒii.; Aquæ distilat. ad. ℥viii. To be used tepid, by mixing a table-spoonful of this lotion with an equal quantity of hot water, as a fomentation for five minutes—a little being allowed to flow into the eye (Mackenzie).

PURULENT OPHTHALMIA.

Latin Eq., *Ophthalmia purulenta;* French Eq., *Ophthalmie purulente;* German Eq., *Purulente Augenentzündung;* Italian Eq., *Oftalmia purulenta.*

Definition.—*Inflammation of the conjunctiva characterized by true chemosis, and by great secretion of muco-purulent matter, which rolls up into flocculi, and floats among the tears; or, becoming purulent in six to twelve hours from the commencement, mixes with and dissolves in the tears* (Pyorrhœa).

Pathology.—Similar to catarrhal ophthalmia in many respects, and affecting the same parts, but with much greater intensity, it is a common and severe disease of warm climates, such as Egypt, Persia, and India. In Egypt it has been endemic for ages; and having passed with the British troops into this country in 1800, 1801, and 1802, after their contest, under Sir Ralph Abercrombie, with the French troops, it obtained the name of *Egyptian ophthalmia,* and excited very great attention at that time, never having been accurately described before. A severe contagious ophthalmia was then introduced into this country, which afterwards prevailed extensively in regiments which had never served in Egypt; and which accompanied the British troops to almost every foreign station to which they were sent, and especially to Sicily, Malta, and Gibraltar. "The disease," as we now see it, writes Dr. Parkes, "is one of the legacies which Napoleon left to the world. His system of making war with little intermission, rapid movements, abandonment of the good old custom of winter quarters, and the intermixture of regiments from several nations, seem to have given a great spread to the disease; and, though the subsequent years of peace have greatly lessened it, it has prevailed more or less ever since in the French, Prussian, Austrian, Bavarian, Hanoverian, Italian, Spanish, Belgian, Swedish, and Russian armies, as well as our own" (*Prac. Hygiene,* p. 495). The French troops suffered no less extensively, for two-thirds of the French army were labouring under it at one time (ASSALINI). It has proved no less destructive in the Belgian and other continental armies.

"It has also been evidently propagated among the civil populations by the armies, and is one more heritage with which glorious war has cursed the nations" (PARKES). Rarely seen, however, in private life, it is more often met with in armies (*ophthalmia Bellica,* or *military ophthalmia*), on board ship, in poor-houses, or in large public schools. When it occurs after the contact of gonorrhœal pus with the conjunctiva, it has received the name of *gonorrhœal ophthalmia;* when it appears in new-born infants, by contact with discharge from the vagina, it has been named *ophthalmia neonatorum,* or simply *purulent ophthalmia.* From time to time, after its introduction into this country, it raged with great severity as an epidemic among large bodies of men, as in the 54th, 52d, and 59th regiments in 1806; also in the Military Hospitals at Chelsea and Kilmainham in 1804, 1806, and 1809, when it was more severe than at any other time. In 1810, on the 1st of December, there were no fewer than 2,317 soldiers unfit for duty from blindness, in consequence of *purulent ophthalmia,* and this number does not include soldiers who had lost the sight of one eye only. Within six years, up to 1810, upwards of 900 cases, exclusive of relapses, had taken place in the Royal Military Asylum at Chelsea (SIR PATRICK MACGREGGOR).

During a period of twenty-seven years (ending March 31, 1854), 2,648, or 5·8 *per cent.,* of 45,411 invalids were discharged from the army for impaired vision; and during the last six years of the period the percentage had risen to 7·5 (DR. PHILIP FRANK).

Symptoms.—The disease is characterized by extreme rapidity of progress—so much so, that patients generally present themselves when a high degree of inflammation has been already reached. Commencing as a common catarrhal ophthalmia, within from six to twelve hours, and rarely exceeding thirty, the inflammatory stage has reached the process of pus formation at some part of the conjunctival surface even before the patient may be aware that he is affected. It often happens that attention is first excited by finding the eyelids adhering in the morning.

The right eye is more frequently attacked than the left, and in general is more severely affected, and the sight of it more frequently lost. Sometimes only one eye is affected, but commonly both suffer—often after an interval of several days before the second becomes inflamed.

The order in which the symptoms succeed each other is as follows:—Itching is first felt in the evening, or a sudden feeling of sand between the lids and the eyeball; sticking together of the eyelids, especially after sleep; inflammation of the conjunctiva, characterized by swelling and rapidly increasing vascularity. The semilunar membrane and *caruncula lachrymalis* are considerably enlarged, and redder than usual. The swelling of the parts is soft, somewhat elastic, and easily made to bleed. The itching indicates suppression of the natural mucous secretion of the conjunctiva of the eyelids, and of the *Meibomian* secretion—a constant and early effect of inflammation on every mucous surface and secreting organ. The thin acrid secretion from the conjunctiva which follows gives the slipperiness to the internal surface of the eyelids, and the *Meibomian* secretion being now increased above its usual quantity, and altered in quality, concretes among the eyelashes, and causes the eyelids to adhere during sleep. The sensation of sand is due to the dilated state of the conjunctival vessels (MACKENZIE).

After twenty-four hours the discharge is still thin, but viscid, and begins to be opaque. It lodges at the inner angle of the eye, and on everting the lids they are vascular and tumid. Not unfrequently a considerable discharge of blood may take place from the conjunctiva, which may be repeated from time to time with temporary relief to swelling, before the puriform discharge commences. It is rather an oozing of colouring matter from the blood, mixed with discharge, than a real effusion of blood from ruptured vessels.

If the patient is scrofulous, the intolerance of light is acute. Within thirty-six hours profuse purulent discharge has commenced, with swelling and great redness of the eye-lids, and inability to open them. *Chemosis* and haziness of the cornea are generally present. In many cases the discharge is so abundant that it flows down the cheek the instant the lids are separated, irritating the skin, and even excoriating the surface. With increase of discharge, the swelling of the lids increases, especially of the upper one, and a proliferation of its papillary structure takes place. The œdema exists in some cases only in particular spots; and *chemosis* gradually extends from the lids over the surface of the eye towards the cornea, its advancing edge being accurately defined, leaving a circle round the cornea. Gradually the swelling closely surrounds the cornea, and at last it may completely bury and overlap its surface, so that its centre can scarcely be seen, and the conjunctiva of the eyeball protrudes between the lids.

Such *chemosis* is generally accompanied by livid redness and swelling of the skin covering the eyelids, especially that of the upper lid, which often reaches a very great size, completely overhanging the lower one.

The discharge may exceed several ounces in the day (VETCH), continuing without much change for twelve or fourteen days, or even longer, and ultimately as the swelling subsides the discharge becomes thin and gleety, and even later "granular lids" remain. The papillæ becoming indurated, forms a granular, scabrous, mulberry-like surface, which, constantly rubbing against the cornea, keeps up a chronic inflammation of the investing membrane, which becomes covered with red vessels, and tends to lose its transparency.

This is a favourable end to the disease; but in less fortunate cases the eyeball may be disorganized. The cornea may be turbid but entire, of a flesh-like appearance from fungous excrescences, penetrated by one or several ulcerations through which portions of the *iris* may protrude, or the cornea may be entirely destroyed by suppuration.

The internal structures of the eye also sometimes suffer in extremely severe cases, the commencement of which is indicated by deep lancinating pains within the orbit, aggravated during the night, coming on most frequently from ten to twelve, and declining towards morning. There is also pulsatory pain in the eye, coming on in paroxysms, or continuing persistent in violence till the cornea gives way. The pain chiefly affects the branches of the fifth nerve. *Supra-orbital* or *circum-orbital* pain is characteristic of inflammation, extending to the sclerotic cornea, choroid, and iris. If these severe symptoms occur, rupture of the cornea may take place—an event always followed by protusion of some of the tunics, generally the iris, which in a few days becomes swollen and covered with yellow lymph, then vascular; finally it is covered by opaque fibrous tissue and cicatrices, thus forming a *staphyloma* and loss of sight. About the eighth day is a common period for the cornea to give way; but it may happen as early as the third or fourth day, when the disease is at the height of its violence, and when the swelling is so great that the parts immediately concerned cannot be examined. When it is thus impossible to examine the parts from the swelling, it may be concluded that the inflammation is confined to the *conjunctiva* if the pain be only scalding, with a sensation of "sand in the eye;" but that it has extended to the *sclerotic* and *cornea*, if the pain be severe, throbbing, and paroxysmal. It is in such cases that the cornea may give way.

Rupture is indicated by a copious discharge of hot fluid, which sometimes gives temporary relief to pain.

The progress of the disease is extremely deceptive. At first a circumscribed portion of the epithelium becomes opaque, soon after the cornea has been seen to be quite clear and transparent. The opaque patch is thrown off, and an ulcer appears which spreads rapidly in depth and along the margin of the cornea. The whole cornea has thus sometimes been thrown off.

The progress of the disease may not be terminated by rupture of the cornea. In a few hours the capsule of the crystalline lens gives way, and allows the lens to escape, especially if a large portion of cornea is destroyed rapidly, when more or less of the vitreous humour generally follows, and sometimes almost the whole contents of the eyeball are evacuated. In such cases no *staphyloma* occurs, but a small, deformed, shrunken eyeball is left deep sunk in the orbit, over which the lids fall in, become concave externally, and remain ever afterwards closed. Patients of a scrofulous constitution are most apt to such disorganization of the cornea.

Constitutional symptoms are not as a rule severe, but children seem to suffer much more than adults; and as the local symptoms grow in severity, the constitution begins to suffer. The pulse becomes frequent and sharp; there is much general uneasiness, and the paroxysms of nocturnal pain prevent sleep. Repeated relapses produce great debility. In Egypt, the disease would thus be continued for two or three months, impairing the general health, and often terminating in diarrhœa, dysentery, or hectic (DR. JAMES MACGREGGOR).

Causes and Propagation.—The contagious nature of this disease is no longer a matter of doubt. Cases in evidence of its contagiousness have been detailed especially by Sir Patrick Macgreggor, and are quoted alike by Dr. Mackenzie and Sir Thomas Watson.

Generally it may be stated that whenever the discharge becomes purulent, it is capable of reproducing itself in the mucous membrane.

The most certain and common cause is contact of pus from another case of the disease, or from *catarrhal opthalmia,* or from *gonorrhœal pus.*

In illustration of these facts the following cases are given by Dr. Mackenzie on the authority of Sir Patrick Macgreggor. They prove, as Sir Thomas Watson remarks, two important facts:—(1.) That the disease is capable of being excited in the eye of a person previously healthy, by the direct application of the puriform discharge from an eye affected with this ophthalmia. (2.) The very rapid operation of the poison so applied.

A nurse of the Military Asylum Hospital, about nine o'clock, A.M., when occupied in syringing the eyes of a patient, who had much swelling of both eyelids, with a profuse purulent discharge, found that some of the matter mixed with the injection, had spurted into her left eye. She was directed to bathe her eye immediately with luke-warm water. Notwithstanding this precaution, about seven o'clock in the evening, the left eye began to itch to such a degree that she could not refrain from rubbing it. When she awoke next morning, the eye was considerably inflamed, the lids were swollen, and when she moved the eyeball she had a sensation as if sand were lodged between it and the eyelids. In the course of the day purulent matter issued from the eye, and other symptoms followed, which were similar to those in the children under her care. The disorder, however, subsided under the usual treatment in fourteen days, the right eye remaining sound during the progress of the disease in the left.

Another nurse, about eight o'clock, A.M., while washing with warm water the eyes of a boy suffering severely from purulent ophthalmia, inadvertently applied the sponge which she had used to her right eye. She immediately mentioned this circumstance to the other nurses, but took no means to prevent infection. Between three and four, P.M., of the same day great itching of the right eye took place, and before she went to bed, it was considerably inflamed. Next morning her eyelids were swollen; she complained of pain on moving them, and the whole anterior surface of the eyeball was much inflamed. A purulent discharge also began to trickle down the cheeks from the inner canthus. The symptoms increased in severity, and, notwithstanding the means that were used for her relief, the eyeball burst in front of the pupil on the fourth day after the application of the purulent matter. The sight of the eye was irrecoverably lost, and the inflammation continued for upwards of three months; but the left eye did not become affected.

Direct experiments were also performed by Dr. Guillie. He took the puriform mucus from the eyelids of some children affected with puro-mucous conjunctivitis in the Hospital for Sick Children at Paris, and introduced it under the eyelids of four children belonging to the Institution for the Blind. These children were amaurotic, but the external surface of their eyes was healthy and entire. In all four a regular puro-mucous conjunctivitis was produced (Dr. Mackenzie, page 442).

The history and progress of the disease since it has been noticed in Europe is also strongly indicative of its contagious nature. It so spread from Egypt both to France and to this country, and to other places where detachments of the Egyptian force were subsequently stationed. Wherever it has prevailed among our troops at home, it has been uniformly observed that it first broke out among soldiers who had come from Egypt, or had communicated with regiments that had been in Egypt. In all cases its origin could be traced to the introduction of fresh troops into the regiment or barracks. It diffuses itself rapidly when once it is introduced into places where numbers of persons are collected together under circumstances favourable to the development of contagious diseases, as among soldiers in barracks, where many live in the same apartment, and use the same towels, and sometimes even the same water for washing in; while officers who live in larger, separate, and better ventilated rooms, generally escape. Its progress is checked by measures which provide against such accidental contagion.

It is not improbable also that the disease may be communicated through the medium of pus particles floating in the air without any substantial applications of the morbid secretion from a diseased to a sound eye.

It is also certain that purulent ophthalmia may result by direct contact of gonorrhœal discharge with the conjunctivæ, and not, as has been believed, by *metastasis* to the eye, without such contact. Of this fact Sir Thomas Watson gives the following well-marked examples:—

> "It is a common persuasion, among the lower classes, that to bathe the eyes in human urine is good for the sight. This piece of practice has cost several persons their vision. A gentleman belonging to the class mentioned to me the other day two cases of purulent ophthalmia so produced, which he had seen among Mr. Guthrie's patients at the Ophthalmic Hospital. In the one, a young woman, not so healthy as she ought to have been, used her own water; in the other, an older woman, for what reason it did not appear, preferred her husband's to her own. Mr. Lawrence alludes to several similar cases. He details an instance also, in which partial sloughing of one cornea occurred; the disease having been caused by the patient's wiping his eyes with a towel soiled with the gonorrhœal discharge from his own urethra. But one of the neatest and most conclusive instances of the production of the disease in this way has been furnished by Dr. Mackenzie. A patient was brought to him from the country with his left eye violently inflamed and chemosed, and discharging a large quantity of purulent fluid; the lower lid everted, and the cornea totally opaque. Thirteen days before, this man, who had then

a profuse gonorrhœa, but whose eyes were perfectly well, while stooping down and shaking away the discharge from his penis, flung a drop of it fairly into his left eye. Violent inflammation immediately set in, was confined to the eye that was thus inoculated, and produced the results I have mentioned: the gonorrhœa going on just as before.

"Numerous authentic cases have been recorded of gonorrhœal ophthalmia produced by the application to the eye of gonorrhœal matter from another individual. Mr. Wardrop met with the following example: An old lady went into the dressing room of her son, who had gonorrhœa, and washed her face with a towel which he had recently been making use of. Purulent ophthalmia quickly supervened, and destroyed the eye in a few days. Delpech mentions the instance of a young and healthy woman, who bathed her eyes with goulard water, by means of a sponge which had been used by a young man who had a clap: violent inflammation soon arose, and the sight of one eye was lost. Several cases of purulent ophthalmia have been observed in laundresses, who had been employed in washing linen foul with the discharge of gonorrhœa."

Mr. Lawrence seems to be of opinion that purulent ophthalmia is not a very frequent consequence of the application of the urethral discarge to the eye of the same person. "When we consider," he says, "how this matter is diffused over the linen of patients, both male and female, how often the fingers must be smeared with it, and how inattentive to cleanliness the lower classes are, we cannot help concluding that the gonorrhœal discharge must be *often* applied to the eyes of the same individual; yet gonorrhœal ophthalmia is comparatively rare." Dr. Mackenzie, on the other hand. thinks that the application of the matter to the eye is seldom made. "The instinctive closure of the eyelids," he observes "when the finger approaches the eye, making it actually difficult for a person to touch his own conjunctiva, unless with one finger he draws down the lower lid, and intentionally applies another finger to the eye, will serve in some measure to explain the rarity of this kind of inoculation," (Lecture xviii.)

Diagnosis.—The principal difference between catarrhal and purulent ophthalmia is that the latter affects the papillary structure of the conjunctiva with more rapidity and intensity, and is thereby apt to become inveterate. "A patient," writes Dr. Mackenzie, "may remain for many months with the conjunctiva of the eyelids in a granular state, his cornea probably vascular and nebulous, but without any purulent discharge, when, after a fit of intoxication, or some other irregularity, the inflammation shall suddenly return to its original form, and with its original propagative power. Hence it may happen that a soldier discharged in the state described, returning home into the country, and then relapsing, may give rise to an ophthalmia which shall spread through many families, with all the symptoms and severity of the original disease."

Prognosis.—The less red and swollen the eyelids are the less severe is the ophthalmia, however abundant the purulent discharge may be; and the less the lids are altered during the acute stage the better is the prognosis; and their condition in the acute stage is a reliable guide as to the safety of the cornea and the general prognosis of the case. The more vascular and chemosed the conjunctiva, the more severe is the purulent ophthalmia; and the more likely is the cornea to participate in the inflammation, especially if *chemosis* is great. If the cornea is not completely destroyed, opaque cicatrices may follow perforation; and other secondary changes may take place during the later stages of the ophthalmia, such as pustules, chronic ulcerations, or vascular corneitis. The disease usually reaches its height in from three to eight days; but it may be greatly prolonged by passing into *pyorrhœa,* or into granular or chronic catarrhal ophthalmia. The sooner the loss of transparency or ulceration of the cornea occurs, the worse is the prognosis; and there is less hope of recovery of useful sight if the cornea is implicated before the ophthalmia has passed its height, or if the *chemosis* is already severe. On the second day from the commencement, when the patient begins to open the lids spontaneously, it is known that the ophthalmia is decreasing; although the suppuration may continue abundant for some time (BADER).

Treatment requires the adoption of antiphlogistic remedies and astringents.

If the disease be seen early, before the ocular conjunctiva has become vascular, or but slightly so, the alum lotion, or nitrate of silver lotion may effect a cure; but if *chemosis* be present, Dr. Mackenzie recommends general blood-letting from the arm or the temporal artery, followed by leeches round the eye to the number of six to twenty-four within two hours after the general blood-letting. If in the course of twenty-four hours the symptoms do not abate, but, on the contrary, if pulsative pain commence in the eye, with circum-orbital pain coming on in nocturnal poroxysms, the repetition of the general blood-letting is necessary.

On the other hand, Dr. Bader's treatment is essentially local, its chief object being to arrest the purulent discharge, and to protect the cornea.

If gonorrhæal pus is known to have come in contact with the conjunctiva, it is safest to touch the entire palpebral conjunctiva with *solid nitrate of silver*; and the same treatment may be adopted at any stage of the ophthalmia, however great may be the swelling of the lids or conjunctiva. There can be no doubt that blood-letting has been carried to a very great extent in this disease; but Sir Thomas Watson justly observes that blood-letting is less likely to influence the disease, (1.) because the part affected is a *mucous,* and not a *serous* or *fibrous* membrane; and (2.) because the constitution does not participate (except as a result) with the local inflammation. It is only indicated in robust patients when the constitutional or inflammatory fever is high, with a hard pulse and increased action of the heart.

Dr. Mackenzie also recommends scarification of the eyeball and conjunctiva of the eyelids, repeated every second or third day, or even every day, as one of the most effectual remedies.

A searching purgative of jalap and calomel is to be given in all cases, and the patient must remain at rest in a well-ventilated apartment, the eyes being shaded from the light. After the *calomel* and *jalap* have acted, the *tartar emetic,* with *sulphate of magnesia,* will be found useful.

Mercury or *iodide of potassium,* in alterative doses, is also found useful in the severe cases, attended with nocturnal circum-orbital pain. Dr. Mackenzie considers that local remedies, such as astringents and escharotics, act with more effect and less danger after depletion, than in cases where depletion is not employed. Local treatment, alternately of a soothing and stimulating kind, *must go along with* the constitutional remedies. The eyes must first be made clean, and free of all discharge, by using the following lotion repeatedly during the day:—

℞. *Corrosive sublimate*, gr. i.; *Sal. ammoniac*, gr. vi.; dissolved in *eight* ounces of water; and to which two drachms of *vinum opii* may be occasionally added.

This cleans the eye, and acts as a gentle astringent; and is most efficient when injected into the sinuses of the conjunctiva with a small syringe, especially into the fold between the eyeball and the upper lid. "*Ntirate of silver* is the best remedy for constringing the inflamed vessels, allaying the painful feeling of sand in the eye, and lessening the discharge" (MACKENZIE). Ten grains to the ounce of distilled water is the most suitable strength (RIDGWAY, MACKENZIE, GUTHRIE); and the solution may be applied every five or six hours, or as soon as the raw painful feeling in the eye is renewed. A quantity of the solution is to be taken up with a large camel-hair brush, with which the inside of the upper eyelid is first to be brushed over, and then that of the lower, not omitting any of the folds formed by the chemosed conjunctiva. Some use the solid nitrate of silver, with which the inside of the lids are rapidly touched once or twice a-day.

In applying these remedies, Dr. Bader recommends that chloroform be used in order to prevent straining of, or pressure upon, the eyeball and cornea; and during the administration of the chloroform the eye ought to be bound up to support it equally, and avoid the risk of perforation, if the patient should strain. The lids are then everted, and the conjunctiva is cleansed from pus with warm water, and wiped with dry lint. Deep incisions, so as to obtain abundant escape of blood, are to be made through the swollen conjunctiva, radiating from the periphery of the cornea, and from the margins of the lids. After this, the bleeding having somewhat ceased, the palpebral conjunctiva, especially the fornix, is touched with the solid nitrate of silver. The surface of the conjunctiva then becomes grey, white, and opaque, and covered with flakes of a similar colour; and warm water must be used to wash away the free particles of nitrate of silver. After replacing the lids, the patient must be put to bed with a bag of ice laid upon the eyelids to keep them as cool as possible. The lids are to be gently separated every quarter of an hour, and some alum lotion poured upon the exposed conjunctiva, to wash away accumulating discharge.

Dr. Bader also recommends, in cases where *chemosis* is great, and the cornea in part or entirely opaque, that an incision be made through the outer canthus (cutting through skin and *orbicularis muscle*), so as to relax the eyelids, thus removing pressure from the cornea, and inducing a free escape of blood. The white sloughs may be thrown off in two or three hours, and, purulent discharge again appearing, the cauterization may have to be repeated, *unless the surface of the cornea has become vascular.* Lint dipped in ice-water, and the use of the *alum lotion* (BADER), or *nitrate of silver lotion* (MACKENZIE) every quarter of an hour during the day, and frequently at night, continued as long as there is increase of purulent discharge, and until the patient is able to open the eyelids spontaneously. The use of these lotions may have to be continued from six or eight weeks.

The citric ointment (*Ung. Hyd. Nitratis Mitius*) is the best remedy for preventing the sticking together of the lids. *Counter-irritation* must also be established by blisters on the temples, on the nape of the neck, or behind the ears. As soon as a counter-discharge is established, there is generally a marked change in the quantity and quality of the discharge from the eye.

The eye not affected should be kept bound up as long as there is the least purulent discharge from the inflamed eye. The bandage should be removed from time to time, and the skin of the eyelids washed with warm water, and afterwards smeared with spermaceti ointment to prevent eczema (BADER).

The patient's friends must be warned as to the contagious and virulent nature of the disease.

Prevention.—Dr. Mackenzie writes on this important heading as follows:—To military surgeons, especially, the means of preventing this destructive disease are of high importance. Some of the following rules they will at all times be able to follow; the others must depend on the higher military authorities:

1. Supposing troops to be sent to any of the countries where this disease prevails, it would be necessary to guard them as much as possible against the exciting causes of catarrhal ophthalmia, in which it appears that the contagious disease originates. It is found in Egypt that exposure to the night air is extremely apt to bring on the ophthalmia of the country. Soldiers on guard there, or at bivouac, should, during the night, cover their heads well; and, if in moist and cold situations, avoid currents of air as much as possible. Dr. Vetch mentions that of four officers who slept in the same tent, in Egypt, two took the precaution to bind their eyes up every night when going to rest, and the two others did not; the latter were in a very short time attacked by the disease, while the other two escaped.

2. Heavy caps and tight stiff collars ought to be laid aside.

3. As soon as there are any appearances of puro-mucous ophthalmia in a regiment, a daily and minute inspection by the medical officers of every individual belonging to it becomes a duty of the first moment, both for the sake of those who may have caught the disease, and for the sake of their comrades.

4. Those in whom the disease is detected should instantly be separated from the rest, and not be allowed to join their companies till they are perfectly cured, and have passed several weeks in an establishment removed some miles from the place where they were attacked.

5. Those patients who are found to be liable to frequent relapses, or who are affected with obstinate granular conjunctiva, should be invalided or sent to a distance.

6. Excessive crowding of the men together, especially in their dormitories, must be carefully avoided, as this of itself appears very much to promote the contagious power and spread of the disease, and to prevent its cure. A well-ventilated hospital, in a wholesome open situa-

tion, is to be chosen. The beds are to be placed asunder. Proper means for disinfecting the air, clothing, utensils, &c., are to be adopted.

7. Those exposed to the disease ought to be made acquainted with the fact of its contagious nature, and warned against the modes in which it is likely to be communicated—as touching the eyes of the diseased person, and then touching inadvertently their own, using the same towel as those affected with the ophthalmia, and the like. Barrack towels must afford a constant medium for the communication of this disease; they ought, therefore, to be discarded, and every man furnished with a towel for himself.

8. It will be found a salutary practice frequently to parade the men, in their respective companies, each with a separate vessel of water, while an officer attends to see their faces and eyes carefully washed.

9. A regiment attacked by the ophthalmia should move from the station where the disease seems to be epidemic.

10. If the number be great who have suffered from the ophthalmia, they should be formed into a battalion, into which no fresh recruits are to enter, and which should be removed to a wholesome locality, and not re-admitted into the service till after several months' separation.

PURULENT OPHTHALMIA OF INFANTS—*Syn.*, OPHTHALMIA NEONATORUM.

LATIN EQ., *Ophthalmia infantum purulenta*—Idem valet, *Opathalmia recens natorum;* FRENCH EQ., *Ophthalmie purulente des nouveaux-nés;* GERMAN EQ., *Purulente Augenentzündung der neugeborenen;* ITALIAN EQ., *Oftalmia purulenta nè bambini.*

Definition.—*Purulent ophthalmia appearing generally on the third day, or within a week after birth, but sometimes not for three or four weeks, and with symptoms similar to those in adults.*

Pathology.—The disease is a very common one; but if neglected it may become a very serious one, and is perhaps the most fertile source of blindness with which we are acquainted. The disease is sudden in its attack, like most contagious diseases, and much more violent than in catarrhal ophthalmia, and in this respect resembles the Egyptian ophthalmia, or the gonorrhœal inflammation of the conjunctiva. It originates most commonly in contagion:—

(1.) By inoculation of the conjunctiva from leucorrhœal fluid (generally acid) during parturition; and thus it may be prevented by repeated injections of tepid water, or weak alkaline solution, into the vagina in the first and second stages of labour, and by carefully washing the eyes of the infant with luke-warm water as soon as it is removed from the mother.

(2.) In its worst form it may result from contact of the conjunctiva with gonorrhœal matter during the passage of the child's head through the vagina; and the same precautions are to be used as in the already named mode of origin.

(3.) It may be induced by exposure to the light, to the heat of the fire, or to the cold draught from the door (catarrhal).

(4.) By intrusion into the eyes of the soap with which the child's head is washed, or of the whisky or gin with which the lower classes are wont to bathe the infant's head, from an absurd belief in its strengthening virtues.

Symptoms.—On the morning of the third day after birth, the infant may be observed to have what the mother and nurse may call a "cold in the eye"—the upper eyelid being somewhat swollen, its edge red, and the eyelashes glued together by concrete purulent matter.

The inside of the eyelids, especially the palpebral conjunctiva and fold formed by its reflection to the eyeball, are extremely vascular and swollen; and for some days a thin mucous or serous discharge may flow from them; but always becoming distinctly purulent at a very early period—as early as the third day (MACKENZIE).

Generally both eyes are affected, at a short interval of time between the affection of the one eye and the other.

The infant keeps the eyes constantly shut, and in this state the eyes may continue for eight or ten days without any affection of the transparent parts, except, perhaps, slight haziness of the cornea and redness of its edge. By the twelfth day the cornea may become opalescent, indicative of commencing degeneration; it may be infiltrated with pus, by which its texture is speedily destroyed, when it gives way by ulceration, and generally the lower part of the iris protrudes through the ulcer. In other cases the whole cornea disappears, exposing the *iris*, with the humors bulging through the pupil, and the lens may fall away, or be discharged through the ulcerated cornea. Dr. Mackenzie relates an instance in which a poor woman brought her child, aged five weeks, for consultation, having been told by her midwife that the disease was common, and not at all dangerous; and with her she had wrapped up in a bit of rag the left crystalline lens, dry and shrivelled up, which had that morning been discharged through the ulceration of the cornea. Dr. Mackenzie put it into water for a few hours, when it regained its plumpness and transparency. He could then see it was enclosed in its capsule, and its texture was quite distinct under the microscope. The right cornea in the same child was opaque and partly ulcerated.

Prognosis.—The complaint is completely within control, if taken in time and properly treated. If it can be seen that the *corneæ* are still brilliant and uninjured, free from ulceration and purulent infiltration, however violent may be the inflammation and profuse the discharge, the prognosis is favourable so far, and the sight in general is safe; but if the corneæ have become opalescent, the prognosis must be unfavourable.

The attempt to see the cornea (unless chloroform be given) may lead to perforation, with escape of the crystalline lens and vitrous humor, and loss of the eye. A view of the corneæ, even if obtained, does not affect the treatment of the cases, but is necessary as to prognosis (BADER). The disease is also more difficult to overcome if the nurse drinks spirits or ales, the child being improperly fed and ill nourished. Specs and opacites on the corneæ may be an inevitable result, or *staphyloma* may remain, greatly impairing vision. "Whenever the person who brings the child to me," says Dr. Mackenzie, "announces that the disease has continued for three weeks or longer without anything having been done for its relief, I open the lids of the infant with the fearful presentiment that vision is lost, and but too often I find one or both of the corneæ gone, and the iris and humors protruding. In this case it is our painful duty to say that there is no hope of sight."

Dr. Bader is of opinion that children may recover useful vision though the entire corneæ, except, perhaps, the posterior elastic laminæ, may have been destroyed; or though a large abscess, or an opacity occupy three-fourths of the surface of the cornea, or a large prolapse of the iris may have been present. An opacity occupying the entire cornea, its curvature being otherwise normal, *may* disappear in about two years without treatment. When the disease arises from gonorrhœal inoculation, the prognosis is doubtful, for unless the disease is taken in hand very early, and treated very energetically, one or both eyes are likely to be lost.

Central capsular, or capsulo-lenticular cataract, is by no means an uncommon result of the ophthalmia of new-born infants, even in cases where there has been no penetrating ulceration of the corneæ. But as the child grows, vision sometimes improves, in consequence of the expansion of the pupil, and if the cataract remains of its original size.

Treatment.—The eyes must be well washed out three or four times or oftener in the twenty-four hours, by the bichloride of mercury and salammoniac lotion, whose composition has been already given—the vinum opii being omitted (MACKENZIE); or by the solution of alum (BADER). It tends greatly to repress the discharge; but is not alone sufficient. *Solution* of the *nitrate of silver* is necessary, in varying strength, according to the state of the conjunctiva—from two to ten grains to the ounce of distilled water to be used every six or eight hours, the solution being applied to the whole surface of the inflamed conjunctiva with a camel-hair brush, as already described. In two or three days the eyes ought to open spontaneously, and in ten or twelve more the acute symptoms may be overcome. A case of moderate severity recovers in from six to eight weeks, and the use of the detergent lotions every two hours, for about three weeks, is sufficient, if the opthalmia is mild —*i.e.*, if the corneæ is clear and the lids open spontaneously (BADER). To prevent the eyelids adhering, *red precipitate ointment*, or the *ungentum hydrargeri nitratis mitius*, or the *glycerine* and *starch ointment*, may be used. The eye not inflamed should be kept bound up. It may be necessary, in severe cases, where the swelling of the lids is such as prevents the cornea being seen, to use the detergent lotions and nitrate of silver solution every five or ten minutes during the day, and every hour at night, for from three to five days, until a view of the cornea can be obtained (BADER). It may be also necessary to take blood by *one* leech from the external surface of the upper eyelid, or from the inflamed conjunctiva by scarification (MACKENZIE). The leeching must not be entrusted to the care of a nurse. The medical attendant must do this himself, and wait till the leech drops off, seeing that the bleeding is often difficult to arrest, and may injure the infant (WATSON).

Castor oil should be given to improve the condition of the intestinal canal; and Dr. Mackenzie considers small doses of calomel (from half a grain to one grain daily) as highly beneficial. Blisters behind the ears are also of great service. Mr. Lawrence's treatment consisted in *magnesia* internally, to keep the bowels open; a little lard along the edges of the lids, that they may not stick together, and a solution of alum (four grains to the ounce of distilled water) injected between and beneath the lids. So successful was this treatment, as witnessed by Sir Thomas Watson, that he would never think of employing any other—the simple and less severe plan being in general quite sufficient.

GRANULAR OPHTHALMIA *—*Syn.*, GRANULAR CONJUNCTIVITIS.

LATIN EQ., *Ophthalmia granulosa, vel trachomatosa*—Idem valet., *Trachoma.*

Definition.—*Multiplication of intra-follicular corpuscles of the conjunctival glands, with germinative cell-growth in the surrounding connective tissue, causing hypertrophy of conjunctival villi, roughness from "granulations," hyperœmia, swelling, and inflammation of the conjunctival membrane.*

Pathology.—It is a frequent and troublesome result of the puro-mucous ophthalmiæ already described, and in this sense may be considered as a form of *chronic ophthalmia.* The prominences on the conjunctiva, and which have been erroneously called "*granulations,*" have generally been believed to be the *villi* or *papillæ* of the palpebral conjunctiva, along with its glandular elements hypertrophied and altered by chronic inflammation. In the normal state the villi are visible under the microscope, although the conjuntiva is not injected; and in a well injected preparation they are visible to the naked eye. In this disease they have been considered as forming the round granules, sometimes of the same colour as the conjunctiva, and only very slightly prominent. These "*granules*" may be greatly elevated from its surface, and from their shape and gelatinous translucency resemble the spawn of fish or frogs. These primary granulations are situated immediately beneath the epithelium, and represent spherical or ovoid bodies, in which nucleated cells, similar to lymph corpuscles, are inclosed in the meshes of an extremely delicate reticulum of connective tissue. They are either surrounded by a well-defined layer of condensed stroma, interspersed with long fusiform cells, or merge diffusely into the tissues in which they are imbedded.

The prevalent differences of opinion regarding the histology of these so-called "*granulations*" are mainly referable to the views held by individual observers as to the structure of the conjunctiva itself—the presence or absence of closed solitary gland-follicles, as normal organs, being the cardinal point at issue—and the number of such glands. It was first announced by Bendz, at the Congress of Brussels, that the conjunctiva was normally possessed of such follicular gland-structures, identical with the closed follicles of the large and small intestines, and with the Malpighian corpuscles of the spleen. Improved methods of observation led Sappey, and C. and W. Kranse, in 1861, to recognize the constant presence of these structures in normal lids; an opinion subsequently confirmed by the injected specimens prepared by Dr. Fischer of the Prussian Army Medical Staff; and since then Dr. Philip Frank (late of the British Medical Staff) has constantly met with isolated follicular bodies in the sub-epithelial strata of the conjunctiva. The reader is also referred to Beales's *Archives of Medicine* for 1862, where a valuable paper by Staff-Surgeon Dr. Marston "*On Ophthalmia*" details the anatomy of the human conjunctiva. Closed follicles have also been observed by Bruch.

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* Not noticed by the College of Physicians.

The so-called "vesicular granulations" are now, therefore, not considered as mere hypertrophy of conjunctival villi, but are ascribed to multiplication (proliferation) of intra-follicular corpuscles frequently combined with germinative cell-growth in the surrounding connective tissue, especially under the influence of irritative agencies, which act upon the follicles after absorption through the epithelial cells (DR. PHILIP FRANK). Stromeyer has shown the prevalence of this follicular disease of the conjunctiva in animals living in a vitiated atmosphere; and the prevalence of granular conditions of the conjunctive in a body of men may be regarded as indicating a contaminated state of the atmosphere in which they habitually sojourn; and its prevalence ought to inculcate the necessity for rigorous sanitation before more serious forms of communicable disease have found their development.

Opinions are now divided as to whether the irritating agencies from ordinary organic effluvia impregnating the atmosphere of an overcrowded, ill-ventilated barrack-room, are capable of producing granular ophthalmia, being then merely a question of time and intensity of action, and whether increased vascularity and epithelial hyperplasia, or profound and persistent follicular changes should result; or whether, on the other hand, a specific emanation proceeding from the lids of affected individuals be indispensible for the production of granular ophthalmia. At the same time, if the perfect eradication of the disease is to be looked forward to, it is of paramount importance to recognize the existence and intense contagiousness of ophthalmia in all its stages. It ought to be considered as endemic in a regiment when cases of primary granulations are diffused in its ranks, even without any case presenting acute inflammatory symptoms being under observation at the time; and in the event of a soldier, who is known to have been the subject of "granular lids," becoming ineffective from the result of a complicated inflammatory attack, every effort ought to be made within the scope of active hygienic arrangements to counteract the spread of such a form of ophthalmia, and so, perhaps, obviate the development of "granular lids" generally in the regiment—a condition which renders the conjunctiva prone to suffer from the most trifling sources of irritation, such as from cold and dust. In 1860 and 1861, Dr. Philip Frank found at the Invaliding Hospital the primary changes constituting "granular lids" in men belonging to *forty-one different regiments,* invalided for other causes from home stations and foreign dependencies—a direct warrant for the belief in the very general diffusion of the lesion, a conclusion fully borne out by the subsequent excellent observations of Staff-Surgeon Dr. Marston, collected at Malta and Gibraltar, also by Surgeon-Major A. Leith Adams, and

Assistant-Surgeon F. H. Welsh, F.R.C.S., of the 22nd Regiment, more recently.

"In order to study the typical character of vesicular granulations," writes Dr. Phillip Frank, in his excellent Report for 1860, published by the Army Medical Department, "they must be sought for in the conjunctiva of apparently healthy eyes; and this has led to their existence being overlooked and denied by many who are not in the habit of examining the conjunctiva prior to the invasion of inflammatory symptoms. Innocent as these primary lesions may appear, they gain in importance by the predisposition they engender to inflammatory attacks, and by the contagious nature of the secretion which, under such circumstances, is furnished by the affected conjunctiva. Exposure to atmospheric vicissitudes and other sources of irritation, which, in a healthy conjunctiva, would lead to an attack of catarrhal inflammation or ordinary conjunctivitis commensarate in its intensity with the nature of the exciting cause, is prone to produce most obstinate structural changes in lids affected with vesicular granulations, on account of the great tendency of these bodies to assume a more advanced structural development under the influence of inflammatory irritation. In many cases, fortunately, the vesicular bodies apparently retrograde, either spontaneously or under the influence of treatment, without leading to any persistent structural lesion. In others a further development of the trachomatous process may take place in such an insidious manner as only to reveal itself by the atrophic changes which constitute its ultimate result. Without any obvious inflammatory attack—without any apparent departure from the normal standard of secretion—the deeper parts of the conjunctival stroma may become the seat of deposits similar to those originally formed in the subepithelial layer. Shrinking of the tissues finally supervenes from atrophy of the cell-brood, which was created at the expense of their proliferating elements. The possibility of this form of atrophy (with which all observers of trachoma have been long familiar as a sequela of inflammatory complications) occurring in the manner described, was first pointed out to Dr. Frank in the wards of Fort Pitt, by Professor Junge, of St. Petersburgh, in a very illustrative case. Well-marked conjunctival shrinking, with nearly perfect obliteration of the retro-tarsal folds, rounding off of the inner margin of the lids with incipient entropion from incurvation of the tarsal cartilages were observed in a soldier invalided for nystagmus and myopia, who most distinctly denied having ever been affected with any form of conjunctival inflammation."

"In other cases—and it is to them that the interest of the military surgeon more particularly attaches itself—the fate of the primitive granulations, and of the conjunctiva affected with them, will depend upon the nature and course of the inflammation by which they may be complicated."

"The superinduced inflammation may either be of *phlegmonous, purulent,* or *catarrhal form;* and, in addition to its influences in promoting the organization of the pre-existing vesicular bodies, which soon represent vascular beads of more or less dense fibro-plastic tissue, may lead to general hypertrophy of the conjunctival elements and to granulations and excrescences of various forms, which may altogether mask the original growths and their metamorphoses, rendering it impossible in a given case to decide whether follicular disease had existed prior to the attack.* Generally, however, when the products of the acute inflammatory complications commence to subside, the more or less altered trachomatous growths are again visible, mostly as discrete, greyish, gelatinous spherules, embedded in the thickened conjunctiva, or forming hemispherical or conical projections, entirely distinct in appearance from the hypertrophied papillæ or ordinary villous excrescences of the palpebral lining. Even in this more advanced stage of organization the lids may again become perfectly quiescent. In other cases severe purulent inflammation will lead to an absorption of the original deposits, which is easily explicable by the melting down of the tissues during the course of profuse suppurative action."

Attention to these primary conjunctival changes is demanded alike in the interest of the individuals and of the community to which they belong. Their individual existence constitute so many *foci* from which a purulent epidemic of ophthalmia may at any time arise. The conjunctival deposits may exist for an indefinite period without giving rise to any inflammatory symptoms, till, in fact, an exciting cause of sufficient interest brings on the acute inflammatory attack, and a "granular" state of the lids is always a potent source of relapse and ophthalmia.

The state of the conjunctiva, as to the freedom from or existence of these "granulations," of every man in a regiment ought to be noted in his medical history sheet with as great precision as is possible. It would furnish most important information at subsequent periods. In illustration of this, Surgeon-Major A. Leith Adams, M.B., and Assistant-Surgeon F. H. Welsh, Esq., F.R.C.S., of the 22nd Regiment—stimulated, no doubt, by Dr. Phillip Frank's excellent paper already noticed—carefully inspected the eyes of each man in the first battalion of the 22nd Regiment, the object being to ascertain the existence or not of the so-called "*granules;*" to note the actual condition of the eyelids of each man as a reference in case of any outbreak of *granular ophthalmia* among them, it being then present in the battalions of the garrison (Malta); to determine also, if possible (should the "*granules*" exist), the actual conditions under which the men were living; and to endeavour to arrive at the cause or causes of their origin and development. The battalion had been three years in the island, had occupied its present quarters two years, and been remarkably free from *ophthalmia* or *conjunctivitis* since its arrival (90 cases out of 808 men in three years).

The number of men inspected was 751, and of these 52·1 *per cent.* presented clear and undoubted evidence of the existence, to a greater or less extent, of "granules" in their eyelids—a perfectly healthy condition being the exception—except in a company occupying huts, and among the married, or those occupying separate quarters. Of those residing in a certain barrack (Florinna) only one could be said to have had a healthy conjunctiva. Among those occupying huts, the proportion of healthy increased with the length of time they had been away from head quarters, and the chances they had had of sleeping in a purer atmosphere (*A. M. D. Report* for 1863, p. 494).

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* The influence of a hyperæmic condition in masking vesicular granulations can be easily appreciated by their disappearing as it were before the very eyes of the observer, when a lid has been kept everted for a short time, and reappearing again when the congestion has been allowed to subside.

Symptoms are to some extent explained at first by the faulty secretion from the granular conjunctiva and by changes on the surface of the cornea. These symptoms are especially impairment of vision; the patient complains that he "cannot see," that the "sight is misty," that "a mist" and "rainbow colours appear round the flame of a candle," and that after sleep or after exertion the eyes are "red and weak." But as a rule, men do not complain, and may not admit that anything is wrong with their eyes.

The granular prominences (so named from the resemblance of the surface of the conjunctiva to a granulating wound surface) vary in different cases. In some cases they are exceedingly numerous, slightly raised above the level of the conjunctiva, giving the inside of the lids an appearance somewhat like that of a piece of *shagreen;* in other cases the granules are comparatively few, but prominent, very soft, vascular, and apt to bleed—often as large as hemp seeds.

On inspecting the eye generally, before opening and everting the lids, in nine cases out of ten no departure from the healthy state will be seen beyond an occasional slight collection of dried secretion at the roots of the lashes, and in the severe form a somewhat increased fullness of the larger sclerotic vessels. In these hyperæmic cases the glueing of the lids and the watery eye, with augmented mucus and tears, are causes of discomfort, the inconvenience being generally in proportion to the hyperæmia; but as a rule the eye remains quiescent when the causes of irritation in the lids have been gradually produced (ADAMS and WELSH).

A powerful reading-glass is necessary in examining lids for granules in their earlier or vesicular stages.

Dr. Bader distinguishes several species of granulations by the following characters:—

(1.) *Little granules, of the size and appearance of soaked sago grains, standing side by side, either in groups or singly, and projecting from the nearly normal or more or less vascular conjunctiva.*

These appear first in the fornix, and are seen in their purest and most typical form in cases which have never been treated. They consist of gelatinous, transparent, intercellular substance, covered by young proliferous epithelial cells and some connective tissue. They are surrounded by enlarged elongated but small blood-vessels, which seem to bear some relation to the arrangement and growth of the granules. They gradually increase in number, and may exist for years without impairing vision. They may subsequently assume the aspect of—

(2.) *Vascular "red" granulations of different sizes—largest in the fornix—with confluent bases resting upon the vascular aud hypertrophied conjunctiva, which in severe cases appear infiltrated with a semi-transparent gelatinous-looking substance; but in slighter cases the sub-conjunctival tissue is not much implicated, and the granular surface of the conjunctiva may be thrown into folds.*

These differ in structure from the species already described, in exhibiting a greater proliferation of epithalial cells, a greater accumulation of the gelatinous intercellular substance, and a higher degree of vascularity. The increase of intercellular substance, with dilatation and increase of conjunctival blood-vessels, and hypertrophy of connective tissue corpuscles, cause swelling of the conjunctiva between the granulations.

(3.) *Large pale-grey, grey-white, or yellowish-grey, and opaque granulations, whose shape, size, and subsequent changes are similar to those of the red ones.*

They rest upon the infiltrated conjunctiva and sub-conjunctival tissue, and reach their largest size in the fornix, and along the upper margin of the tarsus. A few large blood-vessels may be seen among them. The secretion from the eye is less than from the red granulations.

(4.) *Small, easily bleeding, flabby granulations of equal size, which stand side by side, and give the surface a somewhat rough villous appearance. The conjunctiva covering the tissue is swollen, intensely red, and easily bleeding.*

This condition is confined to the papillous portion of the tarsal conjunctiva, while the conjunctiva of the fornix, the semi-lunar fold of the caruncle, are swollen and highly vascular. Sometimes there is much muco-purulent discharge. These granules generally occur in young persons in both eyes simultaneously; and Dr. Bader considers it probable that they are of syphilitic origin.

(5.) *Large, flabby, red, and rather hard and elastic granulations of varying size, resembling condylomata in shape, with hardly any or no purulent discharge.*

They occupy the palpebral conjunctiva, and frequently also the semilunar fold (Hypertrophy of *racemose glands?*). In the fornix they may reach several lines in diameter. They often continue for years without impairing the transparency of the cornea; but when very numerous, they may prevent the eyelids from closing, and cause them to stand away from the eyeball, and sometimes even to become everted. Not unfrequently they are confined to the conjunctiva of one eye only. The sub-conjunctival tissue is much hypertrophied.

This form is rare, and occurs by preference in young persons suffering from glandular swellings in other parts.

Numbers (1), (2), and (3) are closely allied; but treatment and time alter their typical appearance; numbers (4) and (5) are wholly different from each other and from the other varieties.

Dr. Adams and Mr. F. H. Welsh, F.R.C.S., describe three varieties or stages as seen by them amongst soldiers of the 22nd Regiment, at Malta.

(1.) *Vesicles requiring very careful attention for their detection, having the appearance of small, glistening, transparent, hemispherical bodies studding the mucous membrane and projecting above its surface.*

Their size was that of a very small pin's head, their contents translucent and limpid, looking like tense bladders of water. No unusual vascularity existed with these vesicles.

(2.) *Of larger size than* (1), *and differing somewhat in character; their contents being opaque, milky, and more glutinous in consistence.*

They did not project so much above the mucous membrane, because it too was invariably swollen and injected, and so was raised to the level of the "*granules.*"

(3.) *The "granules" appeared like cut globules of opaque jelly, studding* *a red ground-work. They were solid, and had increased in size so much, and so encroached on each other, as to leave only very little interspaces of mucous membrane.*

These stages or varieties seem to be preliminary to the formation of the more solid "*sago grain granulations.*"

If not disturbed by treatment nor intercurrent attacks of inflammation, the sago grain-like granulations may remain for one or two years without disturbing vision, and may only occasionally give rise to "redness of the eyes;" but attacks of ophthalmia gradually become more numerous and severe, and the granulations change colour, becoming red from vascularity. When the surface of the cornea suffers, its upper portion first becomes vascular, probably from friction of the granulations against its surface. The vessels then encroach from the adjacent conjunctiva.

The disappearance of the granulations, either by treatment spontaneously, by rapid elimination of their contents and absorption, lead to destruction of a certain amount of conjunctiva, so that deep cicatrices remain which occupy its entire thickness, and the surface of the cicatrix is anæmic, whitish, or tendinous like.

The conjunctiva in the granular state often secretes an inordinate quantity of mucus, which on any additional irritation of the system, as from the use of spirituous liquors or any local irritation, as from cold affecting the eyes, is apt to become again puriform. When this is the case, the contagious power of the original ophthalmia returns.

Eventually the constitution suffers materially from the persistence of "granular lids."

Prognosis.—Relapses are frequent, especially from intemperance, cold and damp, imperfect ventilation, especially of sleeping rooms; deficient food. In scrofulous subjects the condition, once fully established, is incurable. The granules of the lids become really tubercles.

Treatment is by *local depletion, astringents, escharotics, counter-irritation, tonics, excision,* and *inoculation.*

Dr. Mackenzie recommends local depletion by a few leeches, if the conjunctiva is very vascular and sarcomatous. The leeches are to be applied to the external surface of the lids. He also recommends scarification of the lids; but if the membrane is not much thickened, each granular prominence may be merely divided by a crucial incision, or the membrane "cross-hatched" by slight touches of the lancet. If the lids are much swollen and tense, and seem to press on the cornea, Dr. Bader recommends an incision, about half an inch in length, to be carried through the outer canthus, and that the wound be allowed to bleed freely, and prevented from closure for a week at least.

The object of treatment by *astringents* and *escharotics* is not to destroy the granulations directly, but to excite such irritation in the conjunctivæ as will promote elimination of the material of which the granulations are composed.

The agents most frequently in use are crystals of *borax, sulphate of zinc, nitrate of silver,* and *sulphate of copper,* or its mixture with *alum,* constituting the "green stone," or *Lapis divinus* (see page 423 or 424). The *neutral acetate* or *sugar of lead,* in the form of a powder, has also been used. It is to be applied over the surface by means of a miniature pencil, and allowed to dissolve in the tears. It causes strong contraction of the diseased tissue; the granular prominences shrink, and the membrane appears smooth and uniform.

It is to be applied at intervals of five or six days, till the cure is accomplished. Dr. Bader considers the *sulphate of copper* in glycerine ointment as likely to be of use. The green stone application is the most efficient remedy, care being taken to touch the granulations only, and not the conjunctiva between them, nor the cicatrices, if any exist. It must be repeated every day, or every second or third day, till the granulations have become smaller and less numerous, when "touching" twice a week may suffice.

Inoculation, exciting in the diseased membrane of the conjunctiva the inflammatory action of an acute attack of *purulent ophthalmia,* was suggested by Dr. Henry Walker, as a means of dispersing the granulations. During the ophthalmia the granulations become smaller and smaller, and the vascular covering which forms on the cornea is thrown off in yellow and opaque shreds. In order to excite such an inflammation, the conjunctiva is to be inoculated with matter taken from the eye of a child suffering from purulent ophthalmia,—

> "Take thou some *new infection to thine eye,*
> And the rank poison of the old will die."

The matter is to be applied to the inside of the lids with a miniature pencil; and the first signs of the inoculated ophthalmia ought to appear in from twelve to thirty hours; and however severe it may seem to be, the discharge should merely be washed away with tepid water every hour, and the greatest attention paid to cleanliness. The acute stage will last from one to four weeks, and the subsequent chronic purulent discharge from two to eighteen months, before the granulations will have disappeared. The curative effect may be considered at an end as soon as the eyelids are no longer glued together after sleep.

Inoculation should be confined to those cases in which the entire surface of the cornea, or two-thirds of it, are vascular, and more or less opaque, and the iris barely visible (Bader).

There are also important contra-indications to the practice, such as the existence of *scrofula, rheumatism,* or such like constitutional state of ill-health (Mackenzie).

Towels, lint, sponges, &c., used by patients who suffer from granulations, must not be used by any one else; and the hands must be washed after having touched eyes so affected. The "*authorities*" of schools, barracks, and the like, should be made aware of the contagious properties of "*granular ophthalmia,*" and those suffering from "granular lids" should be kept separate from others not so infected.

The reader is referred to most valuable practical papers by Surgeon-Major A. Leith Adams, M.B., and Assistant-Surgeon F. H. Welsh, Esq., F.R.C.S., in *Army Med. Dep. Reports* for 1869, vol. xi., pp. 423 and 436. And for an account of some other forms of *ophthalmia,* not noticed by the College of Physicians, reference is made to Dr. Mackenzie's classic work "On the Eye," especially to *post-febrile ophthalmitis, idiopathic ophthalmitis, phlebitic ophthalmitis, sympathetic ophthalmitis,* and *intermittent ophthalmia.*

SECTION III.—DISEASES OF THE CORNEA.

KERATITIS.

LATIN EQ., *Keratitis;* FRENCH EQ., *Kératite;* GERMAN EQ., *Hornhautenzündung;* ITALIAN EQ., *Cheratitide.*

Definition.—*Inflammation of the cornea.*

Pathology.—The structure of the cornea comprises—

(*a.*) Several layers of epithelial cells anteriorly, whose aggregate thickness amounts to $\frac{1}{1000}$. These rest upon an elastic lamina, against which a layer of oblong cells are arranged perpendicularly; and, while the superficial layer is flat, there is interspersed a layer of transitional roundish or angular cells.

(*b.*) Transparent, colourless, elastic laminæ, flexible and homogeneous membranes, intervene between the epileptial layers and the lamellated tissue of the cornea. These *laminæ* appear on section as well defined transparent lines, lost sight of in the texture of the *sclerotic* and in the thickness of the *cornea.*

(*c.*) The bulk of the cornea is made up of short lamellæ, in apposition with each other, and arranged somewhat parallel with the surface. Between the lamellæ cells occur ("*corneal corpuscles*") which are mononucleated astomose with each other.

(*d.*) A close net-work of *nerve fibres,* with *ganglionic cells* (which anastomose with the ciliary nerves), lie immediately beneath the anterior elastic lamina; but it is stated that the cornea receives most of its nerves, not from the ciliary ganglion, but directly from the *fifth nerve.*

All of these tissues are liable to be affected by inflammatory changes, from the parenchymatous or "cloudy swelling" of minute tissue up to the crude change obvious to unaided vision. These crude changes are always associated with congestion in the surrounding conjunctiva and sclerotic, and especially marked in the anastomosing wreath of blood-vessels close to the edge of the cornea. The exudation visible in the cornea itself affects most frequently its external lamellæ, sometimes its proper substance, and always impairing its transparency. When blood-vessels become developed in the exuded material, then the cornea appears red. But how many minute changes go on in these delicate and minute tissues before these more obvious events take place? And the disease increasing beyond these obvious events, there may eventually occur such changes as suppuration, ulceration, or a gangrene of the corneal textures.

In most of the ophthalmiæ already described, the *cornea* may participate to such an extent that it may be infiltrated with pus, or destroyed by ulceration, or its transparency injured by constant irritation (as of granular lids), producing a vascular and nebulous state of its surface. It is also a common seat of the phlectenulæ which attend scrufulous conjunctivitis, and layer after layer may be penetrated by ulceration. In *variola,* abscesses may form in the *corneæ,* and lead to loss of vision.

In ophthalmia from cold and rheumatism, the cornea may become ulcerated on its surface or infiltrated with pus. Inflammations of the cornea are greatly promoted by weakness of constitution, as well as by constitutional ill-health, and occurs most frequently in scrofulous subjects. Most frequent in the young: it is seldom seen after middle life. Three forms or varieties of *keratitis* are commonly met with, namely:—

(1.) *Syphilitic keratitis*, which declares itself most frequently between the ages of nine and fifteen—and in girls oftener than in boys—as a result of inherited syphilis, and associated with syphilitic indications in other parts. It may be found in such cases that the mother has had miscarriages or still-born children previous to the birth of the one suffering from *keratitis*; and also that the patient has suffered from other symptoms of syphilis during infancy, such as *snuffles*, a sore mouth, ulcers round the *anus*, skin eruptions, and the like (Bader).

The physiognomy of such patients is characterized by a generally old, pale-looking face—a squarish forehead, with the bridge of the nose frequently wide and depressed. The shape, size, and colour of the permanent incisor teeth are often also peculiar, as pointed out by Dr. Hutchinson. They are small, narrow, squared, of a yellow colour, and more or less deeply notched in a vertical direction.

Syphilitic keratitis generally appears in both eyes, within an interval of a few days or weeks; and may take a chronic course, continuing for several years—at one time being completely opaque or crimson red, and again recovering its transparency after a few weeks (Bader).

It is generally on account of impaired sight simply that such patients seek advice; and the corneal changes may be so great as to reduce vision to a mere perception of light. The impaired vision may be due to the following lesions:—*Opacities in the cornea; alterations of its curvature; opacities in the crystalline lens; changes following choroido-retinitis; inflammation of the optic nerve; or such cerebral changes as hydrocephalus.* Portions of the cornea may be also semi-transparent when the patient is disturbed by the dazzling due to diffusion of light. The vascularity of the ocular conjunctiva is but slightly increased; while a more or less broad pink zone in the sclerotic round the edge of the cornea is usually present.

The centre and lower half of the cornea are the first parts to lose transparency. Small, ill-defined, grey, yellowish, or brownish opaque dots, in groups or singly, appear in its lamellated structure about these parts. The surface of the cornea appears dull or slightly uneven, as if pricked with a pin in numerous places. Within a few weeks transparency may be so impaired that the cornea looks like a piece of ground glass, the depending portions being the more densely opaque, or of a pink or crimson hue, most intense at the surface and periphery of the cornea, and caused by blood-vessels passing from the substance of the sclerotic between the lamellæ of the cornea. The vessels tend to run parallel.

If the syphilitic infection is severe, or if health be feeble, lymph is abundantly developed in the cornea and iris, rendering the cornea yellow and the iris opaque. Permanent opacity may follow their adhesion, occupying most frequently the lower half of the cornea, and faint opacities may remain for life.

With the cornea other tunics are generally implicated; and after corneitis has subsided, the iris presents a characteristic and permanent steel-grey hue, and the ophthalmoscope may disclose changes in the transparency of the lens; its nucleus becomes prematurely large, yellowish, and strongly light reflecting, while dotted opacities occur on its surface beneath the capsule. Changes in the vitreous humour, chronic suppuration of the choroid and vitreous substance, with subsequent softening, shrinking of the eye, and consequent loss of sight are among the extreme results of syphilitic keratitis.

(2.) *Strumous corneitis*, *scrofulous ophthalmia*, or *vascular corneitis*, is associated with the general morbid condition described as scrofula, (page 17, *ante.*) It occurs most commonly in children from eight to eighteen years of age, and in females shortly before or about the age of puberty. There is generally pain in the affected eye, sometimes severe, and extending over the corresponding side of the head and face. The eyelids are generally red along the margins, slightly swollen, and tending to spasmodic closure, when there is abundant flow of tears and intolerance of light. With these symptoms there are generally one or several small ill-defined opacities in the cornea. Opaque and slightly vascular spots at or near the centre of the cornea, at the commencement of the attack, are seen to advance from the margin towards the centre. These are generally superficial, but in weak persons they may extend through the entire thickness of the cornea. The parts of the cornea affected appear grey, greyish-white, or yellowish and opaque, shedding off to the transparent parts, or merging into the sclerotic; and the vascularity of the sclerotic and conjunctiva is most marked next the inflamed part (BADER). In some cases the whole cornea is so much covered with innumerable blood-vessels that it assumes a red colour, and in this state it has been compared to a piece of red cloth, an appearance known by the name of "*pannus*" (MACKENZIE). Ulceration with perforation may occur (BADER).

(3.) *Pustular corneitis*, *phlectenular*, or *herpetic corneitis*, seem to be other forms of the affection generally also associated with *scrofula*, and occurring most frequently in young persons and children. There is extreme intolerance of light, which may continue for months, with slight redness, swelling and spasmodic closure of the eyelids—abundant flow of tears and frequently eczema of the surrounding skin, although the changes in the cornea may be very slight. These changes consist in the occurrence of one or several roundish, small, somewhat projecting opaque nodules in the superficial layers of the cornea. If multiple, they may exist in groups or in rows near the margin of the cornea.

One nodule, or group of nodules, may appear at the apex of a bundle of blood-vessels, diverging over the cornea, thus forming a vascular triangle, ultimately lost in the adjoining conjunctiva.

These nodules often appear first in the conjunctiva, as already described under the head of "*pustular ophthalmia*," or their appearance in the cornea is preceded by attacks of ophthalmia with sclerotic vascularity and intolerance of light. Nodules, pustules, and patches of vascular corneitis, with small ulcerations and opacities, may all exist together, and give a most complicated appearance to several typical lesions. The solid nodule may resemble a pustule from the elevation of layers of corneal epithelium; and suppuration of the nodule may really take place within a few hours, and so change the nodule into an ulcer, which may lead to perforation. The vascularity of the sclerotic and conjunctiva varies with the nature of the several existing lesions; and according to the severity of these several lesions in combination, will be the prolonged duration of the case; and this form of corneitis tends to return for years at about the same season, or at any time when the general health is impaired. The disease also frequently occurs *after scarlatina, measles,* and *variola,* and is one of the forms of *post-febrile* ophthalmia. It has been considered that derangement of the functions of the fifth nerve is generally co-existent, and oftentimes *herpes* of the face or lips, or mucous membrane of the nose or mouth. Division of the fifth nerve in animals produces ulceration, opacity, or inflammation of the cornea.

Treatment of these three varieties must be regulated by the general morbid condition of the system; so that the constitutional state of ill-health must be repaired, as the first essential step towards recovery. The syphilitic and scrofulous, or combined condition of those morbid states must be remedied as far as possible by the appropriate remedies already indicated under the description of these diseases. Regulation of diet, of wines, and of the use of water, are all-important, as already indicated (p. 112, chap. xiii.)

If the lips and nostrils are swollen and red, with a tendency to crusts round the nasal orifices, preparations of arsenic and iron will be found of great use. If the sclerotic is implicated, *bichloride of mercury* is indicated to the extent of one-sixteenth of a grain in half an ounce of water daily; and in children the *hydrarg. cum cretâ.* Dr. Mackenzie recommends *tartar emetic* as a sedative and alterative, both by itself in doses of one-twelfth of a grain, to one quarter of a grain thrice a day, and also along with *Peruvian bark* or *quinia.* He remarks that the combination is no doubt unchemical, but nevertheless he has derived more benefit from these two medicines given together, than from either of them singly. A dry and harsh state of the skin suggests *tartar emetic,* and diaphoretics generally, such as Dover's powder at bedtime.

Calomel with *opium,* to the extent of "touching the gums," is also of great service when much pain is present in paroxysms. It is especially necessary if iritis co-exists.

Turpentine in half drachm to one drachm doses, thrice a day, has also been found of service.

In syphilitic cases, so long as active inflammation seems to be going on, treatment must be directed towards the subjugation of the constitutional contamination. Frictions with mercurial ointment may be required. The *ungéntum hyrageri nitratis mitius,* to the extent of a portion "the size of a small pea" may be rubbed over the eye-brows at bedtime; and at the same time the internal use of the *iodide of potassium* is to be prescribed. Good nourishment is absolutely necessary. The formation of pus or lymph tends to be abundant in syphilitic cases.

With regard to local remedies, the first in importance is the use of atropia, if the tissues are unduly vascular. A few drops are to be applied to the conjunctiva of the lower lid with a camel's-hair brush, from three to six times daily; and, after all vascularity has subsided, it must still be applied three times a week for one or two months. Again, so long as the cornea appears vascular, the application of warm fomentations over the closed lids, every quarter of an hour during the day, and a lintseed-meal poultice at night should be applied, with a view to promote a flow of pus from the conjunctiva or of catarrhal mucus—a pathological process which seems to favour the dispersion of the corneitis. Such local means also favour the rapid development of blood-vessels in the cornea, where lymph tends to precede the formation of pus, and so tends to prevent perforation.

The insertion of a seton into the adjoining temple relieves intolerance of light, or *tincture of iodine* to the skin of the eyelids twice a week. The course of syphilitic lesions of the eye is very slow. It may extend over months; and then permanent impairment of vision may remain, especially from opacities of the cornea.

In *strumous corneitis*, leeches may be required to the temples if there be much pain or redness. Frequent fomentation of the eyelids with lint dipped in hot water; or olive oil, containing some *chloroform* or *morphia*, or both, may be applied to the skin round the eye.

Atropia should be applied to the conjunctiva once daily. If there is much purulent discharge, *alum lotion* should be used, or the lotion of *bichloride of mercury*, with *sal ammoniac;* and some *ungentum hydrargeri nitratis mitius* is to be applied to the eyelids at bed-time; to be washed off in the morning with warm water and alum lotion.

KERATITIS WITH SUPPURATION—*Syn.*, ONYX.

LATIN EQ., *Keratitis suppurans* — Idem valet, *Onyx;* FRENCH EQ., *Kératite suppuré;* GERMAN EQ., *Horn-hautenzündung mit Eiterung;* ITALIAN EQ., *Cheratitide con suppurazione.*

Definition.—*Inflammation with purulent collection between the lamellæ of the cornea, with cloudiness, disintegration, and breaking up of the cornea into fatty granular degeneration.*

Pathology.—The formation of pus in the cornea is usually associated with more or less of ciliary irritation. The pus collection may appear as a layer of yellow substance in the cornea, or reddish if mixed with blood. At first the collection of yellow matter is not fluid, but its transformation into pus usually begins after a few days, generally in the centre of the mass; but often in several points at the same time.

Sometimes the pus sinks down between the lamellæ of the cornea, pressing them apart—hence the name of *unguis* or *onyx,* from its resemblance to the *lunula* of the nail.

Corneal ulcers are a frequent result of this process. Perforation may also occur into the anterior chamber, giving rise to *hypopyon*—that is, to an accumulation of pus or of shreds of lymph at the most depending part of the anterior chamber; or, its inward perforation may give rise to iritis.

An *onyx* is known by its superior limit being convex, and by its remaining unchanged in form and situation whatever be the position of the patient's head, whereas *hypopyon* gravitates to one or other side, according to the direction in which the head is placed (MACKENZIE).

Onyx, originating in scrofulous phlectenulæ or after small-pox, may commence above the centre of the cornea, and diffuse itself irregularly over a large extent, infiltrating a considerable portion of the substance of the cornea.

The pus may be removed by absorption in the course of a few days or a few hours; but that is not usually the happy event.

Symptoms.—Dr. Bader distinguishes three varieties of suppurative keratitis:—

(*a.*) Cases in which the suppuration comes on rapidly—*i. e.,* in from one to three days, with great increase of vascularity and pain, with some chemosis, intolerance of light, and lachrymation. This form often follows operations or injuries, especially blows from stones, and may be accompanied by abrasion of the cornea. It may also occur in the course of *purulent ophthalmia* or *pustular corneitis.*

(*b.*) Cases in which the suppuration appears slowly, having been preceded by protracted corneitis, as in weak ill-fed persons suffering from syphilis.

(*c.*) Cases where there is no intolerance of light, or hardly any, with chemosis mostly serous, with moderate watery or purulent discharge, and with rapid suppuration. This form may be observed spontaneously, or after operations on very old or decrepit persons, and after severe illness or fevers.

The cause of the suppuration, the general health or ill-health of the patient, and the state of the deeper parts of the eye, all influence the rapidity of the progress and the extent of suppuration.

Treatment.—The object is to check suppuration, by warm or cold applications, according to the indications.

If suppuration is accompanied by a sensation of great local heat and much pain, the application to the eyelids of lint dipped in cold water is indicated.

The constitutional indications must be followed out in accordance with the history of the case.

SECTION IV.—DISEASES OF THE SCLEROTIC.

SCLEROTITIS.

LATIN EQ., *Sclerotitis;* FRENCH EQ., *Sclérotite;* GERMAN EQ., *Sclerotitis;* ITALIAN EQ., *Sclerotitide.*

Definition.—*Inflammation of the white or hard membrane of the eye.*

Pathology.—The white or hard membrane of the eye forms a tough and firm, very slightly expansive fibrous capsule, which everywhere closely envelopes the choroid and the ciliary body, and is organically connected with them. It is named the *sclerotic,* and it has a large opening for the passage of the optic nerve, with numerous small apertures for the blood-vessels and nerves of the *choroid.* It is composed of connective tissue, whose elements unite into broad bands, interwoven with yellow elastic fibres, which also unite it with the *choroid* (HENLE). Granular pigment is scattered through the tissue in clumps. At its anterior edge the connective tissue-filaments pass immediately into the cornea, becoming transformed into corneal elements. A thick patch of very fine veins courses round the periphery of the cornea, imbedded in the scleral tissue (the canal of SCHLEMM). On the one side they are connected with the veins of the ciliary muscle, and on the other with the vascular net-work of the sclerotic (LEBER). The *sclerotic* is thickish posteriorly, just at the optic nerve entrance, and within this portion lies the posterior vascular zone.

The outer surface of the *sclerotic* is enveloped by a stratum of loose connective tissue—more scanty, loose, and ragged at the middle and posterior part. Anteriorly it becomes transformed into sub-conjunctival tissue, and contains a fine, close, vascular net-work, composed mostly of ciliary vessels. It is called here the episcleral layer, and at the circumference of the cornea is greatly developed, and very vascular, so much so that in great hyperæmia it is protruded like a pad, and is then described as a vascular ring (VON CARION).

These salient points in the anatomy and connections of the sclerotic are of importance to be noted, as they play an important part in giving character to the morbid appearances seen in inflammations of the eye, with which they are concerned.

Inflammation of the sclerotic is characterized, in the first instance, by proliferation of its connective tissue corpuscles. They swell, and their granular-like contents become cells, which multiply more or less rapidly by division and endogenous growth. Thus they press more and more on the intercellular substance, and on section appear in the form of nests, the branches of whose individual corpuscles anastomose with the branches of other nests of cells. In acute and severe cases its texture may become cloudy by a molecular deposit, or by extensive separation (by degeneration) of granular fat, or infiltration of serous fluid, any or all of which conditions impair its cohesion.

Inflammation of the sclerotic is generally referable to the following circumstances:—

(a.) *Syphilis;* (b.) *rheumatism;* (c.) *the extension of the inflammatory process from other structures, such as the conjunctiva or ciliary processes.*

(*a.*) In the syphilitic form the inflammation is generally circumscribed and associated with inflammation of the conjunctiva and sub-conjunctival tissue.

This form of inflammation generally commences in the ciliary region, where it forms patches of a purple tint, covered with enlarged vessels. The textures appear swollen; and in from five to ten weeks the inflammation may subside, leaving the sclerotic slightly discoloured, semi-transparent, and thinner. Such circumscribed inflammations often appear successively in adjoining parts of the ciliary region of the sclerotic (BADER).

(*b.* and *c.*) *Rheumatic sclerotitis,* where the sclerotic is alone affected, is a rare disease (MACKENZIE, BADER). Generally the *conjunctiva* and *sclerotic* are each affected at the same time, or the *sclerotic* is directly affected after the *conjunctiva,* and both eyes are seldom affected at once.

Symptoms.—The vascular appearances are characteristic, from the anatomical distribution already described. Fasciculi of vessels of a bright red colour advance in radii towards the edge, and sometimes a little over the edge, of the cornea, surrounding the cornea pretty equally on all sides. They appear larger and more turgid than in iritis, and seem to rise more from the surface of the *sclerotic.* The *conjunctivitis* is less expressed, and is never sufficient to mark the radiated inflammation of the sclerotic.

Dimness of vision is constant, depending on haziness of the cornea, attended by slight contraction of the pupil and sluggishness in the movements of the iris. The pupil is seen to be less than that of the sound eye. The iris becomes slightly discoloured; if naturally blue, it tends to green, and the attending *iritis* may proceed even to effusion of coagulable lymph within the pupil. But the degree of iritis in rheumatic sclerotitis is rarely severe (on the authority of Dr. Mackenzie, who has rarely seen rheumatic sclerotitis—where sclerotic is alone affected—terminate in any form of suppuration or of ulceration; and both are very common in catarrhal ophthalmia with rheumatism).

The affected eye feels dry and hot in the early period of the disease, and there may be considerable *epiphera* afterwards.

The pain of *rheumatic sclerotitis* is characteristic. It is of a stinging kind, extending from the eyeball to the orbit and neighbouring parts of the head. Their temperature is increased, and the pain is augmented by warmth and relieved by perspiration. It may also affect the forehead, the cheek-bone, and the teeth, extending sometimes even to the lower jaw, the side of the nose, within its cavities, or the ear. It may be precisely confined to one-half the head. The superciliary ridge is its chief seat, next to that the temple and the cheek. When felt chiefly in the eyeball, it has the pulsatile character of phlegmon; otherwise it is particularly expressed round the orbit, consisting rather in an agonizing kind of feeling, which distresses and wearies out the patient. Evening exacerbations are constant from four, five, six, or eight P.M., and, continuing during the night, they become most severe about midnight, and do not abate till about five or six A.M.

It thus totally prevents sleep, causing great distress, and the patient never fails to dwell on these points.

A considerable amount of symptomatic fever attends the disease, increasing with the nocturnal paroxysms of pain.

Diagnosis lies between *catarrhal ophthalmia, rheumatic ophthalmia,* and *catarrho-rheumatic;* and Dr. Mackenzie gives the following particulars, to distinguish *catarrhal ophthalmia* from *rheumatic:*—

(1.) The *rheumatic* affection has its *seat* in the sclerotic, and frequently extends to the iris and retina—the *catarrhal* in the conjunctiva.

(2.) In *rheumatic sclerotitis* the redness is chiefly radiated or zonular, and seated under the conjunctiva, in the deep-seated conjunctival or episcleral net-work; spots of blood extravasations are never seen.

In *catarrhal conjunctiva* the redness is reticular, and the turgid vessels are obviously conjunctival, capable of movement, and spots of extravasated blood are of frequent occurrence in the conjunctiva.

(3.) In rheumatic attacks, the more the inflammation is limited to the sclerotic the less secretion will there be from the surface of the eye; but the more the conjunctiva is involved, the greater will be the flow of mucus.

(4.) The character of the pain in sclerotitis has been already given; so has that of *catarrhal* conjunctivitis (see page 421).

Causes of rheumatic sclerotitis are generally exposure to cold, associated with the constitutional state of *rheumatism.*

The cold is generally in the form of a draught or continued stream of cold air, sleeping with the head and face exposed to air currents, as in a railway carriage, especially after a hot walk to "catch the train."

It is more prevalent when the wind is cold and north-easterly. The combination of catarrhal *conjunctivitis* with *rheumatic sclerotitis* is one of the most common and one of the most severe and dangerous diseases of the eye.

Treatment.—Circumscribed inflammation, associated with syphilis, generally yields to *bichloride of mercury* ($\frac{1}{24}$th to $\frac{1}{16}$th of a grain, twice daily in water), combined with the local application of *atropia* (Bader). In rheumatic sclerotitis, Dr. Mackenzie advocates bleeding, both general and local. The night after general blood-letting (to the extent of fifteen to twenty ounces) he applies a dozen leeches round the eye, with the object of relief to supra-orbital pain. He has never failed to find the combination of *calomel* and *opium* of use in checking the circum-orbital pain and dissipating the other symptoms. A pill, composed of four grains of *calomel* with one grain of *opium*, is to be given every evening till the gums are tender, when calomel may be omitted, and ten grains of Dover's powder substituted for opium. *Ptyalism* must not be induced.

The hypodermic injection of morphia may relieve nocturnal pains; and chloroform liniment, with belladonna, may be applied to the skin of the forehead and temple.

A large blister to the nape of the neck is also of service. A searching purgative should commence the treatment. *Quinine* and *Fowler's solution of arsenic* are useful tonics during convalescence.

During the whole course of *rheumatic sclerotitis* the pupil of the affected eye ought to be kept under the constant influence of belladonna.

SECTION V.—DISEASES OF THE IRIS.

IRITIS.

Latin Eq., *Iritis;* French Eq., *Iritis;* German Eq., *Iritis;* Italian Eq., *Iritide.*

Definition.—*Inflammation of the Iris.*

Pathology.—The iris is a diaphragm or thin curtain, having a circular opening near its centre, called the pupil, which is capable of being enlarged or contracted by muscular action. This curtain or diaphragm is suspended between the cornea and the crystalline lens by its peripheral or ciliary border, which is continuous with the stroma of the ciliary muscle and the ciliary processes, the heads of which it covers anteriorly. It is inserted about $\frac{1}{14}$th of an inch behind the outer margin of the cornea. The free or pupillary margin of the curtain floats suspended in the aqueous chamber, and is bathed on both sides by the dilute serum of the aqueous humour. When the pupil is moderately dilated the anterior capsule of the lens extends beyond the plane of the iris, which thus shuts off the posterior from the anterior chamber. When the pupil is still more contracted, the iris rests by a broad zone upon the convexity of the anterior surface of the capsule of the crystalline lens, upon which it glides, so that it seems bulged forward like a miniature dome. It is thus by contracting and dilating that it regulates the quantity of light admitted to the retina.

It is composed of *contractile* and *elastic fibres*, *pigment*, *elastic laminæ*, *epithelium*, *blood-vessels*, and *nerves*. It is nourished in a great measure by two arteries completely distinct from those which belong to the other textures of the eye. These are the two *arteriæ ciliaries longæ*. It is frequently the seat of inflammation; and small as the iris is, the inflammation is often seen to be entirely confined to it, or to its surfaces anteriorly and posteriorly. Iritis may thus exist as independently of inflammation in other membranes of the eye, as *conjunctivitis*, *sclerotitis*, or *corneitis*.

From some of the arteries of the choroid, also, as well as from the two large arteries just mentioned, the iris is supplied by a quantity of red blood, large in proportion to its small size, and with a large supply of nerves. It is a texture greatly liable to inflammation, and with it there is always at the same time some inflammation of the *choroid*, *retina*, anterior hemisphere of the *capsule*, the *sclerotic*, and *conjunctiva*. But the iris is the focus or starting point of the inflammation, and the seat of the most striking morbid changes. At the pupillary or free margin of the iris inflammation generally appears to commence, whence it spreads to the rest of the iris, to the *capsule of the lens*, to the *choroid*, and the *retina*. The accompanying *sclerotic* and *conjunctival* inflammations are believed to be sympathetic or reflex. We can now see, by means of the ophthalmoscope, that the condition of the deeper parts of the eye is usually normal in iritis; but there is also this proof to show that the iris is often the only part which permanently suffers—namely, that when the natural pupil is closed up by the lymph of iritis, vision may still be restored by an artificial pupil—thus showing that the choroid and retina have not been impaired (MACKENZIE).

Hitherto the parts concerned in the diseases of the eye that have been described have been of the nature of *mucous* surfaces; but the iris is suspended in an aqueous chamber, which is really a *serous* cavity, lined by a smooth membrane, the source of the aqueous serum which the cavity contains. The pathology of this serous membrane is similar to that of serous membranes in other cavities of the body; but, as Sir Thomas Watson observes, "it is the only *serous* cavity into which we have the privilege of looking, and of noting what is going on, when the membrane that forms its boundary is inflamed." Hence iritis is one of the most interesting, and perhaps also one of the most instructive, of all diseases in the lessons of pathology and of therapeutics, which may be taught by carefully watching the progress of a case from day to day.

There are certain terms used in reference to the *mobility of the pupil*, its *contraction*, and its *dilatation*, which are necessary to be understood: and the following definitions of these terms are condensed from Dr. Bader's excellent account of the iris:—

(1.) The movements of the pupil are "*normal*," or "*the pupil acts well*," if it responds to the requirements of health, as regards contraction, dilatation, and accommodative movements.

(2.) In an eye of a person twenty years of age, in which the medium diameter of the pupil (dilated) is $\frac{1}{5}$th of an inch, the pupil may contract to $\frac{1}{12}$th of an inch, and may be dilated to $\frac{1}{3}$rd of an inch.

(3.) The distance of the margin of the pupil during medium dilatation from the apex of the cornea amounts to about $\frac{1}{7}$th of an inch. The centre of the pupil is situated a little inwards from the axis of the cornea, and is carried a little more inwards still during accommodation.

(4.) *Contraction* of the pupil is either *direct*—*i. e.*, the result of some kind of stimulus applied to the same eye, or it is *indirect* or *consensual*—*i. e.*, the result of some kind of stimulus applied to the fellow-eye.

(5.) The pupil becomes *contracted* under the following circumstances:—

a. When looking at *near* objects, or through a weak concave lens.

b. During the action of the muscles attached to the eyeball, more especially of the internal recti.

c. On the stimulus of light.

d. On direct irritation of the *fifth nerve*, or of its *ophthalmic branch*, or after division of the *Gasserian ganglion*.

e. On the local application of such agents as *aconitum napellus*, *Ruta graveolens*, *opium*, *Physostigma venosum*, or *Calabar bean*.

f. After escape of *aqueous humor* of the *vitreous substance* or of the *lens*.

(6.) The pupil becomes dilated under the following circumstances:—

a. When looking at distant objects.

b. On diminishing the stimulus of light.

c. On the application of such medical agents as *belladonna* supplies.

(7.) The size and mobility of the pupil in health, when tested by the same amount of light, is the standard by which its "*medium size*" is determined; and the terms—"*it is contracted*," "*it is dilated*," "*it acts in concert*" [with the other], "*it is sluggish*," "*it is fixed*," are conditions measured by the same standard.

For example, to say "*the pupil is dilated*," means that the pupil remains dilated, when a healthy pupil exposed to the same amount of light would assume a medium size, or become contracted.

To say that "*the pupil acts in concert*," or "*its central movements are undisturbed*," means, that while the mobility of the fellow-pupil is examined, its movements are similar to those of the pupil under examination.

To say that "*the movements of the pupil are sluggish*," means, that the rapidity of *contraction* or *dilatation*, or both, is slow.

To say that "*the pupil is fixed*," means, that it remains immovable on exposure to the stimulus of light.

(8.) In health, the pupil is circular; but it becomes irregular, if portions of the free margin of the iris advance into the area of the pupil; such irregularities are produced by adhesions, the results of inflammation, or as results of paralysis.

Although the changes which occur in *iritis* are generally described as similar to inflammation of serous membranes generally,—*i.e.*, changes of an *adhesive* kind, attended especially with effusion of coagulable lymph—yet the nature of the inflammation sometimes tends to the formation of *pus* or *serum.* Hence, besides *plastic* or *adhesive iritis,* there are also to be distinguished *suppurative* and *serous iritis.*

The causes and course of iritis have also given special names to different forms of the affection, such as *syphilitic, rheumatic,* and *traumatic iritis, acute, chronic,* and *recurrent iritis.* The College of Physicians have named the following varieties:—*Iritis* (simple), *traumatic iritis, rheumatic iritis, arthritic iritis, syphilitic iritis, scrofulous iritis,* and *gonorrhœal iritis.*

Morbid Anatomy.—The point of origin of inflammation of the iris, and the chief seat of tissue change by proliferation, are the stroma cells; but the connective tissue, intercellular substance, and muscular fibre cells and pigment of the tapetum, each and all take an active part in the process, which is similar to that which obtains in parts which have a basis of connective tissue. The minute textural cell-elements first swell somewhat, their contents becoming cloudy and turbid, and, excreting a molecular mass, may resolve themselves into a fatty degeneration. The fatty particles collect chiefly about the nuclei, which increase in size and become roughly granular, or altered in form. The connective tissue accompanying the blood-vessels is the first to undergo change. Their nuclei rapidly pass into a state of germination and separation. The intercellular substance becomes relaxed and swollen on account of dilatation of the blood-vessels, and serous or gelatinous infiltration is the result.

The new material in the tissue of the iris thus arranges itself in the course of its blood-vessels, or is scattered without order in its substance. Sometimes the morbid product is very scanty, and is indicated by more or less marked discoloration and swelling of the iris, to which the name of *serous iritis* has been given (VON CARION). It affects chiefly the covering prolonged over the front of the iris from the cornea. It generally occurs in the young or middle-aged, appears suddenly, and with more or less irregular dilatation of the pupil and increased tension of the eyeball, from the increased quantity of more or less turbid aqueous humour in the anterior chamber. Deep-seated changes are apt to take place in the *optic disc* and the other tissues of the eye, often followed by softening and shrinking of the eyeball, with complete loss of vision.

In by far the greater number of cases of iritis, however, the inflammatory products are adhesive, and of the nature of connective tissue, occurring as—

(*a.*) *Papillary excrescences,* scattered over the free pupillary margin in the proper struma of the iris. By means of this lymphy material the form and colour of the parts are changed; the size and figure of the pupil undergo alterations, or that aperture is completely closed up; and the motions of the iris are limited or entirely prevented. The lymph may also perforate the anterior or posterior boundary layer of the iris; posteriorly, it may come in contact with the anterior capsule of the lens, to which it may quickly adhere (*posterior synechia*); anteriorly, the swollen and inflamed iris or the lymph may come in contact with and adhere to the cornea (*anterior synechia*). Once union is thus established, the lymphy connections may be drawn out to threads of connection between the parts. In exceptional cases the new material may be of considerable thickness, completely closing the pupil like a plug.

(*b.*) *Granulations* are most frequently seen on the anterior surface of the iris, and especially on prolapsed portions of the iris, where the cornea is perforated by ulceration. If the cornea is entire, they are rarely elevated above the surface; but, nevertheless, they may cover the whole iris, and even the pupil.

The tissue of the iris generally becomes loosened, in consequence of luxuriant development of connective tissue, and becomes somewhat spongy (*iritis parenchymatosa*).

(*c.*) *Gummy tumors* are nodular collections of inflammatory products; varying greatly in size, springing from the deeper tissue of the struma of the iris, and, extending outwards from its anterior surface, may form large tumors. Virchow long ago recognized their nature as similar to the true syphilitic gummy tumors of other parts (VON CARION).

In a few cases of iritis, the products of inflammation have from the commencement the character of pus. Pus-cells have been found in the tissue of the iris; and exceptionally small collections—miniature abscesses—have been found; and, under certain circumstances, the iris, as a whole, has been changed into a mass of pus, being thus entirely destroyed by purulent infiltration. As a rule, however, pus appears in iritis as a secretion on its surface, which gradually collects on the floor of the aqueous humour, and forms a *hypopyon,* consisting entirely of fluid pus, changing its position with every movement of the eye (VON CARION).

The characteristic lesions peculiar to specific forms of iritis may be generally stated to be—(*a.*) for *syphilitic iritis,* the separate nodules of lymph; (*b.*) for *sympathetic* or *reflex iritis,* the unusual enlargement of the vessels of the iris; (*c.*) for *serous iritis,* the enlargement of the pupil, combined with increased tension and increased quantity of aqueous humour (BADER).

A partly *plastic* and partly *suppurative iritis,* accompanied by inflammation of other parts of the eye, may occur during, or soon after, an attack of *measles, scarlet fever, variola, enteric* or *rheumatic fever,* or during pregnancy, or while nursing.

Iritis is almost always complicated with ciliary and sclerotic inflammation to some extent; and most frequently with inflammation of the cornea and capsulitis. Iritis occurs at every time of life—during fœtal life (HIMLY, PAGENSTECHER) and in the new-born infant; but during middle life, from twenty-one to fifty years, furnishes the greatest number of cases.

Symptoms.—There are certain combinations of symptoms characteristic of iritis, from whatever cause it proceeds. These are a peculiar vascularity, a change in colour and general appearance of the iris, irregularity and immobility of the pupil, a visible and varying amount of lymph in, upon, and round the iris. These are the *objective* signs of *iritis*, described in detail under the following heads:—

(1.) *Zonular sclerotitis*, denoted by the fine hair-like vessels running in radii towards the edge of the cornea, so that the cornea becomes surrounded by a zone of fine, straight, converging pink lines. This extremely fine vascular net-work in the anterior episcleral zone is one of the most constant signs of iritis—the first premonition of inflammation, which continues as long as the inflammation of the iris continues, and disappears when that ceases. The infected tissue is at the same time generally infiltrated with serum; and the conjunctiva participating, a *vascular ring* may form round the cornea. These hair-like converging lines stop abruptly at the edge, or juſt before they reach the edge of the cornea, where they dip through the scleroitic to reach the iris. Hyperæmia of the iris itself is not easily seen, except in the later stages of chronic iritis, when the iris tissue is far advanced in atrophy.

(2.) *Peculiar discoloration of the iris*, depending upon its change of structure from proliferation of tissue, so that its surface, so beautifully marked, regular, fibrous-like arrangement, and velvet-like aspect, has a dull, glistening appearance. Its peculiar brilliancy of surface is spoiled. It becomes dull and tarnished, and its colour, if naturally blue or grey, becomes of a dirty slate colour, or yellowish green; if of dark colour, it changes to an ochre, a cinnamon colour, or brownish red. The inflamed eye must be compared with that which is sound.

(3.) *Great sluggishness or complete immobility of the pupil*, a necessary result of the proliferation of the tissue of the iris, limiting the action of the contractile muscular elements in its stroma.

In order to test the reaction of the iris, the patient should be so placed that moderately-strong ordinary daylight falls obliquely, from one side only, upon the eye. The unaffected eye should then be closed, not only with the hand, but also with a folded cloth, so that every trace of light be absolutely excluded from it. The examiner now places himself in such a position before the patient that, while he throws a very dark shadow on the uncovered eye with his hand, he keeps the pupil in plain sight. Now, fixing his eye on the edge of the pupil, and by removal of the hand, a bright light is thrown upon the eye, and then the eye is again shaded; and so on. One or two changes of light and shade will, as a rule, enable us to conclude as to the reaction of the iris; and the slightest puckering of the pupillary margin will be detected.

(4.) Contraction and irregularity of the pupil, caused by increase in its thickness and enlargement of the width of the iris, the result of intumescence from inflammatory proliferation and serous infiltration of tissue.

(5.) A varying amount of "plastic" material in, upon, and round the the iris, so that effusion of coagulable lymph into the pupil and posterior chamber, and occasionally into the anterior.

(6.) Adhesions of the iris, especially of its pupillary margin to the capsule of the lens, and in some rare cases, to the cornea.

(7.) Impairment of vision is often the sole symptom for which advice is sought. It may arise from opacity of the lens or its capsule; dimness of sight, from changes in the aqueous humour; loss of power of accommodation, and inability to contract the pupil; and from changes in the iris and ciliary muscle. The impairment varies in degree—sometimes only a slight "mist" may appear to intervene between the object and the eye; and the cornea may be dim, like roughened glass, disturbing its transparency.

(8.) Pain in the eye, the most inconstant symptom of all—is often absent, or so slight that it scarcely excites the attention of the patient. In other cases it appears early, gradually increases, and finally becomes very severe.

(9.) The constitutional symptoms are alike uncertain. Sometimes there is a good deal of fever and headache, with white tongue, and broken sleep.

Causes.—These are various; but the best ascertained are,—

(1.) Mechanical, chemical, and physical injuries, such as sudden transition from heat to cold, exposure to cold draughts, disease of the eyes in straining after distinct vision of minute objects, especially by artificial light.

(2.) Secondary iritis, the result of extension by continuity of inflammation from parts in immediate anatomical or functional connection with the iris, as *choroiditis* or *keratitis.*

(3.) Certain constitutional affections, especially *syphilis,* to which a large percentage of cases of iritis is due.

Syphilitic iritis in children during the first month of life requires special attention, since it is apt to be overlooked, and to run a very insidious course, with no very striking symptoms. The mothers of such children have generally acquired syphilis at a time not far removed from the period of parturition; and well-nourished children are more disposed to such iritis than others (HUTCHINSON).

Scrofula, often associated with *scrofulous keratitis,* as a secondary disease; *gout,* giving rise to so-called *arthritic iritis,* and generally combined with ill-health, resulting from abuse of beer, alcohol, and tobacco (MACKENZIE, VON CARION).

Prognosis.—Simple iritis in an otherwise healthy person ought to subside in about four weeks; but the changes which may result to the iris depend upon the intensity, kind, and duration of the attack, and also upon the number of attacks. If its progress be not checked, the inflammation creeps from the pupillary margin to the ciliary attachments and ciliary body, to the choroid coat and to the retina, when pain and fever increase. The innermost delicate textures of the eye are spoiled for ever, and total blindness is the usual result.

The proportion of cures is large; but sometimes sequelæ follow, which render the preservation of vision doubtful. Adhesions to cornea (rare), or capsule of lens (common), contraction of pupil, false cataract, atrophy of the eye, and closure of pupil by false membrane may be mentioned.

Treatment.—In every case, and at any stage of iritis, dilatation of the pupil must be brought about and maintained by the use of *mydriatics,* or agents which enlarge the pupil, chief of which is *belladonna* and its alkaloid, *atropia,* so as to prevent irregularity of the pupil and adhesions, anteriorly or posteriorly (*synechiæ*); to secure rest by preventing contraction of the iris and ciliary muscle; and to alter the tension of the eye, and influence the circulation in the *choroid*. In slight cases, *atropia* should be applied from three to ten times daily; and, if there is severe pain, every five minutes for some hours in the evening; and these frequent applications are to be continued for from two to five days, or till pain and vascularity have lessened, the patient being kept in a darkened room.

After all vascularity has subsided, the *atropia* must still be used at least twice daily, and so continued for from two to three weeks, with the view of providing against any relapse (BADER).

A few drops of a solution of from one grain of *sulphate of atropia* to the ounce of distilled water, dropped into the eye, is the most efficient mydriatic. It is most convenient to prepare a solution of two to three grains of *sulphate of atropia* to half an ounce of distilled water, and everting the lower lid, to drop it into the conjunctival sac by means of an obliquely cut quill or a camel's-hair brush. The use of *atropia* cannot be entrusted to the patient; and if symptoms of *belladonna* poisoning should supervene, it must either be discontinued or used in a less strong solution (VON CARION).

The pupil may continue to act sluggishly long after the use of *atropia* has been discontinued—a condition which may be remedied by the use of *myotics*, or agents which contract the pupil, such as *Calabar bean*, the alcoholic extract of which, diluted with *glycerine*, in the proportion of one of extract to thirty or fifty parts of glycerine, and applied with a camel's-hair pencil, is usually employed.

One *may see* the good effects of thus artificially dilating the pupil. On carefully examining an eye where strings of adhesion are visible, connecting the edge of the iris with the capsule of the lens, these adhesions may be *seen* to stretch, and then to break under the dilating influence of *atropia*.

The indications for treatment are—(1.) To subdue the congestion; (2.) To prevent or limit the formation of inflammatory material (lymph, pus, or serum), or to promote their absorption; (3.) To preserve the integrity of the pupil, and to dilate it as soon as possible, believing that a fixedly *dilated* pupil is better than one contracted and fixed; (4.) To relieve pain.

Of the remedies to accomplish those ends, the chief are *blood-letting*, *mercury*, and *belladonna*; and of these Sir Thomas Watson's experience leads him to say that, "If I were restricted to the use of one of these means, I should choose *mercury*; if to two, *mercury* and *belladonna*; but the combined employment of the three has the most powerful effect in curing the disease," (Lect. xix.)

Although belladonna may speedily expand the pupil, it acts much more powerfully, rapidly, and efficiently after general blood-letting (MACKENZIE).

The pupil of a healthy eye becomes dilated to its maximum in from fifteen to thirty minutes after applying to the conjunctiva a solution of the *sulphate of atropia* (gr. one-fourth to one ounce of distilled water). Soon afterwards the ciliary muscle and the power of accommodation become paralyzed. Within three hours the ophthalmoscope will show its effect upon the *retina* and *choroid*, causing dilatation of the blood-vessels of these structures.

Solutions of atropia of two degrees of strength are in daily use, namely:

(*a*.) ℞. *Atropiæ Sulphatis*, gr. $\frac{1}{4}$.; *Aquæ Distillatæ*, ℥i.; solve—for use in inflammatory changes (*keratitis, iritis*). (*b*.) ℞. *Atropiæ Sulphatis*, gr. ii.; *Aquæ Distillatæ*, ℥i.; solve—for use to bring on rapid paralysis of the power of accommodation.

Symptoms of *atropia poisoning* are increased rapidity of pulse (110 instead of 70), dryness of the throat, restlessness, followed by sleep with much dreaming; general excitement, hallucinations, inability to swallow. If the graver symptoms appear, the best antidote is the subcutaneous injection of acetate of morphia (see p. 408).

Blood-letting must in no case be neglected; but the necessity for it and the amount can only be determined by the strength and constitution of the individual patient (MACKENZIE, WATSON).

Bleeding by leeches (two to six), applied at bedtime to the corresponding temporal region, if the pain is severe and not relieved by *atropia*, is considered sufficient by Dr. Bader. Afterwards the bleeding is to be encouraged for one or two hours, and the eyelids kept closed for at least twenty-four hours after the application of the leeches.

Cathartics and *diuretics*, combined with a spare and cool diet, confinement within doors, rest of the whole body, and exclusion of light from the eyes, are powerful aids to cure, and ought to be rigidly enforced. *Antimony* is of use in two ways. It moderates the force of the circulation, and renders the system more susceptible to the influence of *mercury*, given so as to affect the system—the most important remedy we possess for the cure of iritis. The most useful form for its administration is that in which it is combined with *opium*, in small doses, frequently repeated. The object is to affect the gums as speedily as possible—in two to five days—without producing sudden and severe ptyalism. Soreness of the gums and the characteristic fætor of the breath are sufficient signs that the specific influence of the remedy upon the system is in force; and the mouth should be made decidedly sore as quickly as possible. *Two, three*, or *four* grains of *calomel*, combined with *one-fourth, one-third*, or *one-half* of a grain of *opium* may be given every *four, six*, or *eight* hours. It may be given in small doses even still more frequently—one grain of *calomel*, with *one-tenth* or *one-eighth* of a grain of *opium*, every hour; and if the gums do not swell in the course of thirty-six to forty-eight hours, inunction by mercurial ointment may be commenced (SIR THOMAS WATSON). Once the mouth has become decidedly sore, the influence of the remedy must be maintained till the sight is restored, and that may require a month, or longer.

The evidence of the good effects of the specific action of mercury upon the iritis consists in—(1.) The fading of the red zone round the cornea; (2.) The lessening of the drops of lymph which daily seem to melt away from the surface of the iris; (3.) The iris recovering its proper tint, its internal texture becomes cleared and freed from deposit; (4.)

Disappearance of irregularity of the pupil and puckering of the iris, the pupil becoming a perfect circle.

Opiates are, as a rule, always required in iritis, on account of the severity of the nocturnal circum-orbital pains, and on account of the general distress which the patient experiences in the eye. Local inunction of mercurial ointment (*ten* grains), combined with finely-powdered opium (*two* grains), well rubbed into the temple a little before the nocturnal pain is accustomed to recur, is productive of great comfort and relief to the patient (SIR THOMAS WATSON). If the employment of mercury requires for any cause to be suspended, *iodide of potassium* is most useful for maintaining its influence. It is especially useful in the syphilitic form of iritis; and *sulphate of quinia* is specially useful in the scrofulous form.

Tepid water should be used to bathe the eyelids morning and evening, or as often as may be agreeable; and alum lotion may be used if purulent discharge from the conjunctiva exists; although, as a rule, stimulating remedies to the conjunctiva are useless or hazardous in iritis, and are not to be ventured upon in the acute stage (MACKENZIE, BADER).

RHEUMATIC IRITIS.

LATIN EQ., *Iritis rheumatica*; FRENCH EQ., *Iritis rheumatismal;* GERMAN EQ., *Rheumatische iritis;* ITALIAN EQ., *Iritide reumatica.*

Definition.—*Inflammation of the iris, occurring in persons of rheumatic habit, although they may not have suffered from rheumatism in any other part of the body. It is liable to return again and again, and to be combined with catarrho-rheumatic ophthalmia, affecting especially the sclerotic.*

Pathology.—Acknowledging a constitutional connection, rheumatic iritis generally advances slowly and insidiously, and appears often to be immediately excited by the same circumstances which excite rheumatic inflammation in other structures of the same kind, particularly fibrous textures.

Metastasis, as a cause, is now being less and less believed in. Exposure to atmospheric changes, suppressed perspiration, over use of the eyes, are sufficient to establish *iritis* in connection with rheumatism. One or more attacks of the disease may occur every year; and although the tendency of a first or single attack of *rheumatic* iritis is to get well without much or permanent damage to vision, yet the repetitions of the lymph effusions of each successive attack under which adhesions readily form, leaves the eye in a worse condition than before, after each repetition of the disease, till at length vision is completely destroyed. The pupil becomes more and more contracted, and may be at last closed by a plug of lymph.

Symptoms.—Dimness of sight, so that the letters of a book appear pale, is often the earliest symptom. The eye is soon fatigued, and by and by everything is seen as through a thick fog.

The objective changes in the iris and connected parts are similar to those already described; but there are some local appearances, said to be more or less characteristic of the variety of iritis. The redness, for example, accompanying the changes in the iris is by no means considerable; and is at first confined to the sclerotic coat, in which a number of very minute rose-red vessels are seen running towards the cornea. As the redness increases it is seen to arise partly from vessels developed in the conjunctiva; and, although the vascularity is greatest towards the cornea, and fades away towards the folds of the conjunctiva, yet the zone of red vessels does not approach so close to the cornea as in other forms of iritis; a comparatively white ring is left between the cornea and margin of the zone. The colour of the vascular zone, also, is not so bright as in other forms of iritis. It is somewhat livid or slightly purple; and the larger conjunctival vessels at the back part of the eye are tortuous and varicose.

The pain in the eye is in many cases severe and pulsative, and increased on motion of the eyeball. There is pain also beneath the eyebrow, and circumorbital nocturnal pain similar to that met with in rheumatic sclerotitis.

As the disease advances unchecked, the pupil becomes irregular, or triangular, and of a grey appearance, produced by a delicate film of coagulable lymph, which may be brought into view by a magnifying glass of short focus, or by concentrating the light upon the pupil through a double convex lens. Into this film processes, or dentations from the irregular, pupillary margin of the iris, are seen to shoot, and at these points adhesion between the iris and the capsule of the lens is apt to be established, by reason of which vision, all along indistinct, can only be brought to bear on one side or part of an object. These tags of adhesion give the pupil a peculiar appearance when dilated by *belladonna*. Dr. Mackenzie mentions a case in which five tags of adhesion existing, gave the pupil the shape of an oak-leaf when dilated; and sometimes several or all the tags of adhesion may be seen to break across under the influence of *belladonna*, leaving minute white spots on the capsule at the points of adhesion. These adhesions are said to be *whiter* in *rheumatic* than in other varieties of *iritis*.

As the *retina* becomes involved, and the pupil obscured by lymph, the morbid sensibility to light diminishes, and the powers of vision become more and more limited, until little more than perception of light remains.

Inflammation in time subsides, as it does in other rheumatic affections of tissue, even though no remedies are employed; but in such cases vision is generally lost (MACKENZIE).

The constitutional symptoms are generally those associated with rheumatism (which see vol. i., p. 801), and there is especially thirst, whiteness of tongue, and quickened pulse, confined bowels, and a disposition to nausea.

Treatment.—Dr. Mackenzie considers that repeated venesection is almost always necessary, followed by a liberal application of leeches round the eye and on the temples; but the degree of inflammatory fever present must be the guide as to the extent and kind of bleeding.

Sir Thomas Watson considers moderate topical bleedings, counter-irritation, and measures conducive to improve the general health, as the treatment which answers best in *rheumatic ophthalmia*. *Colchicum, bark,* *sarsaparilla*, *iodide of potassium*, are remedies indicated by the *rheumatic* condition, but are not always to be depended upon as useful.

The most marked abatement of the local symptoms takes place as soon as the mouth is made sore by the use of *mercury*. Two grains of *calomel*, with one-third of a grain of *opium*, are to be given, in acute cases, every six hours; and less frequently in chronic cases, taking care not to induce ptyalism, or to make the mouth too sore.

Exposure to cold must be specially guarded against, even in passing from one room to another. Confinement within doors is necessary; and, if the case is severe, confinement to bed. If the patient is unprovided with proper clothing and shelter, it becomes a question, says Dr. Mackenzie, whether or not mercury should be given. "We are almost certain, by its omission, to ruin the eye; and, by its exhibition, seriously to endanger the general health." The patient must relinquish animal food and fermented liquors; and his disordered digestive organs must be set right.

Whether calomel be given or not, powerful opiates are required at night, by inunction of mercurial ointment with opium; or, by fomentation with hot cloths, over which laudanum is sprinkled; or, by hypodermic injection of morphia.

The bowels must be kept moderately opened every morning. Small doses of *nitre* and *cream of tartar* are useful as a diuretic every two hours.

Atropia must be freely used; but it will not have any apparent effect till the inflammation is subdued by the *bleeding* and *calomel*. When the gums become touched, the pupil may be seen suddenly to expand in favourable cases.

ARTHRITIC IRITIS.

LATIN EQ., *Iritis arthritica;* FRENCH EQ., *Iritis arthritique;* GERMAN EQ., *Gichtische iritis;* ITALIAN EQ., *Iritide artritica.*

Definition.—*Inflammation of the iris occurring in persons of gouty habit, in asthenic states of the system, after repeated attacks of gout have produced depression of body and mind, with dyspepsia, flatulence, languor, and irregularity in the excretions.*

Pathology.—Gout is not now believed to be the peculiar inheritance of the opulent and luxurious (see "Gout," vol. i., p. 833). It is not uncommon even amongst the poorest of the people. Dr. Mackenzie seldom met with *arthritic iritis* in regular gouty constitutions, and rarely in the first or plethoric period of gout, while the patients still retained strong powers of digestion, and, having the means and the inclination, regale themselves with large supplies of food and drink. The subjects of this form of iritis have generally been above *fifty* years of age, of sallow complexion—in many instances, but not always so, tobacco smokers and whisky drinkers, who have often suffered from rheumatic affection, much headache, bad gums and teeth, acidity, flatulence, and lowness of spirits. The textures of the iris in such cases is already presumed to be defective in its sanguineous nutrition, the general health having been impaired and broken by intemperate habits. The *iritis* thus occurs in texture already degenerated, and most unfavourable for cure.

The subjects of the disease generally suffer from a plethoric state of the abdominal viscera, the immediate result of deteriorated digestion, and dependent on stimulants for appetite and disposal of aliment.

Symptoms.—The general objective symptoms are similar to those already described; but they are associated with such peculiarities and modifications as to render the disease characteristic and readily diagnosed—

(1.) The conjunctiva, as well as the sclerotic, is loaded with large purple vessels and varicosities of the vessels emerging from the *recti* muscles.

(2.) The sclerotic becomes of a dingy, greyish, violet colour.

(3.) There is considerable secretion from the eyelids, which leads to frequent opening and shutting of the eyelids, and a thick, white, frothy matter, of a sabaceous nature, rests upon the edges and angles of the eye.

(4.) A narrow ring, of a bluish-white colour, exists at the edge of the cornea, or only at its temporal and nasal sides. It is formed by the edge of the sclerotic, which naturally overlaps the cornea. It is not to be confounded with the *arcus senilis,* and it may also be seen in *syphilitic* and *rheumatic iritis* in patients far advanced in life.

Prognosis is more unfavourable than in any other species of iritis; and a first attack may continue for many months, subject to frequent removals. A severe attack once a year, or every two or three years, ends at last in blindness.

Treatment is best commenced by the action of such searching purgatives as *calomel,* in a five grain dose, combined with *one drachm of compound jalap powder.*

The emission of blood and the use of mercury, to affect the system, are not well borne in such cases. The disordered digestive secretions must be set right by such remedies as five grains of Plummer's pills, three or four times a week, followed by a glass of *Pullna water,* or one ounce of the *mixture of senna* (B. Ph.) as an aperient, the following morning, which ought to be continued for several weeks, along with other suitable remedies, so as to change the vitiated habits of the digestive organs.

Alkaline remedies are very useful; and subsequently preparations of *iron,* especially the *carbonate,* Dr. Mackenzie has found of service, especially after regulation of the bowels.

GONORRHŒAL IRITIS.

LATIN EQ., *Iritis gonorrhaïca;* FRENCH EQ., *Iritis blennonhagique;* GERMAN EQ., *Gonorrhoische iritis;* ITALIAN EQ., *Iritide gonorroica.*

Definition.—*Inflammation of the iris, preceded by gonorrhœa, and sometimes with effusion into the joints, and accompanied by excessive pain, intolerance of light, with profuse flow of tears and dusky sclerotic redness.*

Pathology.—There can be no doubt that gonorrhœa in some cases affects the system to this extent, that the textures implicated in the lesions of *rheumatism* are the seat of lesion also after gonorrhœa. Hence, there is *gonorrhœal rheumatism* affecting the larger joints, especially the knees, and the feet and ankles, attended with copious effusion, sometimes into the sheaths of tendons and synovial cavities of the joints, with swelling, pain, and fever, so that the symptoms are severe and the cure tedious (see "Gonorrhœal Rheumatism," vol. i., p. 827).

Gonorrhœal iritis seems but one other form of the same pathological process, which may or may not be preceded by synovitis.

Those who suffer from gonorrhœal rheumatism, either in the form of synovitis or iritis, are generally young men of scrofulous constitution, who live hard, and are careless of exposure to cold; so that each time a gonorrhœa is caught he is liable to an attack of synovitis or iritis, or suffers first from one and afterwards from another.

In some cases no new gonorrhœa occurs; but a second or third attack of inflammation affects the joints or eyes. Generally, one eye only suffers; and the same eye may suffer repeatedly (MACKENZIE). When iritis occurs, the patient generally has at the same time a gleet, as is the case in *gonorrhœal rheumatism;* and the iritis may thus alternate with *gonorrhœa* and *synovitis,* so that when one is in the ascendant, the others are in abeyance. In many instances the patients have their existence rendered miserable by alternate attacks of the three in succession, so that at last they are left in a state of extreme debility, their sight impaired, and their joints incapable of motion (MACKENZIE).

Symptoms.—The inflammation of the eye is generally very severe. It commences with redness of the conjunctiva and the sclerotic, with haziness of the cornea, which is characteristic. The inflammation speedily affects the *anterior* surface of the iris, which loses its natural colour—the inflammation being of the *serous* type.

The pupil contracts, and vision becomes dim. Effusion of coagulable lymph now takes place so profusely that it speedily fills the pupil, and may be so excessive as to fall down into the anterior chamber in curd-like masses, like hypopyon; and may almost fill the anterior chamber to such an extent that no other variety of *iritis* presents this symptom in the same degree (MACKENZIE).

The anterior surface of the iris, in some cases, is covered with lymph, as if coated with white paint.

There is generally violent pain in and round the eye, with watery secretion from it (epiphora) and intolerance of light; chemosis and conjunctival œdema may be considerable, but no purulent discharge exists. If left to itself, the pupil remains contracted, and adheres to an opaque capsule.

Prognosis.—The disease is one of the most severe forms of *iritis* while it lasts, and is more rapid in its progress than any other species of *iritis.* On the other hand, it yields more promptly to treatment than any other, and affords examples of perfect recovery, even when the anterior chambers are filled with lymph. In no other variety of iritis is recovery so striking and complete (MACKENZIE).

Treatment is by repeated and copious venesection, and with leeches round the eye; *calomel* and *opium,* in frequently repeated doses, so as to affect the system as rapidly as possible; and the continuous use of *belladonna.* The bowels should be acted on with searching purgatives at the same time.

SECTION VI.—DISEASES OF THE CHOROID AND RETINA.

CHOROIDITIS.

LATIN EQ., *Choroiditis;* FRENCH EQ., *Choroïdite;* GERMAN EQ., *Choroiditis;* ITALIAN EQ., *Coroiditide.*

Definition.—*Inflammation of the choroid, and generally only part of an inflammatory process extending over a great portion or the whole of the eye.*

Pathology.—The *choroid* forms the posterior segment of the middle tunic of the eyeball, reaching as far forwards as the cornea. Composed chiefly of blood-vessels, and possessing little sensibility, it is much less liable to inflame than the *iris,* which is highly vascular, endowed with great nervous irritability, and which has great proneness to inflammation. In the *choroid* and *iris* (which together constitute the middle tunic of the eyeball,—the white ring of the ciliary muscle connecting them both), almost the whole of the red blood of the eyeball is concentrated. The choroid is chiefly supplied with blood by short posterior ciliary arteries (about twenty in number), which pass through the sclerotic near the optic disc in the region of the *yellow spot.*

The *choroid* membrane is interposed between the *sclerotic* and the *retina,* and reaches forwards to the *ciliary* ligament, or nearly to the *cornea,* where it ends by a series of plaits or folds, named the ciliary processes (about eighty-five in number), disposed in a circle, projecting inwards at the back of the circumferential portion of the iris.

The *choroid* is thickest at the hinder part, where the *optic nerve* is transmitted through a circular opening (*foramen opticum choroidea*). Its rough outer surface is connected to the sclerotic by loose connective tissue (*lamina fusca*) and by vessels and nerves. The inner surface is smooth, and lined by a continuous layer of pigmentary cells.

In the *outer coat* of the choroid the larger branches of the blood-vessels are situated, in the intervals of which are lodged the stellate pigment-cells, with very fine offsets, which intercommunicate so as to form a network or stroma, and which, towards the inner surface of the choroid, passes gradually into a web without pigment, resembling elastic tissue.

The innermost portion of the *choroid membrane* is mainly composed of the fine capillary ramifications of the choroidal vessels. They are more delicate and smaller than in any other texture of the body, and are finer at the back part than at the front part of the eyeball. This fine network approaches as far forwards as to within one-eighth of an inch from the cornea, or opposite to the ending of the expansion of the optic nerve, where its meshes become larger, and join those of the ciliary processes.

The *pigmentary layer* forms a thin dark lining of epithelium to the whole of the inner surface of the *choroid membrane* and the *iris,* consisting of only a single layer of flat six-sided cells, applied edge to edge, like mosaic work. Each cell contains a nucleus and more or less dense molecular contents. On the ciliary process and the iris the pigment is several layers deep, and the cells smaller and rounder. They differ from true epithelium in this—that the same cells seem to exist during the whole of life (Quain's "*Anatomy*," by Sharpey, Thomson, and Cleland).

Ophthalmoscopic Appearances.—The inner surface of the sclerotic reflects the light through the choroid and retina, like the silvering to the glass of a mirror; and the greater the quantity of light thus reflected, the better will the details of the choroid be recognized. The more pigment the less light. The pupil should be well dilated with *atropia*, when the whole of the choroid, from the *optic disc* to near the *ora serrata*, can be seen, and in *albinos* the tips of the ciliary processes (Bader). The points to attend to are—(1.) *The choroidal aperture—i. e.*, to the part of the *choroid surrounding the optic disc.* (2.) *The region of the yellow spot,* which which is readily seen by directing the patient to look direct at the sight-hole of the ophthalmoscope. (3.) *To the equatorial region of the eye,* which is best explored by looking slantingly into the eye. The *individual* capillaries composing the choroid membrane cannot be seen in the healthy eye, being not more than the $\frac{1}{1400}$th part of an inch in diameter; but when filled with blood, they give it the characteristic pale or brilliant red colour one of the most characteristic features of the interior of the fundus of the eye. The colour of the iris may suggest the tint of the choroid,—a *blue iris* being generally associated with a brilliant red choroid, and a *black iris* with a choroid of almost neutral tint. The brilliancy of the general red hue depends upon the quantity and degree of tinting of the pigment and amount of blood circulating in the choroid. Groups of hexagonal cells may be seen as small dots in all eyes in the equatorial region of the eyeball; and in fair eyes, especially of children, on most parts of the choroid.

The particular tint of the fundus of the eye thus mainly depends on the relative proportions of blood and pigment present in the *choroid.* A small amount of pigment with a natural fulness of choroidal vessels gives the fundus a bright, clear, red colour, slightly tinged with orange, the larger choroidal vessels being distinctly visible; a little more choroidal pigment imparts a more decided orange or brownish red (Hulke).

Morbid Anatomy.—*Choroiditis* is fortunately not a frequent disease, but it is a very severe one, and is generally a part only of an inflammatory process extending over a great portion of the eyeball and its contents—notably affections of the *vitreous humor* and the *retina,* the *iris, ciliary body, and lens.* The older ophthalmologists have generally, therefore, described *choroiditis* as "*internal ophthalmia*" (*anterior, posterior,* or *general*), *acute or chronic.*

Proliferation of tissue in the inflamed choroid is generally not great, and evidence of its existence can only be shown by the microscope. The sclerotic in the eyes of young persons may take an active part in the inflammation.

Symptoms.—The following are the forms of *choroiditis,* which are to be distinguished:—(1.) *Simple* or *plastic;* (2.) *Suppurative;* (3.) *Syphilitic;* (4.) *Serous;* (5.) *Traumatic, sympathetic,* or *reflex.*

The general symptoms consist mainly of the sudden occurrence of severe throbbing and darting pains in the eyeball and corresponding half of the head, occurring in paroxysms, and much increased during the night. The eye feels stiff, with a sense of fulness and distension. and so excessively tender that the patient cannot touch it or allow it to be touched. Frequent flashes of vivid, reddish, or orange-coloured light is complained of even in the dark, or a luminous spot is seen in the axis of vision, increased by everything which quickens the circulation. The eye is suffused with tears, and highly intolerant of exposure. Dr. Mackenzie has known an attack of acute choroiditis to come on suddenly in the night, and in the course of a few hours totally to abolish the sensibility of the retina. In such a case vision is rarely recovered from, although redness and pain may be subdued. In other cases, vision becomes dull and misty, and the mistiness rapidly increasing, the eye in a few days becomes completely blind (*aumorosis*).

The subjects of choroiditis are generally past middle life, and more often females than males.

The disease may be confined to the *posterior* part of the choroid. Considerable constitutional disturbance attends the disease. It is expressed in the form of restlessness, sleeplessness, flushing of the face, giddiness, and nausea. The tongue is generally foul, the mouth parched, and there is much thirst. The pulse is quick and hard; and so great has been the pain, and long continued the want of sleep, that Dr. Mackenzie has known such constitutional disturbance to lead to a fatal termination.

Certain symptoms are common to all of these forms, especially loss of transparency of the choroid and atrophy; and as the external and objective symptoms may be altogether absent, *choroiditis* can only with certainty be recognized by the ophthalmoscope.

The changes in the eye, to be recognized by the ophthalmoscope in *choroiditis*, are—(1.) Hyperæmia; (2.) Inflammatory exudation; (3.) Atrophy; (4.) Changes in the *retina*, *optic disc*, and *vitreous humor*.

The signs of hyperœmia of the choroid are increased redness of the fundus, dilatation and varicosity, particularly of the larger choroidal veins; but turgescence of *choroidal* capillaries is not visible individually. The appearance noted in one eye should be compared with the other.

The signs of inflammatory exudation of the choroid vary according to the site of effusion, the amount and nature of the effusion. Infiltration of the stroma may occur generally, when it becomes swollen throughout; or there may be an effusion upon its retinal surface, or into the loose web which connects it with the sclerotic. In this latter site an escape of serum or of blood may separate, loosen, or so break the connections of parts that the choroid may be pushed inwards, and a similar occurrence on the retinal aspect of the *choroid* may separate and detach the retina, thrusting it forwards upon the vitreous humor. More rarely the products burst through the retina into the vitreous humor. A small quantity of slightly turbid serum soaking the stroma of the choroid is sufficient to confuse its ophthalmoscopic details, casting a faint haze over the fundus, which subdues its redness. Extension of the inflammatory process soon passes to the *retina*, and the vitreous humor becomes hazy, either by transudation of the inflammatory products through the retina, or rarely from direct influx through rupture of the tunic, and the opacity is greatest next the site of inflammation in the choroid.

The exudation generally appears of a grey, yellowish, brown, or opaque colour, in roundish spots or larger patches, which are the seat of infiltration, and which ultimately may be the seat of atrophic changes. These inflammatory foci, small and circumscribed, usually first appear at some distance from the optic disc, more rarely in its immediate neighbourhood, and are best distinguished by the intensity of colour between the spots (HULKE, BADER).

When the effusion has been slight and serous, rather than plastic, recovery may take place, when the haziness of the fundus clears off; but very generally the tissues which have been inflamed undergo *atrophy.*

The signs of atrophy consist in the inner surface of the sclerotic coming into view, unmarked by pigment, forming brilliant yellow white figures, bordered with black figures. The exudation either undergoes fatty degeneration and disappears, or it is transformed into fibroid tissue, leaving a bluish-white pearly scar on the choroid, said to be prone to ossification. [Specimens of ossification are to be seen in the Museum of Guy's Hospital (BADER.)] The blood-vessels of the atrophied part are obliterated, partly by the exudation and partly by changes in the contents of the vessels themselves. The transparency of the retina over and near the focus of inflammation is impaired in most cases (HULKE, BADER). *The state of vision varies.* It is distended, if inflammatory foci implicate the retina, especially at or near the *yellow spot;* and as regards prognosis, it is less favourable if the inflammation is at or near this yellow spot.

There are some signs peculiar to the different kinds of choroiditis:—

(*a.*) The large grey or yellow and opaque patches just noticed are characteristic of the simple or plastic form.

(*b.*) Lymph nodules are observed in *syphilitic choroiditis.*

(*c.*) A turbid red colour of the choroid, with increased tension of the eyeball and dilatation of the pupil, is characteristic of the serous form of choroiditis.

In syphilitic choroiditis the exudation may extend over a large and continuous area, or exist in *numerous* separate spots, which are yellowish, white, or opaque nodules, resembling those gummata seen in the *iris* (*syphilitic iritis*), and composed of closely packed caudate cells, which become fibres. They are seated among and upon the capillaries of the choroid. They may sometimes be seen projecting into the *retina.* They often occur round the *optic disc;* and morbid changes in the adjoining parts, especially opacities in the *vitreous humor*, are usual complications.

These are all so many sources of suddenly impaired vision—patients generally complaining of a mist intervening between objects and the eye; or, that parts of an object, or of the letters of a word appear missing; and that to see them the eye or the object has to be moved in certain directions.

In suppurative choroiditis the products are purulent, either in its substance or separated from its surface. The stroma of the choroid is generally destroyed if the infiltration be general; but sometimes only one part may be so affected, the rest having the appearance of a simple serous inflammation, hyperæmic, ecchymosed, and infiltrated with serum. Such suppurative choroidal inflammation is never uncomplicated—all the other parts of the eyeball partake in the process, so that it is really a *suppurative pan-ophthalmitis.*

Serous choroiditis is characterized by the inflammation being of a secretory character—a serous or gelatinous product passing through the *retina* into the vitreous humor, in which the cells of the choroidal stroma show very little change; but the whole of the textures of the eye seem to become implicated in producing the result usually seen in cases of *serous choroiditis.* Fluid is increased within the eyeball, the walls of the globe retaining their rigidity, inflammation of all the interior parts of the eye exist to a more or less extent, and general congestion of parts combine in very considerably increasing the intra-ocular pressure.

The condition known as *glaucoma* is thus brought about—a term formerly applied only to the last stage of *choroiditis,* when the pupil had become fixed, irregular, and dilated, its area greenish, the ciliary vessels enlarged, and the eyeball of stoney hardness, and vision lost. Now the term is applied to a series of lesions far short of these results, and which may be going on in persons apparently with perfect sight. It now implies a series of morbid changes in the eyeball, the most prominent of which (and one which seems to be the basis of all the others) is an increase in the tension of the eyeball, attributable to an increased amount of the *vitreous humor,* and to changes in the contents of the *vitreous chamber,* giving rise to disturbances in the circulation, nutrition, and functions of all the textures of the eyeball.

Sudden and paroxysmal attacks of chronic inflammation (choroidal at first, but implicating all the connections of that membrane) are characteristic of the conditions which bring about *glaucoma.* These attacks are generally sudden, with remissions; or, chronic, with exacerbations, and with few distinct external evidences. Objectively, in such cases, the disease is recognized by excavation of the papilla of the *optic nerve,* by varying degrees of hardness of the globe, and evidences of local congestion. Excavation of the papilla may occur gradually, with more or less marked increase in the *resistence* of the globe, without any indication of inflammation of the inner parts of the eye. Nevertheless, those appear sooner or later, so that symptoms of disturbed tension of the eyeball ought now to be accepted as evidence of *serous choroiditis,* if not of general involvement of the membranes of the eyeball in grave, though chronic inflammatory changes. The glaucomatous process is the same whether acute or chronic, rapid or slow, and the final result is the same. The occurrence, the succession, and the rapidity of the attacks of inflammation depend in a great measure, no doubt, upon the power which the eye possesses of adapting its nutrition and circulation to the disturbances occasioned by the increased tension of the eyeball. This increased hardness of the globe has long been recognized as the fundamental feature of the glaucomatous condition; and thus the student must be practically familiar with the normal tension of the eyeball. It means the resistance which we feel, or the depression which we see, when the eyeball is pressed upon in its equatorial region. In healthy eyes, it varies but slightly. The highest degree of increased tension exists when the eyeball can no longer be "dimpled" by pressure with the fingers. To ascertain the tension—

(*a.*) *By touch:—The eyelids are first to be closed gently, then rest one forefinger upon that spot of skin which corresponds to the* OUTER MARGIN *of the* superior rectus muscle, *near its sclerotic insertion, and place the other forefinger upon a corresponding spot near its* INNER MARGIN. *By gently pressing upon the skin and eyeball with one forefinger* (*as when examining for fluctuation*) *the tunics of the eye are felt to yield more or less readily to the pressure, while the spot upon which the other forefinger rests is somewhat raised.*

(*b.*) *By sight:—Direct the patient to look upwards, and gently press the inner edge of the margin of the lower lid upon the sclerotic. By such pressure the curvature of the sclerotic and that of the other tunics become altered, being pressed towards the elastic contents of the vitreous chamber. The amount of flattening or indentation indicates the degree of tension. If the tension is extreme the entire eyeball will be displaced, no visible indentation being produced.*

The optic disc is the first part to feel the effects of pressure indicated by the ophthalmoscopic appearance of excavation; and, temporary dimness of vision is what distresses glaucomatous patients most, and which coincides with paroxysms of inflammation.

There are also certain symptoms which indicate increase of intra-ocular pressure, namely,—very decided pulsation in the central portion of the retinal vessels; narrowing of the aqueous chamber; dilatation, sluggishness, or complete rigidity of the pupil; limitation of accommodation; and, anæsthesia of the cornea.

Treatment of Choroiditis is similar to that of the corresponding forms of iritis; but when atrophic spots appear on the choroid, no treatment can repair the damage. Mercury cannot be borne to the same extent as in iritis. Opiates are necessary, externally and internally, to overcome pain.

The secretion of the intestines, liver, skin, and kidneys, requires to be regulated.

RETINITIS.

LATIN EQ., *Inflammatio retinæ;* FRENCH EQ., *Retinite;* GERMAN EQ., *Retinitis;* ITALIAN EQ., *Retinitide.*

Definition.—*Inflammation of the retina, which always starts from the connective tissue framework of the membrane, diffused or circumscribed, with mistiness or darkening of the visual field, cloudiness, with or without hæmorrhagic extravasations, and great local congestion.*

Pathology.—The retina is a delicate, almost pulpy membrane, containing the terminal part of the optic nerve. It lies smoothly between the choroid coat and the vitreous humor, on the hyaloid membrane on which it rests, and to which it is closely adherent. It extends forwards nearly to the outer edge of the ciliary processes of the choroid, where it ends in a finely indented border—*ora serrata.* The thickness of the retina diminishes from behind forwards. In the fresh eye it is translucent and of a light pink colour, but soon becomes opaque after death. On its inner surface the following objects are to be seen:—

(*a.*) In the axis of the eyeball, about one-tenth of an inch from the outer edge of the optic nerve entrance, *the yellow spot* (*macula lutea*), transversely oval in shape, and somewhat variable in size (about $\frac{1}{20}$th of an inch in diameter), having in its centre a hollow (*fovea centralis*) where the retina is thinnest. The pigmentary layer of the choroid is visible through it, giving rise to the appearance of a hole.

(*b.*) *The retinal aperture* through which the optic nerve passes, known as the *optic disc* or *blind spot.*

In structure the retina consists partly of nervous elements and partly of modified connective tissue, which envelops and holds together the nerve-elements, entering into the constitution of all the layers of the retina. These nervous elements are arranged in layers, some like *mosaic,* and intimately connected with the specific irritation of the sense of vision; others in layers whose nerve-elements correspond with those occurring in all parts of the brain, so that they may be compared to a flat expansion of a central ganglion.

The several dissimilar strata are piled upon and connected with each other in the following histological order, commencing with the inner surface, namely:—

(1.) *The layer of optic nerve-fibres;* (2.) The *ganglionic layers,* consisting of a layer of "ganglionic cells," among which is found the larger number of blood-vessels; then follow (3.) A filamentous and finely granular layer; (4.), (5.), and (6.) An inner, inter-granular, and outer-granular layer; (7.) An external limiting layer of connective tissue; and (8.) The layer of rods and bulbs—the bacillary or columnar layer—or Jacob's membrane.

The *arteria centralis retina* is an off-set of the ophthalmic, which divides into four or five primary branches as soon as it enters the eyeball, and finally forms a network of very fine capillaries in the ganglionic layers of the retina.

Ophthalmoscopic Appearance.—With strong illumination in the fundus, especially by oblique light, the *retina* may be recognized as a very delicate, bluish-white mist covering the fundus, most marked in the immediate vicinity of the optic nerve. The *macula lutea* is recognized by the absence of the retinal vessels; frequently, also, by strong pigmentation of that part of the fundus, and by a peculiar reflection from the *fovea centralis.* The eye examined ought to fix itself upon the ophthalmoscope, and should be examined with a high power. The *optic disc* is brought into view when the cornea is turned slightly towards the middle line, as a circular greyish-pink disc, encircled by a double ring, and pierced by the trunks of the retinal vessels. These rings are the apertures in the *choroid* and *sclerotic,* the former being the smaller, and constricting the nerve the closest.

Morbid Anatomy.—The products of inflammation of the retina usually appear as an infiltration between and amongst its several layers, or collects upon one or both surfaces. Thus the elements of the retina are changed in various ways by the gelatinous coagulable bases of cell-proliferation. Fatty degeneration may ultimately predominate, so that the connective tissue becomes entirely destroyed. Hæmorrhagic extravasations are of frequent occurrence in the inflamed retina, of slight extent, but often very numerous; and in form they take the shape given them by the meshes of the tissue layers in which they occur. The layer of inflammatory products may be irregularly spread over the whole inner surface of the retina, when the vitreous humor becomes cloudy and the choroid participates in the proliferation.

Sometimes, as in Bright's disease, large quantities of an inflammatory product collect in the posterior half of the *retina* and *optic disc,* often causing there a decided swelling, and with great congestion of the veins, characterized by striated or spotted extravasations. This form of exudation quickly becomes fatty (VON CARION).

In other cases the product may be purulent, suppurative or tuberculous.

The following are the principal forms of *retinitis*:—

(1.) In *diffuse retinitis* there is regular or ill-defined cloudy opacity of the *retina* and *optic disc,* with obliteration of the posterior choroidal boundary; congestion of the larger vessels; inclination to hæmorrhage, and mistiness or darkening of the field of vision, which, increasing, usually calls the patient's attention to his condition. A fog seems to envelop anything he looks at. Sharpness of central definition is decreased with indistinctness of eccentric vision.

This diffuse form often depends on constitutional syphilis, which is solely indicated by the symptoms and history of syphilis, having no other special peculiarities to distinguish it.

(2.) *Exudative retinitis* is characterized by light-coloured spots, bordered by dark pigment appearing in the fundus of the eye, during or after diffused or circumscribed inflammatory retinal cloudiness; destruction of the pigment, and atrophy of the choroid tissue exists.

Externally, the eye may seem perfectly normal; but there may be cloudiness of the vitreous humor, rendering ophthalmscopic examination difficult. There is also mistiness and darkening of the visual field, corresponding in position to the points of exudation, described as thick, white or grey, or bluish mists, or as dark, snake-coloured, or black spots, or irregular interrupted rings, lying over limited parts of the field of vision.

Existing alone, without being diffuse, *exudative retinitis* appears to be most frequently associated with constitutional syphilis (HUTCHINSON).

(3.) *Nephritic retinitis* is characterized by collections of a cloudy substance in the posterior half of the retina, forming a patch-like prominence around the *optic disc.* The exudation is accompanied by numerous hæmorrhagic extravasations, great local congestions, and decrease of vision, which gradually sets in, occasionally interrupted by temporary arrests or improvements, and characterized by a generally irregular cloudiness or darkening of the field of vision, with or without peripheral limitation.

It occurs only in connection with Bright's disease of the kidney; appearing even in the early stage of albuminous nephritis; but more usually in the later stages of chronic cases. The morbid conditions of the kidneys have generally been fatty, lardaceous, or granular, contracted and atrophied, associated with this form of *retinitis.* It may appear whenever albumen is present in the urine, or the retinitis may even be the first prominent symptom of kidney disease.

Treatment cannot be given in detail, as the general hygienic and medical management must be directed towards the cause of the retinitis, and improving the general health of the patient, as indicated by the special case.

AMAUROSIS.

LATIN EQ., *Amaurosis;* FRENCH EQ., *Amaurose;* GERMAN EQ., *Amaurosis*—Syn., *Schwarzer Staar;* ITALIAN EQ., *Amaurosi.*

Definition.—*An affection characterized by certain organic changes in the optic nerve and retina, or other parts of the nervous system essential to vision, causing an inability to perceive objects which lie in certain portions of the visual field, with a distinctness corresponding to the amount of illumination and size of the visual angle. In bad cases the objects cannot be seen at all, when the condition is known as* AMBLYOPIA; *and when light cannot be distinguished from darkness it is known as* AMAUROSIS.

Pathology.—The disease has sometimes been considered as an independent substance affection, the lesion being a grey atrophy of the *optic nerve;* or it is an obscure affection due to visible disturbance of the nutrition of the part, causing lesion in some portion of the *optic nerve* or *retina,* and extending along the nerve-fibres. Inflammation may precede atrophy, arising primarily or extending from other parts of the eye—the atrophy then resulting from proliferation of tissue.

The conditions under which amaurosis has been brought about may best indicate its pathology. These may be summed up (from VON CARION) under the following heads:—

(1.) Mechanical injuries, solution of continuity or sudden contractions of the space of the *optic nerve* or *retina.* Hæmorrhagic extravasations are found which notably break up the nerve-elements, causing them permanently to loose their conductive power. If they are only pressed upon, and no inflammation follow, with progressive absorption of the extravasation, a partial or complete restoration of vision may follow. These extravasations are often seen in the *retina* as a result of hypertrophy of the left ventricle, coughing, or lifting heavy weights. Extravasation causing amaurosis has also been found in the *optic chiasma.* In two cases one or both roots of the optic nerve were found destroyed by splinters of bone, sprung from the base of the skull, in consequence of severe injury. Double amaurosis may result from apoplectic extravasation in the thalamus.

(2.) Sudden interruption to blood supply, as by *embolus,* with sudden and marked contraction of some or all the branches of the *arteria centralis retinæ.* There is evident arterial ischæmia, which may also be the result of retro-ocular proliferation, confined to the deeper-lying parts of the optic nerve and retina. Such a form of amaurosis is oftenest seen in anæmic conditions, as in the latter stages of *diabetes, albuminuria, syphilis, lead poisoning, uterine hæmorrhages, hæmoptysis, hæmatemesis;* and the blindness is generally in both eyes.

(3.) Amaurosis from the influence of material which poisons the blood and acts upon the brain, or affects the *optic nerve* and *retina*, as atropia acts on the *ciliary nerves.* The most marked cases of this kind are from *uræmia* and *lead poisoning; opium, Calabar bean, nux vomica,* and *tobacco.* The extreme use of *quinine,* and abuse of *alcohol* have also been known to produce amaurosis.

(4.) Congestion and hyperæmia of the eye have also produced the affection.

(5.) By far the greater number of cases are grouped together as due to intra-cranial changes of tissue, and known as *cerebral* or *central* amaurosis. But in such cases the impairment of vision is generally due to lesion of the optic nerve at the base of the brain, such as grey atrophy; which may not affect any part of the cranial cavity. *Basilar meningitis* is another local lesion which may lead to amaurosis, or periostitic affections of the basilar bones affecting the optic nerves, and inducing *hemiopia* of the same side in both eyes, or total blindness of one or both eyes. Tumors at the base of the brain, involving the nerve directly or by pressure, in or by the morbid growth, are another intra-cranial case of amaurosis. They are usually found at the base, especially about the *sella Turcica* and neighbourhood of cerebellum. Actual disease of the brain itself may also be a cause, such as *encephalitis, abscess, softening, tubercle, gummy tumors, hydatids, hæmorrhages;* but their relations to amaurosis are very complicated. Generally they cause amaurosis through exciting *meningitis,* which, spreading rapidly, extends to the intra-cranial portion of the *optic nerve,* or influences the *optic cavities.*

Thus the causes may be either *ocular* in the *retina, choroid,* or *optic nerve;* or *extra-ocular,* but *orbital;* or *cerebral,* or *spinal.*

Symptoms vary greatly in each individual case and stage of its course. Impairment of vision is the most prominent symptom, which may be irregularly distributed upon the various parts of the visual field. The field of vision must therefore be carefully and repeatedly examined in all cases of amaurosis; and a record kept from time to time, in order to gain a clear idea as to the amount of impairment of function, and as to the progress of the case. The following points must be especially determined:—(*a.*) Size and form of the whole field of vision; (*b.*) Central acuteness of vision and definition; (*c.*) Manner in which distinctness of perception diminishes in each sector of the field of vision towards the periphery. (See Stellwag, Von Carion, for "Methods of Measuring and Registering the Field of Vision.")

Prognosis.—All hope of recovery is past after *atrophy* of the *optic disc* and *retina* has set in.

Treatment must vary in accordance with the ascertained cause; and hence the student must learn the nature of the several diseases and their management, which are associated with amaurosis.

CHAPTER XVII.

DISEASES OF THE CIRCULATORY SYSTEM.

THE pathology and diagnosis of diseases of the heart and lungs—of the thorax generally, its contents and parietes—are all so intimately related to each other, that the diseases of the circulatory system are best introduced by some preliminary sections, the object of which is to show how diseases of the heart and lungs are especially recognized and appreciated by certain *symptoms*, local and constitutional, as well as by so-called *physical signs*. It is therefore necessary to notice, in these preliminary sections, certain topics, a knowledge of which is essential to the accurate diagnosis of these diseases. These topics may be arranged in the following order:—

SECTION I.—RELATION OF THE THORACIC VISCERA TO THE WALLS OF THE CHEST.

(A.) *Regions of the Thorax.*

In order to enable the student and physician accurately to describe and record their observations, and more conveniently to communicate to others precise information respecting the seat and signs of internal diseases, it has been customary to map out the exterior surface of the chest and abdomen by lines into different compartments termed *regions*. These lines have not always been drawn in the same manner; and therefore, on the contrary, there has ever existed great discrepancy among writers and teachers in this and other countries respecting the number, the extent, and the names of the regions defined by such lines. The regions are quite arbitrary, and the lines described upon the surface are understood to correspond with imaginary planes passing towards the centre of the body. The points or *landmarks* on the surface through which these lines are drawn ought to be at once fixed points and obvious to the senses; so that the boundaries and contents of every region may be accurately defined, with the view of localizing physical signs as precisely as possible.

As it is necessary that a complete inspection of the body shall take in simultaneously the abdomen and the chest, the topography of these regions is generally given together; and the annexed regional plans of the trunk are those devised by Mr. Paxton, of Oxford; and given by the late Sir John Forbes, in his excellent paper "On the Exploration of the Chest and Abdomen," in the first volume of the *Cyclopædia of Practical Medicine*.* Prominent points of the skeleton are here made the basis

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* To facilitate accuracy in this method of recording physical signs, blank outlines of the trunk of the body were first used by Piorry, and more recently they have been largely used both in clinical teaching and in recording cases for publication,

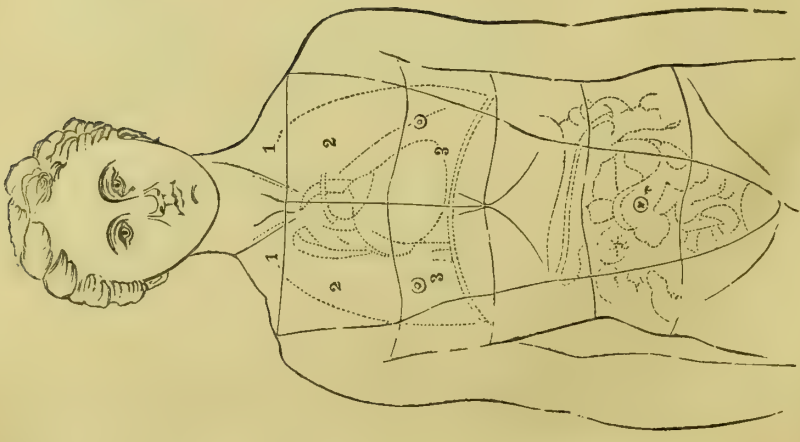

Fig. 7.

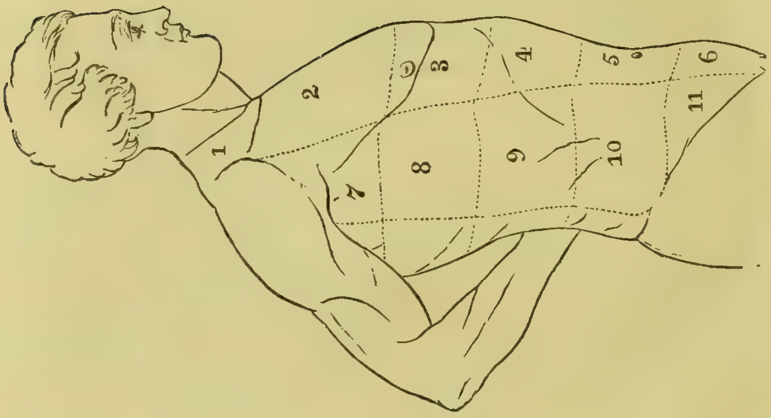

Fig. 8.

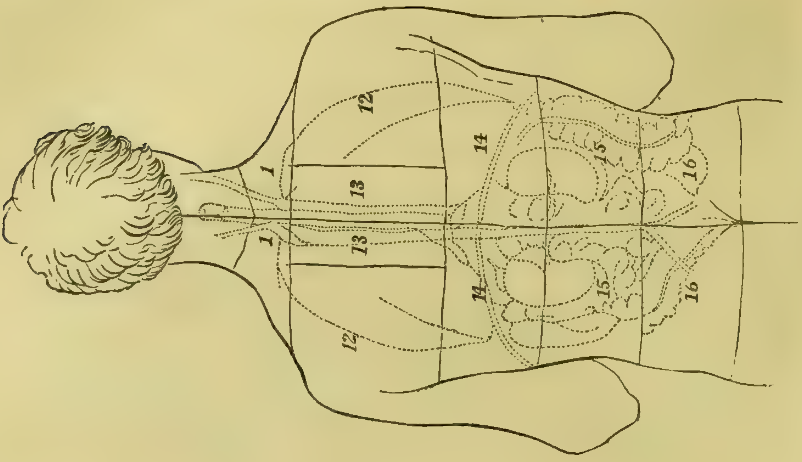

Fig. 9.

of the system by which the regions are mapped out by vertical and horizontal lines. The descriptions of the boundaries and contents of the regions have been compiled on the authorities of Sibson, Walshe, Parkes, Lyons, Fuller, Quain, Sharpey, Thomson, and Cleland.

The vertical lines having relation to the chest are *eight* in number, and run as follows:—(1.) Along the middle of the sternum, from its upper to its lower end; (2.) From the acromial end of the clavicle to the external tubercle of the pubes (right and left); (4.) Along the spinous processes of the cervical and dorsal vertebræ; (5.) Along the posterior

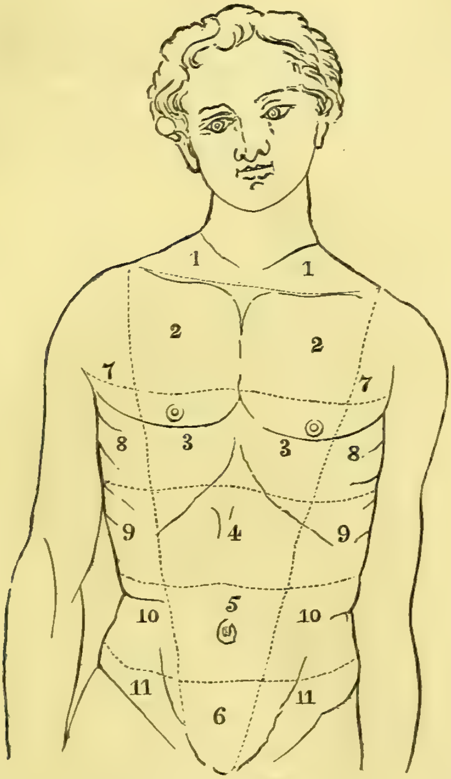

Fig. 10.

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by Professors Bennett, in Edinburgh, Gairdner, of Glasgow, and Beale, of London; the latter of whom has especially described the great advantages from their use, in the sixth number of the valuable *Archives* which he publishes (vol. ii., p. 97). The *outlines* merely of the trunk (without the dotted lines and the numbers), given in the text to illustrate the topography of the chest and abdomen, have been engraved on wood; so that the student can readily indicate the areas of physical signs by pencil, ink, or coloured chalk lines.

My publishers inform me that they are prepared to supply *fifty* blank copies of these outlines, at the cost of one shilling and sixpence, under the following title:—"Outline Figures for Indicating the Areas of Physical Signs in the Clinical Diagnosis of Disease;" for the use of Students and Practitioners of Medicine. Arranged by William Aitken, M.D., &c. Charles Griffin & Co.

or spinal border of the scapulæ, from the clavicular transverse line to the mammary transverse line. (See figs. 7, 8, and 9.)

The horizontal or transverse lines are four in number, and are as follows:—(1.) Around the lower part of the neck, sloping downwards to the upper end of the sternum anteriorly, and to the last cervical vertebra posteriorly; (2.) Around the upper part of the chest in the line of the clavicles; (3.) Around the middle of the chest by the lower edge of the *third* rib, above the line of the male nipple, and touching the inferior border of the scapulæ behind; (4.) Around the lower part of the chest, on a level with the xyphoid cartilage. (See figures as before.)

By these lines the lower part of the neck and the chest is divided into three horizontal and eight vertical bands; and by the intersections of these lines various compartments or regions may be indicated.

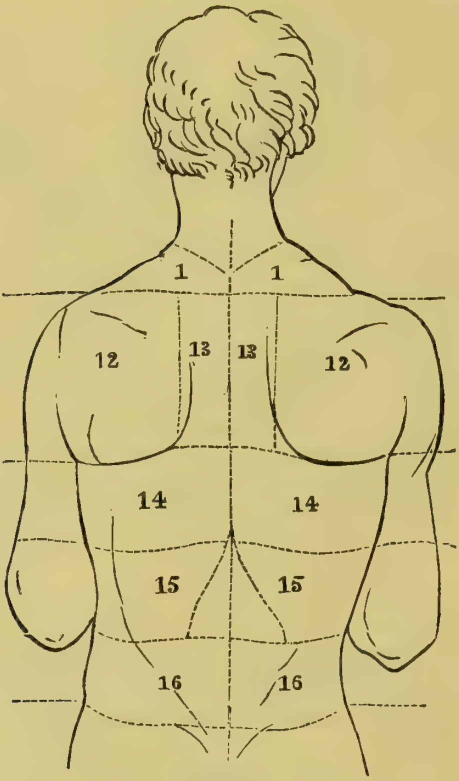

Fig. 11.

The most useful arrangement of the compartments and nomenclature of the regions is as follows:—

Anterior, *lateral*, and *posterior* regions being recognized, the *anterior* are named the *supra-clavicular*, *clavicular*, *infra-clavicular*, *mammary*, *infra-mammary*, *supra-sternal*, *superior sternal*, and *inferior sternal*.

The *lateral regions* are,—the *axillary* and the *infra-axillary*.

The *posterior regions* are,—the *supra-spinous region;* the *infra-spinous* *region* (sometimes called the scapular); the *inter-scapular;* the *infra-scapular* (sometimes called the upper dorsal). Of these regions the three sternal are single; all the rest are double.

The Supra-Clavicular Region is a small triangular space *above* the clavicle on either side, with its base internally at the trachea, its apex towards the outer end of the clavicle, and bounded below by the upper edge of that bone. A line drawn from the outer part of the clavicle to the upper rings of the trachea will limit its upper border.

In this region is found the triangular apex of the lung (figs. 10 and 11), sometimes reaching on the right side a little higher than on the left, with portions of the subclavian and carotid arteries, and of the subclavian and jugular veins. The floor of this region internally is formed by the upper surface of the first rib.

The Clavicular Region is very narrow and oblong, corresponding to the inner two-thirds of the collar-bone. Behind the bone lies on both sides lung-substance; but on the right side, at the sterno-clavicular articulation, is the *innominate artery,* and the *subclavian artery* crosses the region at its outer edge; on the left side the *carotid* and *subclavian arteries* pass upwards, almost at right angles to the bone.

The Infra-Clavicular Regions are nearly square, corresponding to No. 2 in the figures already referred to. Each region (right and left) is bounded above by the inferior border of the clavicle; below by the lower border of the third rib, where it joins the cartilages of the sternum; it is bounded on the outside by the vertical line passing from the acromial end of the clavicle downwards towards the external tubercle of the pubes (on either side); on the inner side the subclavian region is bounded by the edge of the sternum.

Within these limits is placed the upper lobe of the lung on both sides, close to the sternal border of the region. On the right side lie the superior *vena cava,* and a portion of the arch of the aorta. On the left side, close to the sternum, is the edge of the pulmonary artery.

The inferior border of the region on the left side corresponds to a portion of the base of the heart; while part of the right auricle occupies the inferior corner of the region towards the sternum on the right side.

The Mammary Region (No. 3, figs. 7, 8, and 9) has also a square-like form, and is bounded above by the line passing through the lower border of the *third* rib, where it joins the cartilages of the sternum below, by the line passing transversely on a level with the xyphoid cartilage (its upper border); outside by the vertical line passing to the outer tubercle of the pubes (on either side); and on the inner aspect by the edge of the sternum.

The contents of the mammary regions differ greatly on the two sides.

On the right side the lung lies throughout immediately underneath the ribs. Its inferior border turns off almost at right angles from the sternum, at the cartilage of the sixth rib, whence its thin sharp border gently slopes outwards and downwards, so as to occupy the lower part of the region when the diaphragm is depressed. But when the diaphragm is elevated, the liver rises into this region up to the fourth interspace. The fissure between the upper and middle lobes of the right lung passes upwards and backwards obliquely across the region from about the *fourth* cartilage. The fissure between the middle and lower portion passes in the same direction from the *fifth* interspace. A portion of the right auricle, also a portion of the right and superior angle of the right ventricle, lie between the *third* and *fifth* ribs, close to the sternum, in the right mammary region.

On the left side, at about the level of the *fourth* cartilage, the anterior edge of the left lung passes obliquely downwards, having abruptly left the edge of its fellow on the opposite side, so as to expose a free space of variable size for the heart (fig. 12). The edge of the left lung thus reaches the *fifth* rib; whence it comes inwards and downwards to opposite the *sixth* rib or interspace, whence it finally passes nearly horizontally outwards and downwards into the lateral region. The anterior point of division of the lobes of the left lung lies about the fifth interspace, below the nipple. The left auricle and the left ventricle, with a small portion of the right ventricle about the apex, lie in the left mammary region; the apex of the heart generally lying immediately above the *sixth* costal cartilage (fig. 10 and fig. 12, under the description of the heart, a little farther on).

The Infra-Mammary Region has a somewhat triangular form. Its boundary alone is defined by the lower bounding line of the mammary region on each side; below, by the margins of the false ribs; inside, by the xyphoid cartilage or middle line; and outside, by the extension of the line from the acromial end of the clavicle to the outer tubercle of the pubes on either side.

On the right side this region contains the liver, with the edge of the lung overlapping it in front, to a variable extent during full inspiration.

On the left side the stomach and anterior edge of the spleen rise as high as the *sixth* rib in this region; and towards the inner portion of the region the left lobe of the liver lies in front of the stomach.

The Supra or Post-Sternal Region is a small hollow, bounded below by the notch of the sternum, and laterally by the *sterno-mastoid* muscles.

The trachea almost completely fills this region; but on the right side the innominate artery lies at the lower angle; and in some persons the arch of the aorta reaches its lower border just at the notch of the sternum, where it may be felt pulsating. The region contains no lung.

The Upper or Superior Sternal Region comprehends that portion ot the breast bone which is superior to the lower border of the *third* rib.

It covers the left and a small portion of the right innominate vein. The superior cava runs along its right edge; the ascending and transverse portions of the arch of the aorta; the pulmonary artery from its origin to its bifurcation; the aortic valves near the lower border of the *third* left cartilage at its junction with the sternum, or midway between the mesial line and the left edge of the sternum (the pulmonary valves being a little higher than these, more near the surface, and quite at the left of the sternum); and lastly, the trachea, with its bifurcation, on the level of the *second* ribs. The remains of the thymus gland, with areolar tissue and fat, lie in front of these parts, between the lateral pleural boundaries of the upper V-shaped portion of the anterior mediastinum, thus separating the edges of the lungs above towards their apices.

The Lower or Inferior Sternal Region comprehends the remainder of the sternum which lies below the level of the lower margin of the *third* rib. It contains the greater portion of the right ventricle, with the infundibulum of the pulmonary artery, and a small part of the left ventricle. The *mitral valve* is situated towards the upper end of this region, close to the left edge of the sternum, on a level with the *fourth* sterno-costal articulation; the *tricuspid valve* lies nearer the middle line, and more superficially. The edge of the right lung descends vertically along the middle line, and at the upper part of the region is a small portion of the left lung. Inferiorly and deeper seated is a portion of the liver, and sometimes of the stomach, while the line of union of the heart and liver corresponds with where the diaphragm intervenes.

The Axillary Region (figs. 7 and 8, No. 7) extends from the apex of the armpit above to the line continued which marks the inferior border of the infra-clavicular region. In front it is bounded by the posterior border of the infra-clavicular region, and it extends to the external edge of the scapulæ behind on either side. The region can only be brought into view by lifting the arm over the head, or by carrying it away from the side (as in fig. 8). The region is hidden (as in fig. 7) when the arm is at rest by the side. It comprehends portions of the upper lobes of the lungs, a great volume of lung-substance, and, more deeply seated, large bronchi.

The Infra-Axillary Region (figs. 5 and 6, No. 8) is bounded above by the region already defined; anteriorly, by the mammary region; posteriorly, by the scapula; and below it extends to the margins of the ribs.

It contains on both sides the lower edge of the lung, sloping downwards from before to behind. On the right side, also, is the liver, between which and the walls of the chest is interposed a thin layer of lung-substance during a full inspiration. On the left side is the spleen and stomach.

The Supra-Spinous Regions have the same boundaries as the superior fossæ of the scapulæ, and correspond to the posterior surfaces of the apices of the lungs (figs. 9 and 11, No. 1).

The Infra-Spinous Region, sometimes called the Scapular (figs. 9 and 11, No. 12). It is identical with the lower fossa of the scapula. It covers lung-substance.

The Inter-Scapular Region (right and left) lies between the inner margin of the scapulæ, divided into a right and a left region by the vertebral column, from the second to the sixth dorsal vertebra.

It covers lung-substance on each side of the mesian line, the main bronchi, and bronchial glands. On the left side is the œsophagus; and from the *third* or *fourth* vertebra downwards is the descending aorta. The bifurcation of the trachea takes place at the middle line between the two regions, but inclining rather to the right side (figs. 9 and 11, No. 13).

The Infra-Scapular or Lower Dorsal is bounded by the continuity of the transverse line, which forms the inferior boundary of the infra-clavicular region, and which, being continued behind, crosses the inferior angles of the scapulæ and seventh dorsal vertebra. It extends below as far as the twelfth rib, corresponding to the transverse line, carried round, which formed the lower boundary of the *infra-mammary* region in front.

As low as the eleventh rib lie the lungs. On the right side, from the level of the eleventh rib, extending downwards, is the liver. On the left is the spleen, occupying some of the outer portion of the region; while the intestines occupy some of the inner part of the region. Close to the spine, on the left side, is the descending aorta; and on both sides, close to the spine, is a small portion of kidney (fig. 9 and 11, No. 14).

(B.) *Situation of the Organs in the Thorax.*

The *outer boundary* of each lung is marked by a line passing obliquely downwards and outwards from a little outside the centre of the clavicle towards the axilla, and then vertically at a variable distance outside the nipple. Each lung rises from half an inch to an inch and a half above the clavicle, the relative height being unequal but variable. The inner margin of each lung passes downwards and inwards from the apex, and meets with the inner margin of the other lung at the middle line, at a point between the first and second cartilages, or at the second.

The inner margin of the right lung continues vertically downwards along the centre of the sternum, or inclining a little to the left side, as far as the attachment of the xyphoid cartilage (fig. 12).

The inner margin of the left lung leaves the right at a point between the *fourth cartilages,* or a little higher or lower than this, and passes nearly transversely outwards for a short distance in the direction of the *fourth costal cartilage.* It then runs obliquely downwards and backwards in the course of a line drawn from the centre of the *fourth costal cartilage,* half an inch to one inch inside the left nipple, as low as the seventh rib (fig. 12).

The lower boundary of the right lung passes somewhat obliquely and then transversely from the attachment of the xyphoid to the sternum, across the cartilages of the sixth and seventh ribs backwards to the spine, which it touches on a level with the *tenth, eleventh,* or *twelfth* dorsal vertebræ (fig. 11).

The lower boundary of the left lung is a little lower than that of the right, and passes backwards from the point indicated on the seventh rib, to strike on the spine at a point usually a little lower than that on the right side.

The apex of each lung lies beneath the anterior scalenus muscle and the subclavian artery. The apices of the lungs are separated from each other by the œsophagus, the trachea, and the projection anteriorly of the bodies of the *first* and *second* dorsal vertebræ.

The base of the right lung is hollowed by the projection upwards of the liver, which in the centre of the thorax ascends as high as the *fifth* rib or *fourth* interspace. The liver is also separated from the ribs by the expansion of the lungs between it and the thoracic walls.

The base of the left lung may be also pressed upon by the left lobe of the liver; and it is always hollowed out, though to a less degree than the right lung, for the accommodation of the stomach and spleen, and, to some extent also, the left lobe of the liver.

The *heart* lies between the two lungs. The right auricle and a part of the right ventricle are covered by the right lung, the rest of the right ventricle being left bare by the divergence of the left lung from the middle line. The left auricle is covered by the right auricle and by the left lung. The left ventricle lies behind the right ventricle, but projects a little towards the left side, where it is uncovered for a short distance, beyond which its left border is covered by the left lung.

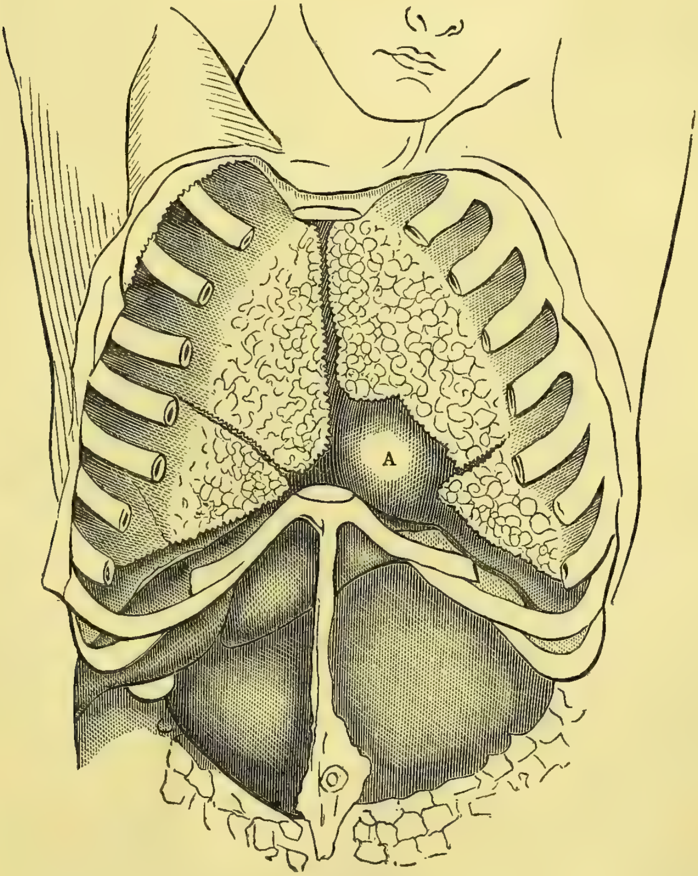

Fig. 12.*

The region corresponding to the portion of the heart uncovered by the lung (fig. 12) is sometimes called the *præcordial* region or space. The *upper boundary* of this space is where the inner margins of the two lungs separate—namely, at the spot between the *fourth cartilages.* The *outer* boundary of the *præcordial* space is indicated by the diverging line of the inner margin of the left lung passing along the *fourth cartilage,* and then obliquely downwards, inside the left nipple. The *inner* margin of the space corresponds to the nearly straight inner margin of the right lung, behind the sternum, near the middle line. The *lower* boundary of the *præcordial* space is indicated by a line passing from the junction of the sternum to the xyphoid cartilage, directly to the left, or with a slight inclination downwards. Above this line is the right ventricle, and farther out is the apex of the left. Below it is the left lobe of the liver, and the stomach, separated only from the heart by the diaphragm and the pericardium.

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* Relative positions of the margins of the lungs to each other, to the thoracic walls, and to A, the *præcordial* region, comprehending the right ventricle of the heart, covered by its pericardium (after Dr. Fuller. See his work *On Diseases of the Lungs and Heart*).

The *præcordial* region is thus slightly pyramidal in shape, its base being about two and a half inches long, and nearly horizontal. Its inner margin is about two inches long, and nearly straight; its outer margin is from three to three and a half inches long, and has a sloping direction from the apex to the pyramid (at the middle line between the *fourth* cartilages), to the outer extremity of the line indicating the base.

This *præcordial space* corresponds to the left half of the lower portion of the sternum, and to portions of the cartilages of the *fifth* and *sixth* ribs; and it may reach even to the junction of the cartilages with their ribs. Its inner and outer boundaries can be marked out only by light percussion; and the lower boundary can only be defined with difficulty by the same means.

Below the boundary of the right lung the liver extends to the margins of the right false ribs, or a little below them. On the left side the space between the lower border of the lung and the false ribs is occupied by the left lobe of the liver, the stomach, the spleen, and by a portion of intestine—the colon principally. The left lobe of the liver stretches across beneath the xyphoid cartilage, and below this to a variable extent towards the left side.

(C.) *Changes in the Position of the Lungs.*

1. *In Health.*—(*a.*) During inspiration the lungs enlarge in all directions; the apices rise higher and the bases descend lower down; the points of union and of division between the inner margins are in the one case raised towards the apices, in the other case lowered down. The *præcordial* space is thus lessened in size by the advance of the anterior margin of the lungs. (*b.*) During expiration the state of things is reversed. The lungs fall from each other; but the point of division between the inner margins may be raised to a level with the *third* rib. The area of the præcordial space is increased. (*c.*) The difference between extreme inspiration and extreme expiration is considerable. In extreme inspiration the inferior boundaries of the lung are often from an inch to an inch and a half lower than they are in extreme expiration. (*d.*) The action of the heart causes a slight

difference. Each impulse presses aside the sloping inner margin of the left lung; but this is so instantaneous that it causes no appreciable alteration when the *præcordial* region is mapped out by percussion. (*e.*) During respiration the thorax enlarges in all directions by the movements outwards and upwards of the superior ribs and sternum, and by the movements downwards and outwards of the inferior ribs. *In women* the movements of the upper ribs are much greater than in men, while the abdominal movements are less. The difference is increased by the use of stays; but it does not appear to be altogether owing to these. In boys the costal movements are often considerable: in old age they are diminished.

2. *Changes by Age.*—In children the still considerably developed thymus gland separates the inner border of the lungs at the top of the sternum; and the point where they come in contact (converging from the apex) is lower than in adults. In children the lungs are also comparatively longer than in adults, and the inferior boundaries are lower down. In old people the lungs often alter considerably in shape, and produce corresponding alterations in position; the lower lobe, particularly in the left lung, becomes more posterior, and the upper lobes or lobe anterior. The lungs at last in old people become even larger above than below; and when mapped out by percussion, their several boundaries are found to have no certain and constant direction.

3. *Changes by Disease.*—One lung being incapacitated, the other lung undergoes supplementary enlargement; the inferior boundary is lowered, its inner margin is pushed to a variable extent across the median line over the heart, diminishing the *præcordial* space from the right or left side, according as the right or left lung is affected. So, also, if one lobe be affected, as by *pneumonia*, *tubercle*, or *cancer*, the other lobe, either upper or lower, as the case may be, is enlarged, and changes its position. In some diseases, as in emphysema, the lung enlarges: and if the emphysema is general, the lungs may meet each other almost at the top of the sternum; may not separate till on a level with the *sixth* rib; may leave the inferior margins at the *seventh*, or even the *eighth* rib; and may give a pulmonary percussion note in the posterior lumbar regions, below the ribs altogether. In cases of enlarged heart, or distended pericardium (unless there be coincident emphysema, or unless the lungs are floated forwards by *hydrothorax*), the lungs anteriorly are pushed aside, the point of separation of the inner margin is raised (especially in pericardial effusion), the inner margin of the right lung is thrown to the right side, and the inner margin of the left lung is thrown to the left side. Aneurism of the arch of the aorta, or tumors in the mediastinum, displace the upper portions of the lung; and tumors may even thrust aside and displace the heart. Abdominal diseases, as hepatic and splenic affections, peritoneal effusions, ovarian, uterine, or other tumors, also press up the thoracic organs, and alter their position. By such morbid states the inferior borders of the lungs may be not lower than the *third* intercostal space, or the *fourth* rib; the heart may be thrust upwards and outwards above and outside the left nipple. The student may also be prepared to meet with cases of more or less complete transposition of the viscera. (See a paper on this subject by Professor Allen Thom-

son, in the *Glasgow Medical Journal*, vol. i.; also in the *Lancet* for August 8, 1863, by my colleague, Professor Maclean.)

SECTION II.—SIGNS OF DISEASE FROM THE SHAPE OF THE THORAX.

The two halves of the thorax are seldom perfectly symmetrical. The right side in the most healthy persons often measures from *half an inch to an inch* MORE than the left; the right *infra*-clavicular space, particularly in right-handed persons engaged in laborious occupations, is apt to be slightly more prominent than the left; the *fourth*, *fifth*, and *sixth* left cartilages often project more than the right; the *infra*-mammary and *infra*-axillary regions may be larger on the left than on the right side; the *infra*-scapular region, on the contrary, is usually larger on the right side, or it may be markedly prominent on both. The nipples (which in adult males are above the upper margin of the *fourth* rib, near its junction with the cartilages) are always equidistant from the middle line, but the left is somewhat lower than the right.

Any marked changes beyond those now noted indicate disease either of the spine, as in *curvature;* or of the spine and ribs, as in *rickets;* or of the organs in the thorax. If *spinal curvature* and *rickets* are determined not to be present, any general expansions of one side as compared to the other, or general contraction, or (what is much more common) local expansion or bulging, or local contraction, depression, or excavation, become very important signs.

The existence of such conditions is ascertained by the eye—namely, by "*inspection*," and accurately by measurement with calipers or with tapes, or ingeniously devised instruments which have been named "*stethometers*" or "*chest measurers*."

General expansion of the thorax is usually produced by effusions of fluid into the pleura, or by extreme cancerous infiltration, or by emphysematous lungs. Much more rarely chronic consolidation will cause general or partial *bulging;* while tubercular deposition at its earliest period has been said also to cause some degree of local prominence; but *retraction* usually follows more or less complete impairment of the functions of the lung, or of a part of it, as in softened tubercle or cancer, and in the period of absorption of pleuritic fluid which has firmly compressed the lung. Heart diseases give rise to local bulging only occasionally in the cardiac region. Aneurisms of the aorta may cause bulgings, while tumors may produce both bulgings and retractions according to circumstances (*MS. Notes* of Dr. Parkes' Clinical Course at University College).

SECTION III.—PHYSICAL EXAMINATION OF THE CHEST.

I. **By Simple Inspection of the Form of the Thorax.**—It is necessary to note,—

(*a*.) Its general shape; and especially as to the condition of the *supra* and *infra*-clavicular spaces in respect of flatness, fulness, retraction, or bulging in these regions; the condition of the hollow above the notch of the sternum (the supra-sternal space); the form of the clavicles and their curvature; the height and breadth of the shoulders; the form of the sternum, as to whether or not it bends outwards or inwards; the curves of the spine; the position of the scapulæ; prominence of their inferior angles, and the firmness or laxity of the *latissimus dorsi* muscle; the distance of the scapulæ from each other and from the middle line; contracted contour or expanded form of the lateral regions; the width, depression, or bulging of the intercostal spaces; and distance of the nipples from the middle line.

(*b.*) Obtain a *general notion as to capacity or size of the thorax, relative to the height, weight, and age of the individual, allowing for fatness or emaciation,* (spirometry; see previous pages, under "*Scrofula,*" p. 27, vol. ii.)

The object of spirometry is to measure the amount of air received into the lungs, so as to learn what the lungs can do. Practically and independently of all instruments devised for this purpose, one of the best modes to determine what the patient can do with his lungs is to make him count *one, two, three, four, &c.*, on till he has no more breath left in him to count, and requires "*to fetch a breath.*" By practice he may now and then count more than he did before; but every one has a number to which they can thus reach, normal to themselves, when the lungs are efficient in capacity.

Hutchinson's spirometer is unwieldy, inexact, and difficult to use; and none as yet have been devised adaptable to useful every-day practice.

(*c.*) Observe the thoracic movements, and estimate in seconds the time taken to complete the inspiratory and expiratory acts; compare the movements of the two sides of the thorax, and also the abdominal respiratory movements with those of the thorax, so as to notice if either takes an undue share in the work of respiration. If the ribs scarcely move, and the parietes generally of the thorax remain at rest, while the surface of the belly rises and falls alternately with the respirations, the act is called *abdominal respiration,* because the abdominal muscles seem to take the larger share in its performance; but if, on the other hand, no motion of the abdomen is visible, the act of respiration is then said to be *thoracic.* Observe whether the whole acts of respiration are quicker or slower than natural—*i.e.*, more or less than *eighteen* to *twenty* per minute, or one to every four arterial beats. Note their frequency per minute. Observe whether they are calm, easy, and fully drawn, or short, forced, hurried, and incomplete, attended by indications of pain, checked or partially arrested by cough. Notice whether respiration is performed through the mouth or nose, or both; and whether the *nares* dilate and contract at each respiratory effort, with any constrained movement of forcible expansion. Note any sensible odour or vapour of the breath, and also its temperature.

II. **By Measurement.**—The use of tape or calipers in deep and medium inspiration and expiration will detect any differences between the two sides, or undue differences in the size of the chest at different times. There is about one inch of an average difference in favour of the right side of the chest compared with the left, and which is consistent with a normal state of the region. A convenient plan is the double tape, originally suggested by Dr. Hare. The double tape is formed by joining two common measuring tapes together, so that the beginning of each may be in the centre of the tape when joined. By putting the point of junction of the two tapes upon the spine, and holding it there tightly, the ends of the tape can be carried round the body at any point, and the circumference of each side read off simultaneously. By taking the size at full expiration and full inspiration, the extent of *expansion* is determined, as well as the absolute and relative size of each half of the thorax.

The measurements (besides the circular) which are most useful are the distance of the nipples from the middle line—their distance from the sterno-clavicular articulations of each side—and the distance of the centre of the clavicle from the lowest point of the false ribs in the vertical line.

The general expansion of a side of the chest is best learned by the use of the double tape, or by the use of some of the "*chest-measurers*" about to be noticed. The difference in the measurement between the fullest inspiration and the fullest expiration gives the general expansion of the lung. In health both lungs expand nearly equally from *three-quarters of an inch to an inch and a half;* or the right may expand a little more than the left. If there is any deficiency of expansion, there must be disease of some kind, but the nature of the disease cannot be known without additional signs.

Local expansion is most accurately determined by the eye, the hand, and the chest-measurers of Drs. Quain, Sibson, or Leared; the exact levels of the measurements being always noted. All these instruments essentially consist of dials, with indices, moved by mechanism, connected with tapes passing round one or both sides of the chest—so many revolutions of the index indicating on the dial so many tenths and hundredths of an inch of expansion of the chest. Dr. Leared's instrument indicates differences on the two sides (*Med. Times and Gazette*, August 2, 1862); and Mr. Henry Thomson, of University College, has also suggested a simple addition to the tape measure, by which it is made differential. These instruments may be had of Coxeter, Weiss, Ferguson, Matthews, and other surgical instrument makers in London. (See New York *Medical Journal*, vol. vi., 1868, for an account of a good *Stethometer*, devised by Dr. Alfred C. Carroll, of New York.)

Dr. Sibson gives the following numbers as denoting the movements of various parts of the thorax in health:—

(1.) The sternum and the first *seven* ribs in tranquil breathing advance forward from ·02 to ·07 inches. The left *fourth*, *fifth* and *sixth* cartilages, and the *sixth* rib, move less than on the right side, on account of the position of the heart. In forced inspiration the movement forwards is from *half an inch* to *two inches.* (2.) The expansion of the *eighth* and *tenth* ribs varies from ·05 to ·1 inch. During deep respiration it is increased, but is less than that of the first five ribs. (3.) The abdomen moves forwards in tranquil inspiration from ·25 to ·3 inches. In deep inspiration the movement amounts to about *one inch.*

There is, however, very great variations in different persons; and "*chest-measurers*" must all be submitted (as *percussion* and *auscultation* are) to the test of comparison between the two sides—the difference which the heart causes between the two sides being remembered (Parkes, *M.S. Notes*).

It is necessary in all these observations so to divert the patient's attention as to cause him to look away from the instruments, otherwise the movements of the chest may become so affected as to vitiate the results.

III. **By Palpation,** or the application of the hand. This method affords more extended information to that suggested by simple inspection, such as of the intercostal distances, lateral and antero-posterior; expansion of the chest in the acts of respiration; appreciation of *vibrations* communicated through the walls of the thorax. The palm of the hand applied to the chest in a healthy state during the act of speaking will appreciate a most delicate *vibratile tremor* (commonly called *fremitus*), and more marked according to the graveness, coarseness, and loudness of the speaking voice. The intensity is generally greater in front than behind, on the right side than on the left, and is stronger towards the sternal than the humeral halves of the region below the clavicles. In disease the *fremitus* on the two sides of the chest must be compared. It is usually *increased* by whatever consolidates the fine vesicular texture of the lung, without obliterating the bronchial tubes. It is *diminished* by the intervention of liquid or air between the lung and the thoracic walls; also by such extensive consolidation of the lung-substance as to fill up the smaller bronchial ramifications leading to the air-vesicles. *Vocal fremitus,* or *vocal vibration,* is observed by placing the hand over the surface of the chest of a person speaking. It is a delicate vibration, easily deadened by the too forcible pressure of the hand. It is more marked in adults than in children, and in males than in females. It is more intense in long-chested than in short-chested persons; and markedly so in thin than in fat people; and is stronger in recumbency than in the sitting posture.

A *rhonchial fremitus* may sometimes be heard, when certain *rhonchi* throw the bronchial tubes into vibrations sufficiently strong to be felt on the surface of the chest. The sibilant sonorous and mucous rhonchi have all this property. *Friction fremitus* may be felt by the hand, when the gliding motion of the pleural surfaces upon each other is attended by a perceptible vibration, by the collision and friction of plastic matter upon the surfaces.

Ordinary fluctuation may sometimes be detected by the hands, combined with shaking or suddenly altering the position of the chest, as by *succussion.*

The hand is also used to appreciate the action of the heart. The heart, after full *expiration,* is felt to beat between the cartilages of the third and fifth ribs, and at the neighbouring part of the sternum, generally immediately below and to the outside of the left nipple. After a full inspiration it may be felt as low as the sixth rib. The frequency of the respirations may also be ascertained by the hand, applied to the surface below the clavicles in the female, and below the epigastrium in the male (WALSHE).

IV. **By Percussion.**—The position of the organs, as stated in the previous section, is ascertained during life principally by the signs derived from percussion; that is, from the nature of the sound produced by striking over the lung either "immediately," as when the chest is gently tapped with the hand or a light hammer; or "mediately," as when a flat body is placed upon the chest, which is then struck by the finger or the hammer. The main object of percussion is to determine the comparative density of subjacent parts.

Mediate percussion is employed almost to the exclusion of immediate. The body which is interposed between the hammer and the chest is called a "*pleximeter*," and consists simply of a flat piece of ivory or wood, or a piece of india rubber, or of the index finger of the left hand laid flat upon the chest. The hammer is made of a piece of whalebone, or of light steel, armed at its head with a piece of india rubber; or the flat edge of the stethoscope encircled by a piece of india rubber; or it consists of a light thimble covered with vulcanized india rubber; or the points of the index finger, or middle fingers of the right hand, may be used for the purpose of a hammer, the nails being cut close.

Mode of Percussing.—The pleximeter is to be placed flat upon the chest, and held close to the walls. It is then to be struck perpendicularly, and with gentle or moderate force. If the fingers of the right hand be used as the hammer, the blow should be given from the wrist only, the elbow and shoulder being quite motionless. The force of the blow must vary according to circumstances. In the supra-clavicular spaces the finger, if it be used as a pleximeter, should be pressed closely into the triangular space, and the blow should fall in the direction of a line passing to the centre of the chest—namely, downwards, forwards, and inwards. If the blow be directed too much forward or backward, it strikes on the clavicle, or the parts at the back of the neck, and the "pulmonary percussion note" is not obtained. The clavicles are best percussed *immediately*, first at the sternal and then at the acromial end. In the *supra-spinous* spaces the blow should fall downwards, inwards, and forwards. Over the rest of the chest the pleximeter may be placed in any direction, provided the precaution is taken that corresponding points shall be struck on the two sides; as a slightly different note is given out, especially in thin people, by the intercostal spaces and the ribs. If the spaces are struck on one side, they should also be similarly, or with like force, struck on the other.

The "*pulmonary percussion note*" is the sound given forth by striking on a part of the chest-wall below which lies a considerable depth of lung. The sound produced does not depend upon the proper tissue of the lungs, but upon the vibrations of the air they contain, and also upon the vibrations of the walls of the chest. The pulmonary substance vibrates but little; it is non-sonorous, and acts rather as a damper of sound. The qualities of sound to be noted are—(1.) *Amount in intensity of resonance*, commonly called *clearness;* (2.) *Duration of the sound;* (3.) *Pitch;* (4.) *Volume of sound.*

The intensity of sound is diminished by the lessened vibration of the walls of the chest, as in pleuritic effusions; or by the lessened vibration of the air, the air being lessened in amount; or by increase in volume of the lung-substance, as by exudation.

The physical conditions which lessen the quantity of air are—(1.) Consolidation of lung-substance from any cause such as exudation, tubercle, great congestion, or œdema; (2.) Compression of the lung from fluid in the pleura, or by tumors.

When the pulmonary percussion note is exaggerated, it may become what is commonly termed "*tympanitic;*" that is, a very clear sound, of less volume, but of higher pitch than the pulmonary percussion note. It is similar to the sound obtained by striking the abdomen when the intestines contain air, or a bladder moderately distended with air. It is generally caused by an increased quantity of air in the lung tissue or in the pleura, as in pulmonary emphysema, or when cavities exist in the lungs in pneumo-thorax. A very thin and flexible wall will often give a tympanitic character to the pulmonary percussion note. There are some exceptions to these statements, namely—(1.) When the air is not apparently in excess, a thin layer of sound lung full of air, lying over hepatized lung, will give forth the sound; (2.) The upper lobes of the lung when hepatized in the lower lobes will also give forth the sound; and (3.) When the lung floats on the surface of pleuritic fluid. *Tubular* sounds are produced when the lung is consolidated, covering bronchial tubes. In pleurisy the intensity of sound is diminished from both causes.

The sound is intensified or made more clear by whatever increases the vibrations of the walls of the chest, as in thin children, or in persons becoming thin, and by increased vibrations of air in the lungs.

The sound elicited by percussion of the chest varies with the region explored. The clearest sounds are obtained from the anterior regions, where the walls are thin; the least clear from the posterior, on account of the thickness of the walls. The degree of clearness also varies in the several *anterior* regions of the chest.

In the supra-clavicular region, the resonance ought to be very clear, approaching a tympanitic sound over the clavicle; where the sound ought to be clearest in the middle third; of high pitch in the inner third; but duller towards the scapular end.

In the infra-clavicular region—between the clavicle and superior margin of the fourth rib—the sound ought to be very clear, and typically pulmonary; less clear and shorter, but of higher pitch, on the right than on the left side.

On the right side, from the fourth rib downwards to the sixth or eighth (where the liver is reached), the sound on strong percussion is less clear, till it abruptly becomes flat.

On the left side, from the fourth to the sixth rib, and from the nipple to the sternum, the sound is dull, from the presence of the heart; except in cases where the lung overlaps the heart in inspiration; where the heart is small, and where emphysema of the lungs exists.

Below these points, on the left side, the sounds of the chest anteriorly are variously modified by the encroachment of the left lobe of the liver, or by the spleen, or by the distension of the stomach, over which the sound may be tympanitic, from gaseous distension; and dull on the inner and outer parts of the region over the more solid organs just named.

Laterally, when the arms are raised, the axillary regions on both sides are highly sonorous.

On the right side, the right infra-axillary region is clear to near the sixth or eighth rib, when it becomes suddenly dull over the liver, the line of hepatic dulness varying during inspiration and expiration.

On the left side, the sound may be more or less dull, from enlargement of the spleen; but when the stomach is distended with gas, it may be tympanitic.

Posteriorly, the scapular regions give a clear sound, though much less clear than the corresponding anterior and lateral regions.

The *infra-spinous* region is more sonorous than the supra-spinous.

The *infra-scapular region* (especially over a portion of the bend of the ribs, uncovered by muscle when the arm is extended), gives a very clear percussion sound.

On the right side BEHIND, the line of hepatic dulness is at or near the eleventh rib; but strong percussion will elicit a flat sound an inch or more higher up.

On the left side BEHIND, there may be tympanitic resonance from gas in the stomach, about the seventh rib; and, about the ninth rib, dulness from the spleen.

In the inter-scapular region, clearness and duration of sound are lessened from the increased resistance of the mass of muscles.

V. **By Auscultation**, which implies "the act of listening," and is termed pulmonary or cardiac according as the sounds listened to relate to the lungs or to the heart. For this purpose the ear may be applied to the surface directly, merely interposing, for several obvious reasons, a fine towel or thin piece of linen or calico between the chest of the patient and the ear of the observer. This is *immediate* auscultation. When a conducting medium—as an instrument called the *stethoscope*—is placed between the ear of the listener and the chest, through which the sounds produced within the chest walls are transmitted, the auscultation is then said to be *mediate*. *Mediate auscultation* is especially necessary when the sounds to be heard are limited to a small and definite region, as in listening to the sounds of the heart. The ear-piece of the stethoscope ought to fit the ear of the observer; or the ear-piece may be removed, so that the end of the stethoscope may be introduced into the orifice of the ear. It is important that the ear be well fitted. The extremity applied to the patient should be about an inch and a quarter in diameter.

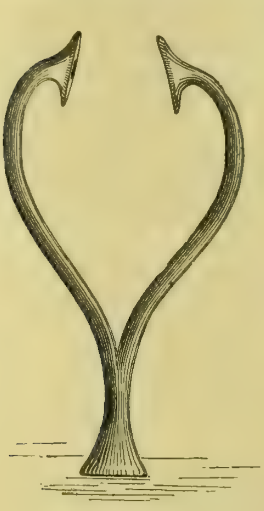

Fig. 13.*

A double or bin-aural stethoscope is also an instrument of great value in the differential diagnosis of sounds associated with lesions in the thoracic organs, especially when the sounds are obscure from their lowness. One form of the instrument seems to be an efficient magnifier of low sounds (LEARED), and another form of the instrument—that of Dr. Scott Alison—is extremely useful in cardiac diagnosis when the action of the heart is rapid or irregular (GAIRDNER). When the first sound is indistinct at the apex, or cannot be identified with the apex beat, or when the second sound is indistinct, or when it is audible only at the base, the first sound being audible only at the apex, the difficulty of recognition of the two sounds is very considerable. It is in such circumstances that the double or bin-aural stethoscope of Dr. S. Alison is of great value, for it enables the sounds to be identified at the points where they can be heard best; and it brings them into accurate relations with each other, because of the two ears receiving at the same time the sounds from two points of the *præcordial* region (GAIRDNER).

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* The Original Double Stethoscope of Dr. Leared.

Dr. Arthur Leared, of London, devised and exhibited a double stethoscope in the Great Exhibition of 1851 (Class X., No. 620), where it attracted little attention by the profession in this country. It consists (fig. 13) of two tubes, one for each ear, made of *gutta percha*, the elasticity of which, acting as a spring, keeps the tubes firmly applied to each ear. The thoracic ends of the tubes are fitted into a hollow cylinder or cup, which is applied to the surface of the chest. Dr. James E. Pollock, of London, called the attention of the profession to an American double stethoscope, in the *Lancet* of April 12, 1855, at the request of his friend Dr. Coulson, neither of whom were aware that Dr. Leared had devised and exhibited such an instrument in 1851.*

Another form of double stethoscope has been devised by Dr. Scott Alison, and named by him the "differential stethoscope." Like Dr. Leared's instrument, it has a tube for each ear; but each tube has

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* From discussions which followed in the medical journals, I have been led to make a statement in previous editions which does an injustice to Dr. Cammann; and I am glad to be able to insert this note from the American reprint of this text-book. The editor of the reprint writes as follows:—

"The writer (Dr. Clymer) is indebted to Dr. James R. Leaming, of New York, the literary executor of the late Dr. Cammann, and his clinical assistant at the time named, for the following facts:—In the spring of 1852, Dr. H. W. Browne brought one of Marsh's patent double stethoscopes to Dr. Cammann's class at the Northern Dispensary. Dr. C. at once said that the principle was not a new one, and that he had at the time one of Landouzy's poly-stethoscopes sent to him from Paris, which was on the bin-aural principle. He also observed that it was a shame for a medical man to patent an instrument intended for the profession. He then proposed to his assistants to begin a series of experiments for the purpose of perfecting a stethoscope which would render the bin-aural principle practically available. With the assistance of Drs. H. W. Browne and C. P. Tucker, and the mechanic employed, the instrument known as 'the self-adjusting bin-aural stethoscope' was in the summer of 1852 perfected, and freely given to the profession without annoying restrictions. Soon afterwards he received a letter from Dr. Marsh, threatening a prosecution for the infringement of his patent. He took this letter to his lawyer, A. P. Mann, Esq., and authorized him to answer it, denying Marsh's claim to the discovery of the bin-aural principle; and, as the two instruments were widely different in construction, refusing to make any terms or compromise, and expressed a willingness to contest the case at law if necessary. Thus not only did Dr. Cammann not patent the admirable mechanism of his stethoscope, which was clearly his own invention, but placed it freely in the hands of the profession, and stood ready to defend his right to do so. He had not been in Europe since 1830, and consequently could not have carried home Dr. Leared's idea (1851); nor is it probable he ever heard of Dr. Leared's a separate cylinder or cup, which admits of being applied to any part of the chest. Dr. Leared's instrument enables us to hear sounds emanating from any given portion of the chest with both ears simultaneously; while Dr. S. Alison's instrument enables us to listen to the sounds emanating from two different parts of the chest at the same moment, so that we may compare the sounds at any two points, or on opposite sides of the chest, by a series of consecutive observations. If two sounds exist, one at each point, and if they differ in intensity, the weaker sound is eclipsed or nullified.

All forms of stethoscope require much practical study and experience to use them with success. The ear-piece should fit the ear exactly in all forms of stethoscope; "and therefore, when a well-made stethoscope has been selected, and an ear-piece chosen which fits the ear comfortably, the student should keep to that one, and familiarize himself with its use" (FULLER).

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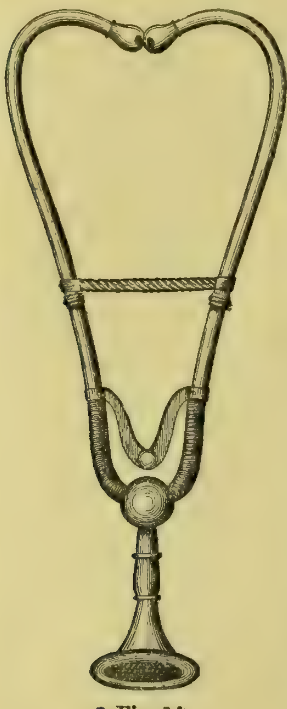
stethoscope. He never called it 'Cammann's Double Stethoscope;' this was done by the instrument maker.

"It has been said that he did not appreciate the value of his own instrument, possibly because he did not assert his claim to its originality, and from the fact that after the first year of its introduction he used it sparingly, finding that its constant application to the ear sensibly dulled the delicate acuteness of hearing so prized by the expert.

"To understand the *rationale* of this effect, it is well to premise that the double stethoscope conveys the same sound-impression through a distinct channel to each ear, just as the stereoscope presents an individual picture of the same object to each eye; and the effect in each case is to produce in the brain a clearer perception of the entirety of the sound heard or the object seen than can be done by one ear or one eye alone; but no instrument can do this perfectly unless both ears or both eyes are equally good. In ordinary sight or hearing, the eyes and ears being dual organs, one rests while the other is in action. Any one looking through the stereoscope a short time will be sensible of fatigue and temporary impairment of sight; and should it be too long and too constantly continued, the injury would probably be permanent; and the rule holds equally good in using the bin-aural stethoscope, for sight and hearing are cognate senses. It was a practical knowledge of the evil effects of a too constant application of the double stethoscope that caused Dr. Cammann to restrict its employment, believing it more important to preserve the delicate sensitiveness of the expert ear, than that the sound-impression should be made with its utmost power. It is gratifying to know that there are those in England who appreciate Dr. Cammann's unselfish labour. Dr. Alison, in his work *On the Chest*, pp. 322, 323, and 324, gives a description of this instrument, accompanied by a wood-cut, and accords to Dr. Cammann the full credit of rendering the bin-aural principle available. In describing the construction of his own instrument—the differential stethoscope—he acknowledges his indebtedness to Dr. Cammann in adopting the mechanism of his instrument, which he pronounces beautiful." (Fig. 14.)



* Fig. 14.

* Self-adjusting Bin-aural Stethoscope of Dr. Cammann.

The main object of the stethoscope is to *circumscribe* and localize the sounds that we hear; the chest end of the instrument should therefore be as small as possible, in order the better to appreciate the precise seat of the greatest intensity of sound. To ascertain this, the instrument should be "worked towards the sound," and then "from the sound," right and left, up and down, till its end is on the exact spot whence the sound proceeds in its greatest intensity. By working the stethoscope round and round, and gradually contracting the circle, the *area* of the sound can be ascertained; and hence so far the lesion producing it can be localized (HYDE SALTER).

Five circumstances affect the quality of a stethoscope:—(1.) *The material which allows the least amount of sound to be lost, and least of all perverts or modifies the sound,* is the best for a stethoscope—namely, some *porous* wood which is a good conductor of sound. Cedar and deal are the best woods for the purpose. The denser the wood the more are the sounds apt to be modified; therefore ebony is to be condemned. (2.) *The stethoscope should be of one piece of wood*—turned, in fact, out of a solid block. It ought not to be part ivory and part cedar. (3.) *As to length and thickness,* the length most convenient is that which permits the instrument to be carried in the crown of one's hat; and it may be solid if the wood is very porous; but generally it is hollow. (4.) *The chest end* should be small, not larger than *one inch and a quarter* in diameter; because the smaller it is, the greater is its localizing power, and the narrower are the limits within which the seat of any particular sound can be determined. The chest end of the stethoscope should also be narrow, and smoothly rounded over the edge. (5.) The *ear-piece* should be *large* and *flat,* to secure apposition and occlusion; but in this respect each man must fit his own ear (Hyde Salter "On the Stethoscope," *Brit. Med. Journal,* January 31, 1863). The woodcut (fig. 15) represents the section of a good stethoscope as given by Dr. Hyde Salter. It is half the "natural" diameter —*i. e.,* one-eighth the size. The ear-piece is flat and broad, and the most careless application of the ear would produce perfect occlusion; the chest end is small, with a narrow and rounded edge. The measurements are—length, seven inches; diameter of ear-piece, three; diameter of chest end, one and a quarter; circumference of shaft, one and a quarter.

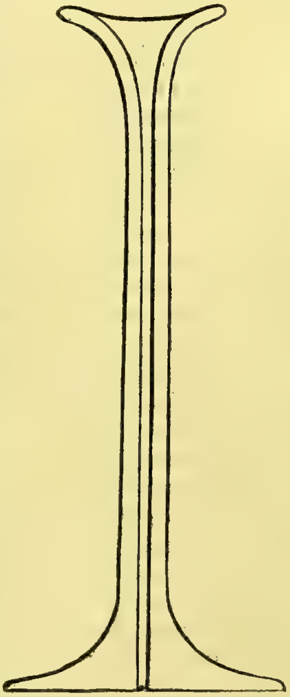


Fig. 15.*

Dr. C. L. Hogeboom, of New York, has suggested an improvement in all stethoscopes, by stretching evenly and tensely over the pectoral extremity a piece of parchment, so as to be in contact with the skin, and sufficiently firm and elastic to compress the tissues and transmit sonorous vibrations. He claims that it intensifies the sounds, and that their source is more circumscribed. In Cammann's stethoscope it lessens the roaring. To auscultation of the larynx and trachea it is peculiarly adapted, as well as to that of the heart (*New York Med. Jour.*, vol. iii., 1866).

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* Section of a good form of Stethoscope (HYDE SALTER).

But *immediate auscultation* is both simple and easy, and ought to be learned and practised by the student in every case where it is practicable; although, for reasons of delicacy, fastidiousness, or filth, on the part of the patient, it may be necessary to have recourse to the stethoscope. In many suitable cases the chest can be more quickly and quite as satisfactorily explored by *immediate* as by *mediate* auscultation.

Auscultation in children, who are apt to be frightened by the appearance of an instrument, is in general better effected by the ear directly applied to the chest. In such *immediate auscultation* the ear should be firmly and accurately applied to the surface, the pressure not being too light or too strong. Very young children must be held in the nurses' arms, so that the chest may become prominent, and the ear of the examiner be conveniently applied. The manner of breathing should be first noted, whether by the mouth or nose, abdominal or thoracic. Vocal resonance can best be estimated when the child is crying—the cry replacing the voice. The maximum intensity of respiratory murmur is, *anteriorly*, from the clavicle to below the nipple, on the right side, and not quite so far down on the left; *posteriorly*, the inferior part of the interscapular region; the minimum as in adult.

In the practice of auscultation, whether mediate or immediate, both patient and physician should be placed in positions of least restraint for both. The chest should be free of all clothing, except it may be a thin cotton towel or chemise, for the sake of cleanliness or decency. The anterior regions are best explored while the patient sits in a chair, the arms hanging loosely at the sides. To explore the posterior regions, the body should be slightly bent forward, with the arms slightly crossed. For auscultation of the lateral region, the arms ought to be raised and crossed over the head.

If the patient cannot leave the bed, he may sit up in bed, or be turned over from side to side, according to the part to be explored. The examiner must not stoop nor bend his head too much; the stethoscope being evenly and closely applied to the surface of those parts of the chest to be examined, and not with too much pressure. Special care must be taken that it is not tilted up, so that air may enter and move from below upwards; and also that a fold of dress, or any such thing, may not rub against its stem.

To become familiar with the knowledge to be acquired by means of auscultation, much time and labour must be devoted to its practice, alike on persons in health and in disease. A verbal description of the sounds to be learned is difficult, because the impressions made on the senses of one person cannot be communicated exactly by language to another; and the distinctions which subsist between the sounds heard in health and those in disease are not yet regarded as similarly significant by all; nor is their individual importance yet clearly determined in relation to practice.

In the practice of auscultation the examiner must learn first to appreciate the *natural respiratory and vocal sounds,* so that he may be able to detect such *modifications* or *changes* of them which amount to physical signs of disease.

The *healthy vesicular murmur* of the lungs is "a diffused, soft, breezy, sighing sound, not to be described, whose probable site of production is the air-sacs of the lungs" (HYDE SALTER). It is divisible into two sounds, inappreciably separated from each other—the *sound or murmur of* IN-*spiration,* and the sound or *murmur of* EX-*piration.*

The EX-*piratory murmur* in some is shorter, less intense, of lower pitch than the IN-spiration on the *left* side. In some it is said to be altogether absent on that side. The duration is increased in old people, while its intensity is less and more prolonged in the right clavicular region than in the left (GERHARD, LOUIS).

The intensity of the vesicular murmur varies in different healthy persons of the same age, sex, conformation, and apparently similar condition.

It is loud and well-marked in infancy and childhood—so loud as to be distinguished by the distinctive name of "*puerile respiration.*" It becomes more subdued in adult age; and in old age it is frequently very feeble, and known as "*senile respiration.*" Generally it is greater in the female than in the male, in the upper regions of the chest.

With regard to the degree and character of the inspiratory murmur in the several regions of the chest, the following excellent summary is given by Dr. Clymer:—

"*In the clavicular regions* it is generally stated to be of greatest intensity on the right side; but Dr. Flint says, from his own observations, that though its pitch is higher on the right side, its intensity is almost invariably greater on the left. Not unfrequently the expiratory murmur is prolonged on the right side to quite or nearly the length of the inspiratory, and its pitch on that side is sometimes higher. Dr. Flint also asserts, that on the right side the two murmurs are occasionally separated by an appreciable interval.

"*In the sterno-clavicular* portion of the infra-clavicular region it is apt to be notably modified by sounds produced in the superficial bronchial tubes and trachea.

"*In the mammary and infra-mammary* regions the inspiratory murmur is appreciable, but less intense, lower in pitch, and softer than in the clavicular regions, and the expiratory murmur is rarely heard.

"*In the supra-spinal scapular* region the respiratory murmur is less intense than anteriorly, and the inspiratory murmur is sometimes more marked on the left side, while the expiratory is more prolonged on the right.

"*In the infra-spinal scapular* region the murmur is more intense than in the upper region, but less in degree than in front.

"*In the interscapular* region, owing to the nearness of the large superficial bronchial tubes, the murmur is decidedly bronchial, like that heard over the inner third of the clavicle.

"*In the axillary* regions the inspiratory sound is very loud, especially in the upper part, and the expiratory murmur is much more frequently distinct than in the middle and inferior-anterior or posterior regions."

When the stethoscope is placed over the supra-sternal fossa, a sound like that produced by air driven forcibly through a tube of a certain calibre is heard, and may be divided into two times, one coincident with inspiration, the other with expiration, and separated by a brief interval. The quality of both sounds is peculiar and characteristic, and is said to be *tubular;* the *inspiratory* sound is of higher pitch than that of vesicular respiration, and the *expiratory* is intenser, longer, and higher in pitch than the inspiratory; this is *tracheal respiration.*

Laryngeal respiration is said by many writers to differ greatly from tracheal respiration; but, according to the observations of Dr. Flint, the difference is limited, as a general rule, to intensity; in other respects they are essentially the same (*loc. cit.*, p. 131).

Auscultation of the Voice.—It is important to attend to the sounds of the voice as they are transmitted through the chest. Modified by the size of the tubes, and the nature of the substance through which they pass, they become signs of the condition of the organs transmitting them. On this subject Dr. Clymer writes as follows:—"In vocal auscultation Cammann's stethoscope or the naked ear should be used, and the patient instructed to count *one, two, three,* slowly, distinctly, and moderately loud, and to repeat these numbers as often as is necessary. Over the trachea the voice seems concentrated and coming into the ear of the explorer: it is more or less distinct, resonant, and gives the sensation of a peculiar shock and fremitus; this is called *tracheophony.* The phenomenon of the direct entrance of articulated words into the ear at the point auscultated is called *pectoriloquy.* When the stethoscope is placed over the thyroid cartilage, and the patient is directed to speak, the voice will be found to be transmitted generally with less intensity, shock, and vibration, than from the trachea; this is named *laryngophony.* On listening over the chest, either immediately or mediately, to the sound of the voice, articulated words are not heard, and the resonance is less intense than over the trachea, is more diffused, seems farther from the ear, and usually the shock is wanting; and over certain parts of the chest, in many instances, there is no fremitus; but differences exist in different persons, and in the several regions, and in the corresponding regions of the two sides. Over the first divisions and subsequent larger subdivisions of the trachea,—the larger bronchial tubes,—as on each side of the upper part of the sternum, at and between the scapulæ, and in the axillæ, there is still considerable resonance, though diffused and distant, and frequently some fremitus; this is natural *bronchophony,* or bronchial resonance (*Physical Exploration of the Chest,* 2d ed., p. 147).

Auscultation in Disease.—The modifications of the *respiratory murmur* in diseased conditions of the lungs are changes in its *duration* and *intensity, rhythm,* and *special character;* and (with slight alterations) are thus described by Dr. Clymer:—

Its *duration* and *intensity* may be—(1.) Exaggerated or puerile; (2.) Weak or senile; (3.) Suppressed.

Its *rhythm* may be altered to—(1.) Incomplete; (2.) Jerking; (3.) Divided; (4.) Prolonged in the Expiration.

Its *character* may be—(1.) Harsh; (2.) Bronchial or blowing; (3.) Cavernous; (4.) Amphoric.

Duration and intensity may be (1.) *Exaggerated*—called also *supplementary* (ANDRAL), *puerile* (LAENNEC), and *hypervesicular respiration*,—is simply increased intensity or loudness, without change in *quality, pitch*, or *rhythm*. It has been stated that the degree of the respiratory murmur varies in different persons, and in the same regions of the two sides of the chest; and this should be remembered in estimating its value as a sign of disease.* When accompanied by a greater number of respirations than natural, decidedly heightened in the anterior and superior regions, or diffused over one side, it is indicative of disease in a portion of the lung more or less distant, or of the opposite side. It is most frequently met with in solidification of the lung, as in pneumonia, large tubercular deposits, carcinoma, pulmonary apoplexy, or where a considerable portion of lung is deprived of air from a mechanical cause, as the pressure of an enlarged bronchial gland or air-tube, blocking up of the bronchi from plastic exudation, and in pleurisy.

Duration and intensity may be (2.) *Weak*, and known as *senile respiration*, indicated by diminished intensity or feebleness of the respiratory murmur, its other characters remaining the same, happens in a number of pulmonary disorders, and is a sign of much value, often marking the site of the lesion. It may be close to the ear (*superficial*), or more or less distant (*deep-seated*). It is generally persistent, though it may be intermittent. The expiratory sound is rarely heard, except in emphysema, where, along with feebleness of the murmur, it is often distinctly prolonged. The murmur may be actually diminished in degree from imperfect production, or the effect may be caused from its reaching the ear through some solid or liquid between the lung-surface and the chest-walls. It is caused by (1.) An obstruction in the air-tubes, as in laryngeal disorders or foreign bodies in a primary bronchus, narrowing of the bronchi from inflammation and its products, spasm or permanent contraction of the air-tubes, and their compression by tumors, enlarged bronchial or lymphatic glands; (2.) Obstruction of the air-cells, in pulmonary tuberculosis, œdema, pneumonia, hemorrhage; (3.) Over-distension of the air-cells, in emphysema; (4.) Gaseous, liquid, or solid effusions into the pleural sac; (5.) Impaired thoracic movement caused by pain, as in acute pleurisy and intercostal neuralgia, or from paralysis of the respiratory muscles, as in general paresis and hemiplegia. When weak respiration is limited to the apices of the lungs, and is accompanied by diminished resonance, it generally denotes tubercles; heard in the anterior, superior, and middle regions, with increased resonance, it is a sign of emphysema; and at the base of one or both lungs, and remote, with more or less dulness on percussion, it indicates pleuritic effusion.

Duration and intensity of respiration may be (3.) *Suppressed*, as in the entire absence of the respiratory murmur. It may be permanent or intermittent; and is met with in the same pulmonary conditions as weak respiration. It is chiefly valuable as a sign of pleuritic effusion, single or double.

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* According to Walshe and Fournet, when exaggerated respiration is a sign of disease, the excess of intensity is on the side of expiration, whilst in health it is on the side of inspiration.

The rhythm of respiration may be altered by being (1.) *Incomplete*, when there is lessened duration of the inspiratory murmur, and is of two kinds; (*a.*) When its beginning is abridged (*deferred inspiration*), and it is not heard until an appreciable interval elapses after the commencement of the inspiratory act and the air-cells are fully distended, and then as a short wheeze, as in emphysema, bronchitis, and pleuritic effusions; (*b.*) It may be initially evolved, but abruptly stopped before the inspiratory act is ended, with a sort of hitch (*unfinished inspiration*); its site is in the bronchioles.

The rhythm of respiration may be altered to (2.) *Jerking, interrupted, wavy, cogged-wheel respiration* (*inspiration entrecoupée, respiration saccadée*) when the respiratory murmur, instead of being continuous, is broken and whiffy. When partial, it is the result of incomplete dilatation of the air-cells from some cause or other. Its site is almost always the apex of the lung anteriorly, and oftener on the right than the left side. It is thought by many to be a valuable sign of incipient phthisis. When general, it is due to sudden arrest in the dilatation of the chest-walls from pain or deficient innervation, as in asthma, pleurisy, pleurodynia, palsy. It is also met with in pleuritic adhesions.

The rythm of respiration may be altered by being (3.) *Divided*, when a distinct interval elapses between the inspiratory and expiratory murmurs. It is caused by over and permanent dilatation of the cells hindering the expulsion of the air, and is usually heard in the middle regions anteriorly, as in emphysema.

The rhythm may be altered by (4) *the Expiration being prolonged*, "when it is the only or chief alteration in the respiratory murmur, and there is no change in degree or pitch: it is a diagnostic sign of much significance if properly estimated. Natural prolonged expiration happens in a certain number of persons, but only on the right side. Its value as a morbid sign is measurably coincident with its site. When heard over a limited space in the upper chest regions, it may indicate tuberculosis; when more general, and heard in the middle regions, emphysema; or it may indicate only temporary obstruction or compression of the air-cells."

The character of the respiration may be (1.) *Harsh, rude, broncho-vesicular* (FLINT), *vesiculo-bronchial* (DA COSTA) *respiration*, "when it has less of the vesicular quality than the healthy respiratory murmur, being less soft, of higher pitch, more blowing, and not so equable. *In inspiration*, the vesicular and tubular qualities are united, the duration short, the pitch more elevated, and the intensity variable; *expiration*, sometimes wanting, is, when present, always prolonged, of higher pitch, and sometimes of greater intensity than inspiration, to which it succeeds after an appreciable interval (FLINT). It denotes a certain amount of pulmonary condensation, from deposition or compression, and happens in phthisis, pneumonia, pulmonary apoplexy, cancer, fibroid degeneration, melanosis, œdema, and in pleural effusions of a certain amount. For its presence, the degree of condensation of the lung should not be sufficient to abolish vesicular respiration, which must to some degree, however slight, be heard in inspiration."

The character of respiration may be (2.) *Bronchial* or *blowing,* "when it is in all respects the same as natural laryngo-tracheal respiration, and in certain pulmonary disorders takes the place of vesicular respiration. Its production and course are rapid. Inspiration is tubal, of short duration, incomplete, and of high pitch. Expiration is nearly or quite as long as inspiration, and sometimes longer, of greater intensity, and higher pitch. From the sudden interruption of the inspiratory murmur, a distinct interval occurs between it and the beginning of the expiratory. It is always associated with condensation of the lung-tissue, and is heard in phthisis, pneumonia, pleurisy with effusion, uniform dilatation of the bronchi, with induration of surrounding tissue, &c. It may be *diffused* or *tubular.*"

The character of respiration may be (3.) *Cavernous,* "when it resembles the sound produced by blowing into a hollow space; inspiration is of slow production, of low pitch, and not tubular in quality; *expiration* is of lower pitch than *inspiration.* Its common site is the superior regions. It betokens a pulmonary cavity or globular bronchial dilatation."

The character of respiration may be (4.) *Amphoric,* "when it gives the sensation of blowing into a large cavity with thick walls filled with air, and is imitated by blowing gently into a narrow-necked glass bottle. It is distinctly metallic and musical. It is caused by the air in the bronchial tubes acting on the air in the cavity. It may accompany both respiratory sounds, but is most often heard in inspiration; it is generally circumscribed or only partially diffused. It is heard in pneumothorax with pulmonary fistula, and large tubercular cavities."

"**Rhonchi.**—In pulmonary disorders we meet with certain accidental sounds, caused by more or less disturbance of the natural respiratory process, and produced either in the air-tubes, the air-sacs, or in cavities formed in the lung-tissue in the course of disease; these endo-pulmonary sounds are called *rhonchi, râles,* or *rattles,* and may be dry, moist, or of an indeterminate character. They are classed as follows by Dr. Clymer:—

(1.) Dry or Vibrating Rhonchi,	High pitch,	Sibilant.
	Low pitch,	Sonorous.
(2.) Mucous Rhonchi,	Crepitant,	Very fine.
	Moist crackling,	Less fine.
	Mucous,	Unequal, fine and coarse.
	Gurgling or cavernous,	Very coarse.
(3.) Intermediate Rhonchi,	Clicking,	First dry, then thickly Moist (gummy)."
	Crumpling,	

Exo- or peri-pulmonary sounds happen in disease of the investing membrane, and these, from their site and genesis, are called *pleural friction-sounds.* These sounds are described as—grazing, rubbing, grating, creaking, crumpling.

"**Changes of Voice.**—The modifications of the voice which occur in disorders of the lungs and pleura are valuable signs. The natural tracheal or laryngeal voice may be heard in unnatural sites, or natural bronchial resonance may be materially altered in intensity, pitch, concentration, and apparent proximity to the listening ear, or it may be diminished or suppressed. According to Dr. Flint, the *whispering voice* may also undergo changes. These unnatural changes of the voice may be ranged under the following heads" (CLYMER):—

Intensity,	Exaggerated resonance. Bronchophony. Exaggerated bronchial whisper and whispering bronchophony. Diminished or suppressed resonance.	
Character and pitch,	Pectoriloquy,	Cavernous. Amphoric.
	Cavernous whisper.	
	Ægophony.	

Heart's Sounds.—When the lungs are healthy, the intensity of these sounds is directly as the distance of the point at which they are examined from their centre of production. In some diseases of the lungs the conducting power of the media being changed—increased or lessened—the intensity of the heart's sounds will be increased or lessened (CLYMER).

With the view of affording the means for a comparative study of the auscultatory phenomena, the following tables, relative to the natural sounds in health, and the altered or morbid sounds in disease, are compiled from the writings of Walshe, Thompson, Wood, and Bennett; and it is hoped they may furnish an outline to guide the student in appreciating the morbid states of the lungs and heart.

The arrangement in a tabular form has been preferred, because it is believed the description of the sounds are more easily studied and compared with each other by this mode of arrangement than any other.

These tables are as follow:—

Table I. Thoracic sounds of *Respiration* and of the *Voice* heard in *Health*, p. 507.

Table II. Thoracic sounds of a *morbid type*, sometimes called *Rales* by French, and *Rhonchi* or *Rattles* by English and American authors, evolved during the acts of *Respiration*, pp. 508 and 509.

Table III. Thoracic sounds of a *morbid type*, evolved during the act of *articulation of the voice*, p. 510.

TABLE I.—THORACIC *SOUNDS* OF *RESPIRATION* AND OF THE *VOICE* HEARD IN HEALTH.

Sounds of Respiration.	Synonym.	Character of the Sounds.	Common Site of Production.
Vesicular.	Respiratory murmur of inspiration and expiration.	*Inspiration.*—A soft diffused murmur of a gentle *breezy* character, increased in intensity with rapidity and force of respiration, and prolonged by a full inspiration. *Expiration.*—Slightly harsher and more hollow, weaker and shorter, usually not above one-fourth the length of inspiration.	Entrance of the air and its expulsion from the air-cells and terminal portions of the bronchi —vibrations of tissue therewith connected.
Puerile.	Loud Vesicular murmur.	The respiratory murmur of children and women louder than that of adults, but with characters as above.	
Bronchial or Tubal.	Tracheal.	*Blowing* as of air passing quickly through a tube; higher in pitch than the vesicular sounds; more rapidly evolved. *Expiration* as long, or nearly so, as *inspiration;* generally a perceptible interval between *inspiration* and *expiration.*	Sites corresponding to the bifurcation of the trachea, the upper part of sternum, and between the scapulæ.
Sounds of the voice through the lungs. **Natural Bronchophony.**	Vocal resonance. Pectoral Vocal resonance.	Obscure, thrilling sound of the voice, diffused, and conveying the idea of distant origin. Articulation sometimes appreciable. Resonance more marked in males than females, and in adults than children; is only markedly present over the first bone of the sternum and in the inter-scapular regions.	The voice in articulation, passing down the trachea and bronchi, is obscured, intercepted, weakened, and diffused, by passing through the spongy pulmonary vesicular tissue to reach the surface of the chest.

TABLE II.—THORACIC *SOUNDS* OF A MORBID TYPE, SOMETIMES CALLED *RALES* BY FRENCH, AND *RHONCHI* OR *RATTLES* BY ENGLISH AUTHORS, EVOLVED DURING THE ACTS OF *RESPIRATION*.

A.—IN THE PULMONARY SUBSTANCE.

English Name of Sound (THOMSON).	Synonym.	Character of Sound conveyed by	Relation to Inspiration and Expiration.	How and Where Produced.	Diseases with which it is most usually Associated.
I. Bubbling. a. *Bubbling rhonchi or rattles.*	Mucous rhonchus or râle.	The bursting of bubbles of some size, unequal and varying in number, modified by coughing and expectoration.	Co-existing with both.	Bubbling of air through liquid (mucus, blood, or pus) in bronchial tubes of the size of a crow quill, and heard in the central or middle part of the lungs.	Bronchitis after secretion has become established.
b. *Small bubbling rhonchus or rattle.*	Subcrepitant or submucous râles or rhonchi.	The bursting of more minute bubbles, producing weaker sounds.	Co-existing with both movements, but predominating during inspiration.	Bubbling of air through more or less viscid fluid in minute bronchial tubes, as at their peripheral distribution.	Capillary bronchitis of both bases of lungs, tubercular bronchitis of apex, resolution of pneumonia.
c. *Gurgling rattles.*	Cavernous rhonchi or râles.	The bursting of bubbles obviously of large size, with a hollow gurgling sound, or a metallic sound if the bubbles be small.	Co-existing with both acts.	Caused by the bursting of bubbles in a hollow space, enclosed by more or less dense and smooth walls the more so, the more perfect the gurgling.	Excavations from tubercle or other causes, dilatations of bronchi, pus in the pleura, with a bronchial fistula.
II. **Clicking,** or	Humid crackling, humid crepitation, humid crackling rhonchus	Successive clicks, few in number, and tending to pass into the bubbling rhonchi, especially during expiration.	Co-existing with both acts, but more regular and distinct during inspiration.	Mechanism of its production obscure. It probably originates in the interior of softened tubercles, which have just commenced to communicate with the minute bronchi.	Generally in *direct* connection with tuberculous exudation which has commenced to soften.
Crackling.	Dry crackling rhonchus, dry crepitation.	A succession of three or four minute dry, short, sharp, crackling sounds, permanent in many cases when once established, and tending to pass into the clicking sound. At first it may disappear for a day or two, and again recur.	Co-existing exclusively *with inspiration.*	Mechanism of their production undetermined. Conveys the impression of being evolved at a distance from the surface, and in the great majority of cases is found in the *infra clavicular* and *supra clavicular* regions.	Generally observed on the eve of the softening process in tubercles.

III. Crepitation.					
a. *Primary.*	Crepitant râle or rhonchus.	The idea of crepitation, like that produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear (WILLIAMS). An immense number of sharp sounds, conveying the notion of minute size and dryness.	Co-existing exclusively with *inspiration*, and at first towards its close only.	Probably due to the sudden and forcible expansion of delicate tissue, altered in its physical properties by the inflammatory state, and which probably undergoes minute ruptures.	Primary idiopathic pneumonia, or the pneumonic state established round tubercles. The crepitation of early pneumonia.
b. *Secondary.*	*Rhonchus crepitans redux.*	Crepiti of a bubbling nature, slowly evolved, few in number, and unequal or dissimilar and irregular in occurrence.	Audible in expiration as well as inspiration, but pertaining specially to the latter.	Probably due to the bubbling of air through fluid contained in the minute bronchi.	Co-existing with the resolution of pneumonia.
IV. Vibration.					
a. *Sonorous rhonchus.*	Sonorous râle.	A musical sound of a vibratory, deep, or grave tone, attended with fremitus of the walls of the thorax over a variable surface. *Snoring*, *humming*, *cooing*, and *bass* notes are its varieties.	Co-exists with inspiration and expiration, but especially marked in the latter, to which it may be limited.	Arises in the larger bronchi, and suggests the idea of vibrations.	Essentially associated with bronchitis.
b. *Sibilant rhonchus.*	Sibilant râle.	A high-pitched whistling sound, of variable intensity and duration, and irregular recurrence. *Clicking*, *whistling*, and *hissing* varieties are described.	Co-existent with inspiration and expiration, especially marked in the former, but occasionally limited to either.	The influence of the passage of air on a local accumulation of viscid mucus.	Pulmonary emphysema and bronchitis.

B.—ASSOCIATED WITH THE MOTIONS OF THE PLEURA.

V. Friction.	Grazing, rubbing, grating, creaking, are varieties of this sound.	The sensation of friction by a series of abrupt jerking sounds, rhythmical with respiration, few in number and superficial in seat, limited in extent, attended with fremitus, palpable to the hand, and perceptible to the patient.	Invariably heard in inspiration, or in both respiratory acts.	The rubbing of two opposed serous surfaces together, attended by inflammation.	Pleurisy, or any cause of roughness on the surface of the pleura.

TABLE III.—THORACIC *SOUNDS* OF A MORBID TYPE EVOLVED DURING THE ACT OF *ARTICULATION* OF THE *VOICE.*

Name of the Sound.	Character of the Sound.	Physical Conditions under which it is supposed to be Produced.	Diseases with which it is commonly Associated.
I. Bronchophony.	Exaggerated resonance of the voice, unattended with articulation, diffuse or concentrated, and rarely producing any tactile sensation to the ear.	Increased density of the pulmonary tissue surrounding pervious bronchi, with enlarged calibre and hypertrophy of the substance of the bronchi. The more homogeneous the consolidation the better probably is the sound transmitted.	Hepatization of lung; dilated bronchi; pleurisy, with effusion, when hepatization co-exists.
II. Pectoriloquy, or Pectoriloquous Bronchophony.	Complete transmission of articulated words from the walls of the chest into the ear; the resonance being generally circumscribed and limited in extent.	When solid masses of lung lie between a large bronchus and the parieties; when a moderate-sized excavation exists, with smooth and dense internal surface.	Various morbid states in which such physical conditions may exist,
III. Amphoric resonance.	A ringing metallic sound reverberating through a cavity, and resembling that produced by speaking into a broad-mouthed empty pitcher.	The voice reverberating in a large cavity, communicating with a bronchus by a small aperture.	Chiefly in phthisis.
IV. Ægophony.	A sound—vibratory, tremulous, cracked, and irregular, limited in its seat, comparable to the bleating of a goat, or to the voice of the exhibitors of Punch; synchronous with the articulation of each word, or following like a feebly whispered echo from a distant source. The sound appears to flutter tremulously about the applied end of the stethoscope.	When a stratum of fluid contained in the pleura compresses the lung, the voice in the bronchial tubes is thus rendered more distinct, by the compression of the pulmonary texture, and is thrown into vibration by the layer of fluid.	A tendency to it during the early period of pleuritic effusion, equally diffused and small in amount.

SECTION IV.—RELATION OF THE PARTS OF THE HEART AND GREAT BLOOD-VESSELS TO THE WALLS OF THE THORAX.

A knowledge of the exact position of the several parts of the heart, particularly of its valves and orifices, and of their relation to fixed points on the surface of the chest, is essential to accurate diagnosis. The size of the heart, and of its several parts, may thus be relatively determined in the living subject: so also its relative position, and any amount of displacement it may have undergone, and whether or not its valves or orifices are diseased. The base of the heart, being the most fixed part, is the most convenient from which to trace the outline of the heart, and to determine what parts correspond to certain fixed points upon the surface of the chest.

The heart is situated obliquely in the cavity of the thorax, from above downwards, from before backwards, and from right to left. It lies behind the middle and lower bone of the sternum, also behind the cartilages of the third, fourth, and fifth right ribs, near the sternum, and the cartilages of the third, fourth, fifth, and sixth ribs on the left side, in front of the bodies of the sixth, seventh, and eighth dorsal vertebræ. It rests immediately above the diaphragm upon its cordiform tendon, the serous layer of the pericardium only being interposed. Owing to the obliquity of its position, the *line of the base of the heart* looks upwards and backwards towards the right shoulder. *The apex* points downwards and forwards towards the space between the cartilages of the fifth and sixth ribs on the left side, where its impulse may be felt during life. *The base of the heart* is on a line with the interval between the cartilages of the second and third ribs.

The region of the heart's superficial dulness is known as the *præcordial* region, the limits of which, as already defined, correspond to a vertical line through the centre of the sternum; and about the middle of the bone, nearly on a line with the cartilage of the fourth rib, the edge of the left lung separates from this middle line, and passes obliquely to the left side—thus exposing a small portion of the pericardium (fig. 12, p. 487), which is uncovered by lung. The exposed surface has a triangular shape; the apex above, the base below. The parts of the heart thus exposed beneath the pericardium are a part of the left ventricle, near its apex, and a portion of the apex of the right ventricle. This triangular *præcordial* space is on a plane below the nipple and the fourth rib (fig. 12 A). Its base is on a line with the cartilage of the sixth rib; its right boundary nearly a verticle line through the centre of the sternum; its left boundary is an oblique line through the cartilages of the fifth and sixth ribs on the left side. Within these limits the heart is in contact with the parietes of the chest, yielding a characteristic sound to percussion.

The sac which encloses the heart has a pyriform shape, the base below, the apex above, exactly the reverse of that of the heart. Thus, the *base of the pericardium* is on a line with the upper part of the xyphoid cartilage; *its apex* is a short distance above the origin of the large vessels, and generally on a line with the articulations of the cartilage of the second ribs with the sternum; but may extend as high as the level of the articulation of the first ribs with the sternum. The sac is wider at the centre (corresponding to the greatest transverse diameter of the heart) than it is at the base; and towards its centre it extends more towards the left side.

The line of the base of the ventricular portion of the heart is from three to three and a half inches below the clavicles (left and right respectively), and on a line with the junction of the cartilage of the third left rib and fourth right rib with the sternum.

The line of the base of the left ventricle rises as high as a line drawn across the junction of the cartilage of the *third left* rib with the sternum —*i. e.*, about three inches below the clavicle on that side.

The line of the base of the right ventricle corresponds to a line across the upper margin of the junction of the cartilage of the *fourth right* rib with the sternum—*i. e.*, about three inches and a half below the clavicle on that side.

The impulse of the apex of the organ is to be felt between the fifth and sixth left ribs, near where the body of these ribs joins the cartilage. The apex is a little below the fifth left rib, slightly to the left of its junction with its cartilage, and on a line with the articulation of the xyphoid cartilage with the sternum. The nipple in the male has been considered to be a useful guide. It is said to be upon the fourth rib, or over its upper or lower edge, a little more than an inch to the left of the junction of the rib with its cartilage. The edge of the left ventricle reaches the nipple on the left side.

The length of the ventricular portion of the heart is determined by the length of a line drawn from the middle of the sternum, between the cartilages of the third ribs, to below the fifth left rib, slightly to the left of its junction with the cartilage.

The greater part of the *right ventricle* lies behind the sternum: at its upper part it extends slightly to the right of this bone, a small portion extending under the cartilages of the fourth and fifth right ribs close to the sternum. Its apex is to the left of the sternum, a little above the apex of the heart; and a part of the right ventricle extends under the cartilages of the fourth and fifth right ribs, close to the sternum. The inferior margin of the right ventricle is nearly on a line with the junction of the xyphoid cartilage and the sternum. The anterior wall lies immediately under the sternum. It is overlapped at its upper portion by both the right and the left lung.

The *left ventricle* is covered by the left lung; and all its anterior surface is to the left of the sternum, extending from the cartilage of the third left rib to the interspace between the fifth and sixth left ribs, near where the cartilage joins the body of these ribs. It lies between the sternum and the nipple on the left side, to which its left margin reaches.

The *right auricle* lies to the right of the sternum, entirely covered by the right lung. Its appendix lies behind the cartilage of the third right rib, its tip rests against the right side of the ascending portion of the arch of the aorta, and is on a line with the pulmonary valves.

The *left auricle* is entirely covered by the left lung. Its appendix is the only portion seen when the pericardium is laid open. It lies behind the cartilage of the third left rib, close to the sternum, resting against the left side of the commencement of the pulmonary artery.

The line of the base of the auricles is on a line with the interval between the junction of the second and third ribs with the sternum, the greater portion of it being under the sternum.

Relative Position of the Orifices of the Heart.—The *right auriculo-ventricular orifice* lies behind the centre of the sternum, on a line with the lower margin of the articulation of the cartilages of the fourth rib with the sternum.

The *left auriculo-ventricular orifice* is on the same level, but on a plane posterior to the right. It lies behind the cartilage of the fourth left rib, near to or behind the sternum.

The *valves of the pulmonary artery* are on a line with the space between the cartilages of the second and third ribs to the left of the sternum, and very close to this bone. In some instances they may lie a little lower down—namely, on a line with the junction of the cartilage of the third left rib with the sternum, and immediately under it.

The *aortic valves* lie behind the sternum, on a line with the junction of the cartilages of the third rib with the sternum, and towards the left edge of this bone. When the valves of the *pulmonary artery* are situated lower down, the *semilunar valves* of the aorta will be lower also, and on a line with the interval between the insertion of the cartilages of the third and fourth ribs.

A line drawn across the inferior margin of the third ribs corresponds to the *base of the valves of the pulmonary artery*, and to the *free border of the aortic valves.*

The *right ventricle* ascending higher than the left, *the orifice of the pulmonary artery* is on a plane higher than that of the aorta; hence the *pulmonary orifice* is the highest up, as well as the most anterior, of all the orifices of the heart.

The *aortic orifice* lies behind the pulmonary orifice, but on a lower plane.

The *left auriculo-ventricular orifice* is immediately behind the aortic orifice, but on a lower plane.

The *right auriculo-ventricular orifice* is nearly on the same plane as the left, but more anterior, about three quarters of an inch lower than the pulmonary orifice.

The *ascending* portion of the arch of the aorta curves to the right of the sternum, between the cartilages of the second and third ribs. In this part of its course it is still within the pericardial sac, and (in the dead subject) lies at the depth of an inch and a half from the surface, the margin of the right lung and the pericardium being between it and the parietes of the chest.

The *transverse portion of the arch of the aorta* crosses the trachea at the centre of the first bone of the sternum on a line with the lower margin of the articulation of the cartilages of the first ribs with the sternum, and at a still greater depth from the surface.

The *arch of the aorta* approaches most closely to the parietes of the chest at the point at which the *arteria innominata* comes off—i. e., *on a line with the junction of the cartilage of the second right rib with the sternum.*

The *origin of the pulmonary artery* is on a line with the junction of the cartilages of the third ribs with the sternum. The tip of the left auricle rests against its left side. The pulmonary artery ascends about two inches before it divides; and at that point a portion of the margin of the vessel comes to the left of the sternum between the cartilages of the second and third ribs. The division of the artery is on a line with the upper edge of the cartilage of the second ribs where they join the sternum.

The *ascending vena cava* passes through the diaphragm by an opening, which corresponds to the upper part of the xyphoid cartilage.

In various morbid states the apex of the heart is formed by the *right ventricle* alone, or by the right and left together: in hypertrophy of the right side, for example, associated with *bronchitis* or with *emphysema.* When the right ventricle chiefly enters into the formation of the apex of the heart, the apex is then broad and rounded, rather than of the normal conical form, and is evidence of long-standing pulmonary obstruction (BELLINGHAM, BOUILLAUD, FULLER, PEACOCK, WALSHE, WILKS).

SECTION V.—DIMENSIONS OF THE ORIFICES OF THE HEART.

The arterial orifices progressively increase equally till the meridian of life; after which, as age advances, the aortic orifice enlarges more rapidly; so much so that in old age it sometimes is larger than the pulmonary artery orifice. Beyond fifty years this is apt to occur.

In children, up to the sixth or to the tenth year, the orifices (arterial) are both the same; after this the pulmonary orifice increases more rapidly, and becomes larger than the aortic.

The two auriculo-ventricular orifices progressively grow or enlarge at a uniform rate, till the right side acquires the start of the left at an early age.

The healthy working of the heart is consistent rather with its relative size, and the relative dimensions of the orifices to each other, compared with the size and weight of the body generally, the condition of other organs, such as the lungs, the liver, the stomach, the spleen, and the kidneys, than with the absolute size of the heart itself. In health, there is found to be a certain determinate relative magnitude of the four orifices of the heart to each other; and therefore a relative magnitude of the areas of those orifices, as shown by Dr. Herbert Davis, in *Proceedings of Royal Society,* No. 118, 1870.

Measurements of the four orifices of the heart have now been recorded with great exactness by several anatomists, to the *thousandth* part of an inch; and the mutual relationship of the *areas* of these orifices is of practical importance in connection with cardiac lesions. To Bizot in France, and to Drs. Peacock and Reid in this country, we are mainly indebted for the most careful and trustworthy measurements of the circumferences of the orifices.

The mean circumference of the four orifices of the heart, expressed in English inches, is as follows:—

CIRCUMFERENCE * MEASUREMENTS (DR. REID).

Orifices.	Male.	Female.
Tricuspid, . .	5·3	4·9
Pulmonic, . .	3·7	3·5
Mitral, . . .	4·6	4·2
Aortic, . . .	3·2	3

CIRCUMFERENCE MEASUREMENTS (DR. PEACOCK).

Orifices.	Male.	Female.
Tricuspid, . .	4.74	4·562
Pulmonic, . .	3·552	3·493
Mitral, . .	4	3·996
Aortic, . .	3·14	3·019

The mean areas * of the four orifices are thus found to be:—

AREA IN SQUARE INCHES (DR. PEACOCK).

Orifices.	Male.	Female.	
Tricuspid,	1·78	1·6	= 1¾ sq. in. nearly.
Pulmonic,	1·00	·97	= 1 ,,
Mitral,	1·27	1·27	= 1¼ ,,
Aortic,	·78	·67	= ¾ ,,

AREA IN SQUARE INCHES (DR. REID).

Orifices.	Male.	Female.
Tricuspid, .	2·24	1·9
Pulmonic, .	1·01	1·
Mitral, . .	1·7	1·4
Aortic, . .	·8	·71

Thus the *tricuspid*, having the largest area, is more than double the size of the aortic opening; and the law which Dr. Davis believes to be distinct and constant, regulating their relative magnitude in health, is found by comparing the *ratios* of the areas of the corresponding orifices, expressed as below:—

$$\frac{\text{Area of Tricuspid,}}{\text{Area of Mitral,}} = \frac{1{\cdot}78}{1{\cdot}27} = 1{\cdot}4 \text{ nearly.}$$

$$\frac{\text{Area of Pulmonic,}}{\text{Area of Aortic,}} = \frac{1}{{\cdot}78} = 1{\cdot}3 \text{ nearly.}$$

Or the area of the *tricuspid* bears nearly the same ratio to the area of the *mitral* which the ratio of the area of the *pulmonic orifice* does to that of the *aortic* orifice,—*i. e.*, if the *tricuspid orifice* were twice the size of the *mitral orifice* in area, the *pulmonic orifice* ought to be twice the size of the *aortic orifice* in area. The two *ratios* differ only by about *one-tenth*.

In man, the horse, donkey, dog, ox, calf, sheep, and pig, the same result has been found; and generally it is observed that the area of the *tricuspid orifice* is larger than the area of the *mitral orifice* by 1·3 inches nearly; and so also is the area of the *pulmonic orifice* larger than the *aortic orifice* by the same amount.

One healthy orifice being known, the area of the corresponding opening on the other side can thus be calculated; and any deviation may be determined, and amount of contraction or dilatation fairly estimated. So, also, if the areas of any three of the openings be known, the area of the fourth can be correctly estimated.

Generally speaking, the *tricuspid orifice* ought to be larger than the *mitral orifice*, and almost *half as large again* as the *aortic orifice*.

The *mitral orifice* ought to be larger than the *orifice of the pulmonary artery*, and larger than the *orifice of the aorta* by more than *one-fourth*.

The *orifice of the pulmonary artery* ought to be larger than the orifice of the *aorta* by about *one-eighth* (WILKS).

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* The area is got by multiplying the square of the circumference by ·07958.

The *mitral orifice* tends to diminish under all forms of disease which diminish the supply of blood coming from the lungs to the left side of the heart—*e. g.*, smallness of the chest and lungs compared with the size of the person, and imperfect respiratory acts. Such forms of contraction are independent of organic disease of the part, and are unlike induration, which implies increased development of tissue round the orifice which leads to its contraction. Under this latter condition ulceration may ensue, when the anterior curtain of the valve is generally partially destroyed, when large soft vegetations or concrete fibrin are deposited from the blood, and are found adhering to the diseased parts.

If the *mitral orifice* is relatively small and contracted, the left auricle may become surcharged with blood. Its walls are then apt to become thick, tough, and hypertrophied, in place of being uniformly thin. Pulmonary obstruction is associated with this condition, and enlargement of the right side is a subsequent effect.

Aortic obstruction or contraction enlarges relatively all other parts of the heart.

The orifices of the right side of the heart exceed in size those of the left, and more especially the *auriculo-ventricular orifices*, compared with the arterial orifices; but in advanced life the *aortic orifice* tends to exceed the *pulmonary*.

The researches of Bizot, Peacock, and Reid, all show that the size of all the orifices of the heart is relatively greater in the male than in the female, and that their size progressively increases as life advances in both sexes.

The following are the average dimensions of the orifices of the heart given by Bizot:—

	MALE.	FEMALE.
Right auriculo-ventricular orifice,	54·12 lines.	48·25 lines.
Left auriculo-ventricular orifice,	45·17 ,,	41·3 ,,
Pulmonary orifice,	32·2 ,,	30·7 ,,
Aortic orifice,	31·15 ,,	28·4 ,,

SECTION VI.—RELATIVE BULK AND WEIGHT OF THE LUNGS AND HEART.

BULK.—From birth to extreme age the volume of the heart is continually increasing, very obviously up to the twenty-ninth year. Such growth implies—

(1.) Progressive enlargement of its orifices.

(2.) Increasing thickness of walls of ventricles, and especially of the left.

(3.) Progressive increase of weight and bulk—the bulk varying from 12·5 to 19·8 cubic inches in normal adult hearts.

The thickness of the parietes of the heart varies also with age; those of the *left* ventricle are thicker in the male than in the female at every age, and the thickness increases as age advances. The parietes of the right side increase, but in a much less ratio.

The thickest part of the parietes of the left ventricle is at the centre, next at the base, and it is thinnest at the apex (the mean thickness is under *six* lines). The thickest part of the parietes of the right ventricle is at the base, where it is above *two* lines. The thickest part of the *septum ventriculorum* is at its centre.

There are certain diseases which tend to increase the weight of the heart, without the valves or aorta being necessarily diseased. For example, *chronic bronchitis* tends considerably to increase the weight, and so does *Bright's disease of the kidneys.* The weights (abnormal) of the heart arrange themselves in the following order, beginning with the highest:—

1. Without material disease of valves, aorta, or lungs, Dr. Peacock records a weight of 40 ounces 12 drachms.
2. With adhesion of pericardium alone.
3. With *aortic* disease the enlargement is considerable.
4. With *aortic valve* disease there is greater enlargement.
5. With mitral valve disease, enlargement is not so considerable as in either aortic disease or aortic valve disease.

WEIGHT.—The absolute weight of the lungs and of the heart increases as life advances, and is greater in the male than in the female at all ages (BIZOT, BOYD, PEACOCK).

The following are the records given by Dr. Boyd in his paper already referred to:—

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE LUNGS AND HEART, AS TO AGE AND HEIGHT.

Age and Sex.		Body-weight.		Body-height.	Weight of Right Lung.	Weight of Left Lung.	Weight of Heart.
Years.		Lbs.	Oz.	Inches.	Ounces.	Ounces.	Ounces.
1 to 2	Male,.........	14	6	28·5	4·12	3·99	1·66
	Female,......	13	2	27·7	3·58	2·95	1·47
2 to 4	Male,.........	20	0	31·6	5·45	5·08	2·14
	Female,......	18	7	31·6	5·04	4·24	2·11
4 to 7	Male,.........	25	8	37·5	6·27	6·01	2·77
	Female,......	24	9	37·0	5·52	5·45	2·3
7 to 14	Male,.........	42	6	47·0	10·14	10·38	4·25
	Female,......	38	6	45·0	8·82	8·17	4·38
14 to 20	Male,.........	68	0	60·5	20·4	19·67	7·61
	Female,......	63	14	57·7	17·52	15·08	8·46
20 to 30	Male,.........	92	14	66·75	32·34	30·09	10·96
	Female,......	86	13	62·0	21·89	16·71	9·08
30 to 40	Male,.........	98	3	66·5	28·47	24·29	11·36
	Female,......	87	0	62·0	18·74	17·64	9·45
40 to 50	Male,.........	102	0	66·8	31·21	28·63	11·53
	Female,......	84	9	62·0	19·73	17·47	9·6
50 to 60	Male,.........	102	0	66·0	30·32	26·29	11·83
	Female,......	86	0	62·0	19·48	17·08	10·44
60 to 70	Male,.........	103	13	65·7	28·52	24·16	12·94
	Female,......	86	14	61·5	20·32	16·59	10·64
70 to 80	Male,.........	106	13	65·7	18·77	17·11	13·14
	Female,......	80	4	61·0	16·76	14·59	10·10
Above 80	Male,.........	99	0	66·7	30·46	24·30	12·1
	Female,......	79	5	60·0	18·22	15·23	10·27

SECTION VII.—MODE OF EXAMINATION OF THE HEART.

In order to determine the nature, the situation, and the extent of morbid changes in the heart, it is necessary to be able to recognize readily any alteration in the heart's impulse, either as regards its strength or the situation in which it is felt—it is necessary to be able to detect any difference in the extent and degree of the heart's superficial dulness, or any change in the character of its sounds different from those which are normal.

Inspection, palpation, percussion, and auscultation are all therefore capable of affording valuable assistance in arriving at a diagnosis; and the following statements are condensed from the admirable writings of Drs. Bellingham, Fuller, Gairdner, Stokes, and Walshe.

The inspection of the external surface of the thorax, and the application of the hand to the *præcordial* region should never be omitted. Positive information is obtained by these two methods of examination, which mutually assist one another.

The exact point at which the apex of the heart comes in contact with the parietes of the chest may be determined simply by inspection. The *strength* or *feebleness* of the impulse of the heart is to be determined by the application of the hand.

By inspection it is ascertained whether the two sides of the thorax are symmetrical; and (in connection with cardiac diagnosis) whether there is any bulging in the *præcordial* region, or any unusual pulsation at any part of its parietes in the large arteries which come off from the arch of the aorta, as well as in the jugular veins or epigastric region.

By *the application of the hand*—palpation—the force or impulse of pulsation is determined, the frequency or slowness of the heart's action is judged of, and the regularity or irregularity of its movements. We may likewise appreciate by this means any *tremors* or *frictions* which accompany its action in the pericardium. To determine the impulse of the heart, the hand must be placed directly upon the surface of the chest; but *mediate* palpation may be used by placing one end of the stethoscope over the part where the impulse is, when the extent to which the instrument is elevated, and the force with which this is accomplished, will give an accurate idea of the strength of the heart's impulse, especially in hypertrophy, or in hypertrophy with dilatation of the ventricles.

In healthy persons with well-formed chests the impulse of the heart is so slight as not to be perceptible to the individual himself; and it is felt only at one spot—namely, between the cartilages of the fifth and sixth ribs on the left side—*i. e.*, from one to two inches below the nipple, and to its sternal side. When the parietes of the chest are much loaded with fat, the impulse is scarcely perceptible to the hand; while in thin persons it is evident to the eye.

The impulse is somewhat stronger in the erect than in the recumbent posture. A forced inspiration diminishes it, and causes it to be felt lower down than usual; while a deep inspiration elevates the ribs, without raising the heart in the same degree.

In a forced expiration the impulse is more perceptible, and is felt higher up. In examining the heart it is therefore necessary to make the patient vary his position, and to examine the heart both during inspiration and expiration. Calmness and tranquillity on the part of the patient must be obtained, because mental excitement, as well as exercise or exertion, increases the impulse of the heart. A diminished impulse, circumscribed or feeble, is due to feebleness of the action of the heart from disease or alteration of its muscular tissue—as in softening or fatty degeneration of its tissue, or general debility of the system; or owing to disease in the lungs or pericardium—as from effusion into the sac—when the apex of the organ may be prevented from coming in contact with the parietes of the chest. The impulse is also diminished in cases of attenuation of the walls of the ventricles, with dilatation of their cavity. Emphysematous lungs may likewise overlap the heart, and prevent its impulse from being felt. Increased impulse of the heart is generally due to some morbid state of the heart itself. It is stronger than natural in hypertrophy of the walls of the left ventricle, and is greatest in hypertrophy with dilatation of the ventricles. In such cases the impulse is slow, gradual, heaving, double, and occasionally so violent as to shake the bed on which the patient rests. This slow, progressive, heaving impulse is produced by no other cause than hypertrophy with dilatation of the ventricles of the heart; and in such cases the extent of surface over which the impulse is felt is much increased, and the whole side of the chest is sometimes elevated by the action of the organ. The double impulse which can be felt is due to the fact that the *diastole* as well as the *systole* of the ventricles is accompanied by an appreciable impulse. Adhesions of the pericardium to the pleuræ of opposite sides may so bind down an enormously large heart that its impulse will not be felt.

The situation of the impulse of the heart may be altered by displacement of the heart itself, as in cases of empyema, towards the left side when the right pleural cavity is distended with fluid, and to the right side when the left pleural cavity is distended with fluid. The impulse may then be felt on the right of the sternum. In cases of ascites the heart may be pushed upwards, and its impulse felt on a plane higher than natural; so also in cases of ovarian and other abdominal tumors, hysterical tympanitis, or in advanced stages of pregnancy. In emphysema of both lungs the heart is displaced downwards, so that the impulse is then felt sometimes as low down as the epigastric region. When fluid is effused into the pericardium, the site of the impulse is somewhat elevated; and as the amount of fluid increases, the impulse becomes weaker, unequal, undulatory, or irregular; and when the effusion is very considerable the impulse will be altogether absent.

In hypertrophy of the left ventricle, with dilatation of its cavity, the impulse is felt lower down than natural, more to the left side, and occasionally on a line vertically from the axilla.

In hypertrophy with dilatation of the right ventricle, the impulse is felt lower down, and more to the right side than natural, and not unfrequently on a line with the xyphoid cartilage.

In the former case (hypertrophy with dilatation of the *left* ventricle) the impulse is progressive, heaving, and strong, elevating the hand or stethoscope of the observer, and felt over a very much larger surface than natural; in the latter case (hypertrophy with dilatation of the *right* ventricle) the impulse is felt over a more circumscribed space, and is neither heaving, prolonged, nor very strong. But the downward displacement of the heart is not due *only* to the hypertrophy. Three causes conspire to produce it: one is the hypertrophy of the organ; another is the dyspnœal inflation of the lungs; and another is the flattening of the diaphragm, which always exists where there is an abiding source of dyspnœa. This descent and flattening of the diaphragm is due to the instinctive efforts made in all forms of dyspnœa to obtain more air. The large heart is thus pushed downwards by the dyspnœal lung inflation, and pulled downwards by the diaphragm (Hyde Salter, *Brit. Medical Journal*, February 8, 1862).

Turgescence and pulsations in the jugular veins are to be appreciated by inspection and palpation. They are signs which accompany advanced stages of some cardiac diseases. Turgescence is the most common, and occurs in cases in which an impediment exists to the free passage of the blood through the right side of the heart; and in such cases, when the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and so permits regurgitation into the auricle at each systole of the right ventricle, we have pulsation of the jugular veins as well as turgescence. Pulsation is always most evident immediately above the clavicles, and may extend half-way up the neck.

When the *mitral valve* permits regurgitation, a "purring tremor" may be felt when the hand is placed over the region of the *mitral valve;* and a similar tremor may be felt over a dilated aorta when the valves are incompetent, or when a communication exists between a vein and an artery. The jarring pulse in the radial artery, in cases of aortic valve incompetency, is an instance of the "purring tremor" felt in an artery of small calibre.

Percussion.—The whole of that portion of the anterior wall of the chest behind which the heart is situated is sometimes called the *præcordial region;* but *anatomically* it is a region more limited, corresponding to that part of the pericardium of the heart uncovered by lungs (fig. 12, *ante*, p. 487).

Both regions, however, are definable by the limits of a dull sound on percussion and more slight percussion.

The portion of heart uncovered by lung seldom exceeds *two inches* in any direction. It has a triangular shape, the base below, the apex above. It consists of a portion of the apex of the right ventricle, and of part of the left ventricle near its apex, and is on a plane below the nipple and the *fourth* rib. Its base is on a line with the cartilage of the sixth rib. Its apex is at the point where the margins of the opposite lung begin to separate from one another—*i. e.*, immediately below the *fourth* rib. The triangular boundaries of this anatomically true *præcordial space* are constituted—(1.) On the right side of the thin edge of the right lung, by nearly a vertical line through the centre of the sternum; (2.) On the left side of the thin margin of the left lung, by a more or less oblique line through the cartilages of the *fifth* and *sixth* left ribs; (3.) Below, it is bounded by the diaphragm.

Over this region, to slight percussion, a sound less dull than that yielded by the liver is elicited, and on stronger percussion a difference of sound can be detected where the thin margin of the lungs covers the heart. The mode of percussing this region, so as to mark the lung part and the heart part, is to lay one finger over the decidedly dull part, and another over the slightly resonant edge of the lung, when, by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls of the chest may be traced with accuracy, unless fat obscures the resonance (Hope).

Another means of estimating the size of the heart is by *auscultatory percussion.* This requires two competent persons to determine the result, and is managed as follows:—"A solid cedar cylinder, six inches in length and one inch in diameter, cut in the direction of the fibres, and with an ear-piece attached, is applied to the centre of the *præcordial* region, while the ear is applied to the other end: percussion is then made by another person from the point near where the cylinder is applied towards the limits of the heart in every direction. So long as percussion is made over the body of the heart, a distinct sharp shock is felt directly in the ear; but as soon as the limits of the heart are passed, this sharp shock immediately ceases, even in passing from one solid organ to another in contact with it, as from the heart to the liver" (Drs. Cammann and Clark). Practice will enable a discrimination to be made between the characteristic sound of the heart and the diffused shock produced by striking the ribs.

The mean diameter of the healthy adult heart region (not the anatomical *præcordial* region only) has been found to be as follows:—

		Inches.	Lines.
Vertical diameter,	Male,	4	0
	Female,	3	7
Transverse diameter,	Male,	4	4
	Female,	4	1
Right oblique diameter,	Male,	4	10
	Female,	4	10
Left oblique diameter,	Male,	3	10
	Female,	3	7

Generally the region of this *deep-seated dulness* extends transversely from the left nipple to a little beyond the right of the sternum, and vertically from the third to the sixth ribs. The true anatomical *præcordial space* may be diminished if the lungs are largely developed, and may disappear if the anterior margins of the lungs are emphysematous, so that their edges meet in front of the organ.

The region of the heart's superficial dulness is increased whenever the heart is enlarged, or whenever fluid to any amount is effused into the sac of the pericardium. If the walls of the ventricles are hypertrophied, or if their cavities are dilated, the dulness will extend over a wider surface, and its extent is in some degree a measure of the increased size which the organ has attained. The enlarged heart pushes aside the lungs, and a larger portion of it comes in contact with the parietes of the chest. In hypertrophy with dilatation, the heart attains the largest size that it is capable of, and the præcordial region may yield a dull sound over a square surface of from two to six inches. When hypertrophy predominates over dilatation, the space which yields a dull sound is wider from above downwards. When dilatation predominates over hypertrophy, the region in which a dull sound is yielded transversely is wider. When there is fluid in the pericardium, a larger surface than natural in the *præcordial* region yields a dull sound; the degree of dulness is more pronounced, and the sensation of resistance considerably greater than in the former case.

The situation of the dulness, its amount and degree, are the guides as to its probable cause. When it is caused by enlargement of the heart, the site of the dulness is lower down and more to the left side than when it depends upon liquid effused into the pericardium. If a large amount of fluid is contained in this sac, a dull sound may be elicited by percussion as high as the articulation of the second rib with the sternum, and even in some cases as high as the first rib. The degree of dulness over fluid is also much more marked than over the heart itself, and the resistance to the finger is greater. Solidification of lung in the immediate vicinity of the heart will cause an apparent extension of the cardiac dulness.

Auscultation and the Sounds Associated with the Action of the Heart.—Auscultation of the heart, like that of the lungs, may be either mediate or immediate; but generally there are many objections to immediate auscultation. In the case of females it is indelicate; in dirty persons it is disagreeable; while in contagious diseases it is not without risk; besides, there are some situations in which either the ear cannot be applied, or in which the stethoscope is much more convenient. In cases of valvular disease of the heart the stethoscope, and especially the forms of the double stethoscope already described, are absolutely necessary in many cases.

The phenomena of the heart's action, as observed by the eye on the heart of a living animal exposed to view, or as appreciated by the ear and hand applied over the cardiac region of man, are of the most definite nature; and in determining the alterations of sound produced by disease, it is necessary to bear in mind the occurrence and sequence of the following incidents.

The two auricles of the heart contract at the same instant of time, and the contractions of the right and left ventricles are also simultaneous. The contraction of the ventricles follows immediately that of the auricles. The relaxation of the fibres of each part of the heart follows immediately their contraction, and a short but distinctly appreciable period of complete repose of the whole intervenes between the relaxation of the ventricles and the secondary contraction of the auricles. Each complete revolution of the heart is thus accompanied by two successive sounds, separated from each other by two intervals of silence. These two sounds *are unlike;* and the two periods of silence *differ in duration.* Within the limits of health these sounds have a *variable duration,* and *limit of surface* over which they are heard, as well as of *intensity*, and of a certain *rhythm*. The *rhythm* of the heart is maintained when its two sounds succeed each other, and are followed by an interval of repose, which varies in length according to the duration of the previous systole, and according to the rapidity with which the sounds succeed each other.

The first sound is coincident with the systole of the ventricles, the impulse of the apex against the side of the chest, and the pulse of the carotids. It is sometimes also called the *systolic* or *inferior* sound of the heart. It should be listened to over the apex of the heart, and the *carotid pulse felt* at the same time.

The second sound is synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state of the carotids. It is sometimes also called the *diastolic* or *superior* sound. It should be listened to at *mid-sternum*, near its left edge, and on the level of the second interspace.

A very short silence (which only becomes obvious when the pulse does not exceed sixty in a minute) succeeds the first sound; but a distinctly appreciable period of silence and complete repose—a pause of some duration—succeeds the second sound.

The *first sound* is of a prolonged and dull character compared with the *second sound*, which is more quick, short and clear, and bears a close resemblance to that produced by *lightly tapping, near the ear, the knuckle of a bent finger with the soft extremity of a finger of the other hand* (HOPE).

The Line of Transmission of these sounds respectively, it is of the greatest practical importance to observe; because, being so constant in health, any variation indicates some modifying cause.

The *first sound* passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way. Its intensity diminishes much more on the way to, and at, the right acromial angle. The propagation backwards of the first sound is thus clearest and fullest to the left—so that, while audible at the left back, it may be inaudible at the right.

The *second sound* has the region of the base for its centre; and in nine people out of ten is heard more clearly at mid-sternum, *on the level of the second interspace*, than at any point of the præcordial region (WALSHE). It radiates towards the right and left acromial angles, and with greater clearness to the left than the right, while posteriorly it is heard at the surface with less clearness and distinctness on the right side than on the left.

Next to the lines of propagation of the sounds being determined as above indicated, it is necessary to analyze the sounds, and compare them —(1.) At both sides of the apex and region of the base; (2.) At base and apex on the same sides of the organ. (3.) At base and apex of opposite sides. In any one of these regions, compared with the other, the sounds in health are found to vary so materially in positive and relative properties that any single description of them cannot be given.

The natural sounds of the heart thus indicated may be of abnormal character, as regards *intensity, pitch, duration, quality, rhythm, reduplication*, and *apparent distance*, at each of the following points:—namely, at the third interspace, and along the third rib for the distance of three inches from the left edge of the sternum, *for all sounds;* at the point where the apex beats, *for mitral sounds;* at the left of the sternum, and over the ensiform cartilage (if the apex be not in this position), *for tricuspid sounds;* at third left costal cartilage and adjoining part of the sternum, *for both aortic and pulmonary sounds;* at second right and second left costal cartilages, *for the sounds of aortic and pulmonary orifices respectively* (*What to Observe*, p. 43).

The term "*sound*," in reference to cardiac diagnosis, is understood to refer to the natural sounds of the heart, either normal or modified in character, as detailed above.

It is important to observe the relationship of the sounds of the heart to the pulse. The first sound anticipates, by a very short but appreciable interval, the pulse at the wrist. An interval consistent with health is undetermined; but it may be stated generally, that if the diastole of the most distant arteries, such as the posterior tibial, behind the inner ankle, or the arteries on the dorsum of the foot, is so much retarded as to become synchronous with the *second sound*, the state indicates disease. It is a frequent attendant on insufficiency of the aortic valves.

When the sounds of the heart, after a certain number of perfectly regular beats, are suspended during the time usually taken to perform an entire revolution of the cardiac functions, the sounds of the heart are said to *intermit.* A sudden pause or silence then occurs, and such an intermission sometimes happens more or less regularly, or after a fixed number of regular beats. This constitutes *intermittence* or *intermission* of the heart's action. It may occur in individuals otherwise in perfect health; but it is common in diseased states of the valves or orifices of the heart, where some impediment exists to the direct passage of the blood, or where regurgitation is permitted.

Morbid Sounds of the Heart, or Murmurs.—The term "*murmur*" is applied to a sound superadded to the normal sound of the heart, and which may occur with one or more of the natural sounds. The term was first employed by the late Sir John Forbes, and has been very generally adopted since. The natural sounds may be so obscured, or even obliterated, that the *murmur* or *morbid sound* is alone heard. According to their supposed seat of production these *murmurs* are—(1.) *Endocardiac*, sometimes called *valvular;* (2.) *Pericardiac*, also called *exocardiac.*

All endocardiac or valvular murmurs yield a "*blowing*," "*rough*," "*rasping*," "*sawing*," "*booming*," or "*bellows*" *sound*, as in the whispered expressions of the words "*who*" or "*awe*," the double letter "*ss*," or the single letter "*r;*" and there are certain spots where they may be heard in their greatest *maximum force;* namely,—(1.) A few lines above the left apex; (2.) Just above the ensiform cartilage at mid sternum; (3.) On the level of the third interspace; and (4.) At the junction of the third left cartilage with the sternum.

The physical causes which may explain the *mechanism* of these murmurs are due either—(1.) To pure constrictions of natural orifices; (2.) To pure widenings of natural orifices; (3.) To pure roughness of surfaces; or (4.) To the association of the latter condition with either of the two former.

When murmurs are due to such single or combined mechanism, they are said to be *organic murmurs*, to distinguish them from *inorganic* murmurs due to certain morbid causes not yet well understood.

These *inorganic* murmurs are connected—(1.) With certain states of the blood, as in spanæmia; or (2.) With dynamic or functional action of the heart itself.

It is of the greatest importance to determine, therefore, the nature of a *murmur*, especially as to whether it is really *organic* or *functional.*

In this investigation the essential points to be inquired into, to guide the diagnosis, are as follows:—(1.) Observe the relationship of the murmur to the systole or diastole; that is, the *rhythm* of a murmur, or the position, in point of time, which it holds during the different physiological acts which constitute a complete cardiac pulsation—namely, the *contraction*, the *dilatation*, and the *period of rest* of each of the cavities; (2.) The spot of its maximum intensity on the surface of the chest; (3.) The direction in which the murmur is transmitted; (4.) Its quality and pitch; (5.) State of the natural sounds of the heart which may remain; (6.) Presence or absence of any audible phenomena in the arteries or veins, or both; and (lastly) The duration and clinical progress of the case.

Each orifice of the heart may be the seat of two murmurs, *constrictive* and *regurgitant*, *with* or *against* the current; and thus *eight murmurs* are the total number the occurrence of which is possible. The essential characters of these murmurs are condensed as follows, from Dr. Walshe's *Practical Treatise on Diseases of the Lungs and Heart;* and from Professor W. T. Gairdner's paper, entitled "A Short Account of Cardiac Murmurs," *Edin. Med. Journal,* Nov., 1861.

In determining the attributes of a cardiac murmur, the first step in the inquiry is to determine which is the *second* sound of the heart, and which is the *first,* (see p. 523). In the *rhythmical* succession of the heart's actions the phenomena which we can appreciate externally are a little later than the commencement of the heart's action. *Before there is either sound or impulse the peristaltic contraction has already taken place;* and whatever the pathological origin or seat of the murmur may be, those which immediately succeed the first sound and the impulse, correspond to the period of ventricular contraction; and those which succeed the second sound, correspond to the period of ventricular dilatation.

Dr. Gairdner gives the following classification and account of cardiac murmurs:—

1. An *auricular systolic* or (*presystolic bruit*) murmur is one which *precedes and runs up to the* FIRST SOUND of the heart, and which is in all probability produced in one or other of the auriculo-ventricular orifices, inasmuch as it coincides with the forcible emptying of the auricles into the ventricles through these orifices. Its reasonable interpretation, therefore, is *obstruction to the current of the blood* ENTERING *a ventricle.* If the left auriculo-ventricular orifice is affected, the murmur will be found to have the character of a *mitral murmur,* and to have its area at A (fig. 16). If on the contrary, the *tricuspid* orifice be obstructed, the murmur will occupy the triangular area C (fig. 16).

2. A *ventricular systolic* murmur *succeeds and runs off from the* FIRST SOUND; and it may be produced either in the *auriculo-ventricular* or in the arterial orifices. In either case it coincides with the *emptying* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is *a*

murmur of regurgitation; if, on the other hand, it is of *arterial origin,* it is *a murmur of obstruction.*

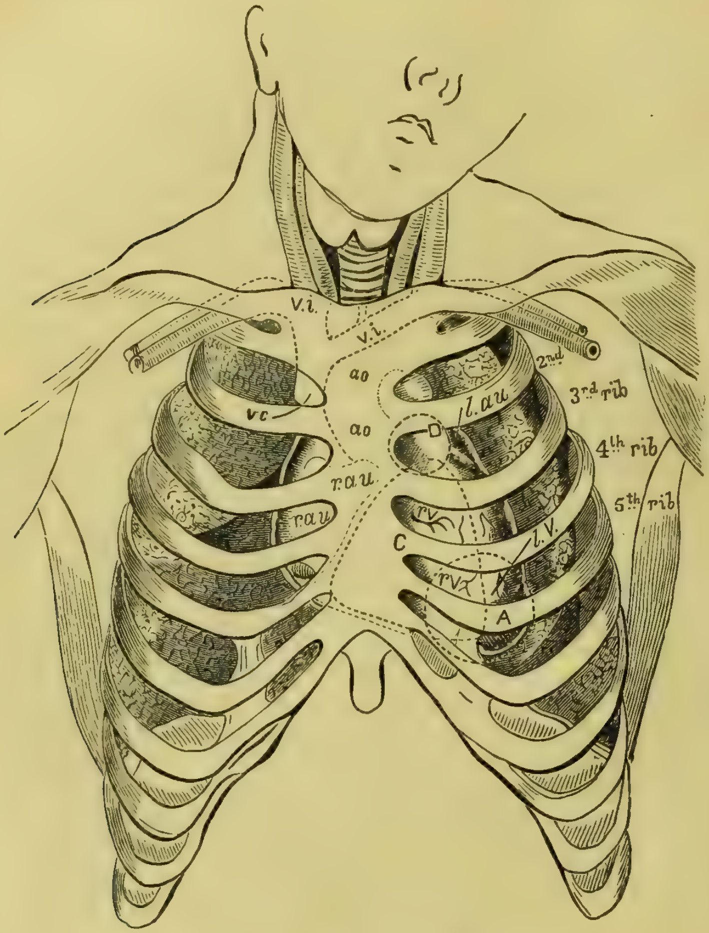

Fig. 16.*

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* The heart: its several parts and great vessels in relation to the front of the thorax. The lungs are collapsed to their normal amount, as after death, exposing the heart. The outlines of the several parts of the heart are indicated by very fine dotted lines. The area of propagation of valvular murmurs is marked out by more visible dotted lines. A, the circle of mitral murmur, corresponds to the left apex.

The broad and somewhat diffused area, roughly triangular, is the region of tricuspid murmurs, and corresponds generally with the right ventricle where it is least covered by lung. The letter C is in its centre.

The circumscribed circular area, D, over which the pulmonic arterial murmurs are commonly heard loudest. In many cases it is an inch, or even more, lower down, corresponding to the *conus arteriosus* of the right ventricle, where it touches the walls of the thorax.

The internal organs and parts of organs are indicated by letters as follows:—*r, au,* right auricle, traced in fine dotting; *ao,* arch of aorta, seen in the first intercostal space, and traced in fine dotting on the sternum; *vi,* the two innominate veins; *rv,* right ventricle; *lv,* left ventricle.

A *ventricular systolic murmur* may thus have four distinct solutions among organic valvular diseases. If the area be *mitral,* it is *a murmur of mitral regurgitation.* If the area be *aortic,* it is *a murmur of aortic obstruction.* If the area be *tricuspid,* it is *a murmur of tricuspid regurgitation.* If the area is that of the origin of the *pulmonary artery,* it is *a murmur which indicates pulmonic obstruction.*

3. A *ventricular diastolic murmur* succeeds and runs off from the second sound, and may be produced either in the *auriculo-ventricular,* or in the *arterial* orifices. In either case it coincides with the *filling* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is a murmur of obstruction; and if *arterial,* it is *a murmur of regurgitation.*

A *ventricular diastolic murmur* may thus have four distinct solutions among organic valvular diseases. If its area is *mitral,* it is *a murmur of mitral obstruction;* if its area is *aortic,* it is *a murmur of aoratic regurgitation;* if its area is *tricuspid,* it is *a murmur of tricuspid obstruction;* if its area is of the origin of the *pulmonary artery,* the *murmur denotes regurgitation from the pulmonary artery.*

The most frequent combinations of these murmurs are those which denote—

1. Combined aortic obstruction with regurgitation, indicated by ventricular systolic and ventricular diastolic murmurs.

2. Mitral contraction, indicated by *auricular systolic* murmurs or *presystolic bruit.*

3. Various combinations of the two preceding forms, the aortic and mitral valves being both diseased.

4. Mitral obstruction with dilated right ventricle, and consequently tricuspid regurgitation, indicated by *auricular systolic murmur,* heard over area A (fig. 16); and ventricular systolic murmur, heard over area C.

The rarest of all *murmurs* are those which denote *obstruction of pulmonary artery,* and those of *tricuspid obstruction.* These murmurs are still more rarely observed singly, being usually in combination with diseases causing murmur on the left side of the heart.

In a very able communication to the Hunterian Medical Society of Edinburgh, Dr. J. Warburton Begbie has recently called attention to "the diagnostic value of an accentuated cardiac second sound" (*Edin. Med. Journal,* June, 1863). This accentuated second sound—equivalent to an intensified or greatly pronounced sound—is heard in instances of aortic aneurism and aortic dilatation, associated with atheromatous degeneration, as well as in some cases of hypertrophy and dilatation of the left ventricle. In a case of aortic aneurism the second sound of the heart has been observed so intensified or accentuated over the base of the heart as at once to be recognized even by *tyros* in the art of auscultation. When this sound occurs, which is of a *booming* or *ringing* character, it is to be presumed that the aortic valves are competent. If they were insufficient, a diastolic murmur would be the result, as the prominent physical sign, and be apt to cause the most skilful physicians to overlook the existence of aneurism.

Modern diagnosis localizes murmurs chiefly from the observation of the areas of transmitted sounds already indicated. There are four distinctive areas to which murmurs arising at these orifices may be propagated.

The accompanying wood-cut (modified from those of Professors W. T. Gairdner, of Glasgow, and Luschka, of Tubingen) (fig. 16, p. 526), indicates the areas.

1. **Murmur connected with the Mitral Valve, Orifice, or neighbouring portion of the Left Ventricle,** may be the result of inefficiency of the valve, by changes in its structure, or from roughness of its edges, as by vegetations, shortening of the *chordæ tendiniæ,* or fibrinous coagula amongst them, causing obstruction. It may also result from simple roughness or deposit on the under surface of the valve without positive insufficiency. It is a *ventricular systolic murmur,* of maximum force, heard at and immediately above, or to the outside *of the left apex,* and which may completely or partially cover the first sound of the heart at the left apex, but which may also preserve its natural characters towards the base.

The pulse is generally small, weak, irregular, *intermittent,* and *unequal;* and especially is this *systolic murmur* faintly or wholly inaudible at the right apex, the mid-sternal base, the pulmonary and aortic cartilages. It is more or less clearly audible about and within the inferior angle of the left scapula, and beside the dorsal vertebræ from the sixth to the ninth. This *murmur* is rarely of high pitch; and once established it is permanent.

To find the area of this murmur it is requisite to determine the exact seat of the apex beat, the patient lying a little to the left side, or even on the face. If there is no distinct apex beat, find the most remote point downwards and leftwards at which the impulse of the heart is discernible; test this point by percussion, to observe if it corresponds with the margin of the cardiac dulness; test it also by auscultation, to hear if the *first sound* is conveyed thither with special distinctness. If a murmur concurs in position with the seat of these different phenomena, and if its seat of diffusion is *round this point nearly in a circle* (fig. 16), *it is probably of mitral origin.*

2. **Murmur associated with the Tricuspid Valve** may be due to regurgitation, or to the sharp collision of blood among thickened and roughened *chordæ tendiniæ.* It too is a *ventricular systolic murmur,* heard of maximum force *immediately above or at the ensiform* cartilage; inaudible, or nearly so, at the left apex, and very faintly, if at all perceptible, in the left vertebral groove opposite the lower angle of the scapula. It originates in the right ventricle; and when due to regurgitation, there is distension and pulsation of the auricle, vena cava, innominate and jugular veins, the distension of the latter being visible. It is generally a soft *murmur,* of low pitch, and rarely masks the systolic sound completely. It is a rare *murmur,* and often escapes detection from two causes—namely, a powerful mitral *murmur,* with which it is usually associated, or a deep-seated venous *hum.*

Professor W. T. Gairdner does not consider that *tricuspid murmurs* are rare, at least those of regurgitation. The area of tricuspid valve murmurs is over the right ventricle, where it is uncovered by lung—*i. e.,* at the lower part of the sternum, and over the whole space between this and the seat of the mitral murmur. It is usually but little audible above the level of the *third* rib, and is thus distinguished both from the pulmonic, and still more from the aortic murmur. Its area is indicated by the triangular space in fig. 16; but in cases of considerable hypertrophy and dilatation of the right side of the heart, especially in connection with emphysema (when the ventricle pulsates in the epigastrium), the murmur is heard loudest towards the xyphoid cartilage, and along the margin of the seventh left costal cartilage.

3. **Murmur connected with the Aortic Valve** habitually signifies a rough constriction of that orifice, and in rare cases has been traced to fibrinous coagula impeding the egress of the blood. It likewise is a *ventricular systolic murmur*, heard of maximum force at *mid sternum, opposite the third interspace, or upper part of the fourth rib.* It abruptly loses force between this point and the left apex, where it may be almost inaudible. Faintly perceptible at the second left cartilage, it is clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposit the second, third, and fourth dorsal vertebræ, thence rapidly losing strength downwards. It originates at the aortic orifice, and disappears about the sixth dorsal vertebra. It is propagated into the arteries of the neck. It is a high-pitched, harsh, loud, and prolonged *murmur.* The concurrence of ventricular hypertrophy increases its intensity and prolongs its duration the more contracted the orifice is. The area of this murmur corresponds generally to the regions of the sternum, and is often absolutely loudest close to the xyphoid cartilage.

4. **Murmur connected with the Orifice of the Pulmonary Artery** may indicate obstruction or simple roughness in its valves, or pressure on the vessel by adventitious masses in the pericardium. It is a *ventricular systolic murmur*, heard of maximum force at the *sternal edge of the third left cartilage*, or a little lower down, and imperceptible in the back. It is rarely met with.

5. **The Murmur indicative of Obstructive Narrowing of the Mitral Valve** is a *ventricular diastolic murmur*, heard in maximum force *immediately above and about the left apex.*

6. **The Murmur which indicates the probability of Tricuspid Narrowing or Obstruction** is also a *ventricular diastolic murmur*, and is heard in maximum force at the *ensiform cartilage.*

7. **The Murmur which indicates Regurgitation at the Aortic Orifice** is likewise *ventricular diastolic*, and is heard of maximum force at mid sternum, opposite the third interspace or fourth cartilage; and it is often carried down loudly to the left apex. It is usually of an inspired blowing character, sometimes almost hissing, rarely rough, and completely fills up the interval of repose and silence which ought to follow the second sound. It differs from *constrictive* aortic murmur in being heard with almost as much intensity *about the ensiform cartilage as opposite the third interspace.* When it covers completely the second sound of the heart at the point of its maximum intensity, the valves may be presumed to be utterly imcompetent.

8. **The Diastolic Murmur connected with insufficient Pulmonary Valves** is so rare that it is only mentioned here to complete the notice of cardiac murmurs which may be heard.

The relative frequency of intra-cardiac organic murmurs Dr. Walshe states to be in the following order, commencing with the most common —namely, *mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.* These may, however, be variously associated together; and when they co-exist, they are to be distinguished by the *rhythm*, the *pitch*, and the *character* of the aspiration.

The point at which a murmur is produced being in the majority of cases one of the four valvular orifices, all doubtful murmurs should be tested in the first instance on the supposition that they are valvular. With this object in view the most important practical points to be determined are,—(1.) The actual size and position of the heart, and the relation of its several parts to the thoracic walls (as described in the previous sections); (2.) The anatomical *præcordial* space must especially be accurately defined; (3.) The exact point of the apex beat is to be determined; (4.) The character of the impulse both of the right and left ventricle should be carefully studied; (5.) Determine by careful stethoscopic observation the exact seat and the limits of the diffusion of the murmur actually under observation.

The following short summary by Professor Harvey of Aberdeen may be of use as an aid to memory:—

"1. A *systolic* murmur denotes contraction of ventricle, with a blood-current from it either into aorta in front or into auricle behind.

"A *diastolic* murmur denotes dilatation of ventricle, with a blood-current into it either from aorta in front or from auricle behind.

"A *Presystolic* murmur is coincident with contraction of auricle, and a blood-current from it into ventricle. It is called presystolic because it just precedes the ventricular systole.

"2. In view of the *natural* direction of the blood-current, certain murmurs are sometimes called *direct*, others *reflex:* e. g., *Mitral direct* (= Mitral obstruction); *Aortic direct* (= Aortic obstruction); *Mitral reflex* (= Mitral regurgitation); and *Aortic reflex* (= Aortic regurgitation).

"3. *Composite* murmurs denote *co-existing* valve-lesions, as thus:— S + D; P + S; D + P; S + D + P. And if to these the letters *a* or *b* (apex or base) were affixed, the notation would indicate the several valves implicated. (See third paragraph following.)

"4. Murmurs vary in *length, rhythm*, or *measure.* S may run up to D; D to or beyond D*; P always runs up to S, but may begin at or near D.* These differences are due, or mainly due, to differences in the rate of the heart's action.

"5. Murmurs vary often, within short limits of time, in *character*, changing from soft or blowing to harsh or musical; or in *intensity*, changing from loud and distinct to soft and low—or to *nil*, i. e., *vanishing* for a time—audible to-day, but not to-morrow,—this hour, but not the next.

"Relative order in the tendency to this variation:—(*a.*) Presystolic mitral; (*b.*) Systolic mitral; (*c.*) Diastolic aortic; (*d.*) Systolic aortic (Prof. Sanders, *Ed. Med. Jour.*, July, 1869).

"6. Neither the *extent* nor the *gravity* of the valve-lesions is determinable by murmurs *alone.* Slight lesions may give rise to loud or harsh murmurs, and grave lesions to feeble murmurs.

"Nay, neither actual *obstruction* nor yet actual *regurgitation* (aortic or mitral) is predicable in any case from murmurs *alone.* The proof must rest on other grounds; for the valve-lesion may amount to nothing more than mere *roughening;* and an "Aortic direct" is not seldom an affair of pure anæmia."

The characters of murmurs Dr. Harvey also describes as follows:—

Systolic Murmurs.

Mitral Reflex.	*Aortic Direct.*
Loudest at, or near, or around apex. Feeble or wanting at base. Not heard above base, nor over carotids. Often diffused over left side of chest, and sometimes audible near lower angle of scapula behind. May be soft (bellows-like) or rough—sometimes musical.	Heard at and above base; perhaps most distinct over end of second right rib near sternum. Feebler over body of heart. Audible usually over carotids; rarely over left side of chest; but sometimes as high as the spine of scapula behind. May be soft or rough—sometimes musical.

Diastolic Murmurs.

Mitral Direct.	*Aortic Reflex.*
Begins just before the ventricular-systolic sound, in which it merges, often abruptly. Heard over and around apex, seldom elsewhere. Usually rough—vibrating.	Begins with and follows the ventricular-diastolic sound. Heard at base, but loudest over body of heart and along sternum. Audible, usually, over carotids, but often faintly. May be more or less loud and prolonged; usually soft, sometimes rough, occasionally musical.

The Pericardial Murmurs consist of *friction* or rubbing sounds, analogous to those already described in the pleuræ (Table, p. 509), and result from the movements of two opposed surfaces on each other, having been rendered dry or rough by change of tissue or exudation. Pericardial murmurs are almost limited to cases of inflammation of the pericardium. These friction murmurs are generally double, and are sometimes louder during the diastole than the systole of the ventricles. They appear to be superficial or near, and are seldom audible beyond the limits of the *præcordial* region. They never replace the ordinary sounds of the heart, and are entirely independent of them. Their duration is usually short, frequently ceasing entirely after having been heard for a few days, and not unfrequently changing their character and seat within the period that they are audible. A peculiar vibratory thrill, sensible to the hand laid upon the parietes, frequently accompanies them.

Section VIII.—Significance of the Pulse in Cardiac Disease.

Certain forms of cardiac disease are capable of impressing peculiar and well-marked characters upon the pulse. In health the pulse of the carotids ought to correspond with the ventricular systole, and with the first sound of the heart; and when the heart, the arterial system, and the blood are each in a normal condition, the force, the strength, the frequency, and the fulness of the radial pulse may be taken as a measure of the strength or feebleness of the systole of the left ventricle, of the rapidity with which the movements of the heart are performed, and of the amount of blood transmitted at each systole of the left ventricle. The beat of the pulse in the radial artery ought to be a little—a very little—later than the ventricular systole. The interval is almost imperceptible, unless the pulse is unusually slow. In the dorsum of the foot the interval is more easily appreciated. It is advisable to place one hand upon the præcordial region, or to auscultate the region of the heart, while the finger is on the carotid pulse, to determine these points in all cases. The pulse at the wrist cannot be trusted to in all cases for determining the exact time of the heart's beat.

In softening of the heart the pulse is sometimes much less frequent than the cardiac systole, because the impulse fails to be transmitted (FULLER, J. C. B. WILLIAMS, GAIRDNER).

In hypertrophy of the left ventricle, when the parietes are increased in thickness, the systole is strong in proportion, the blood is propelled into the aorta with increased force, and the radial pulse is strong and hard. Its velocity may not be increased, but the systole takes a longer time to be completed, and the pulse will "dwell longer under the finger." When dilatation is combined with hypertrophy, so long as the circulation continues free, the pulse will be full, or of larger volume, because the amount of blood propelled at each systole will be larger; but when dilatation is combined with attenuation, or if dilatation simply prevails, the radial pulse will have nearly opposite characters to those stated. It will be soft and weak.

The pulse in aortic regurgitation acquires a peculiar character. It is jerking and receding, though regular; while the pulsations of the arteries of the upper extremities and of the neck are visible, as if "leaping." It has been named a "locomotive" pulse by Bellingham and Todd—*i. e.*, the arterial tubes are SEEN to move by elongation—"leaping forth at each beat of the heart." This is sometimes termed the "pulse of unfilled arteries." In well-marked examples it appears as if the blood was divided into separate little balls, which pass in rapid succession under the finger. The sensation is better *seen* and felt in a large artery, such as the brachial, and two or more fingers should be laid on the line of artery. The arteries, when tortuous especially, appear like worms under the skin, wriggling into tortuous lines at each pulse (WILLIAMS). This kind of pulse is also sometimes observed in *aneurism of the ascending or transverse portion of the arch of the aorta,* as well as in cases of disease of the aorta itself, when it has become rigid, elastic, and dilated.

Intermission of the pulse indicates the slightest degree of derangement of the heart's action. It is not uncommon in persons advanced in life, in gouty subjects, and in derangement of the digestive organs with flatulence. It is also met with in cases of disease of the valves, or of the muscular tissue of the heart.

An unequal pulse is one in which some of the pulsations are strong, and others weak.

An irregular pulse is one in which a few rapid beats are succeeded by one or more slower beats, and when the interval between them is different.

An unequal and irregular pulse are much more unfavourable signs than a simply *intermittent pulse.* Both are met with in the same cases—in certain diseased states of the valves at the left side of the heart, or in morbid conditions of the muscular tissue.

In contraction of the left auriculo-ventricular orifice, the pulse, in addition to being weak and intermittent, will become small, irregular, and unequal, although the heart's action continues to be strong. "The heart may often beat so violently as to shake the patient in his bed, while the pulse is small, weak, and irregular. It appears as if there were two *pulses;* one is slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations" (ADAMS).

In mitral regurgitation, when the closure of the valve is very imperfect, the pulse becomes weak and small, and will intermit if the circulation is hurried; and when a considerable quantity of blood is permitted to regurgitate into the auricle, the pulse will also become irregular and unequal.

In contraction of the aortic orifice (when it becomes extreme only) the pulse becomes small, and intermittent or irregular, resembling the pulse of considerable constriction of the mitral orifice. When the contraction is slight, it is neither weaker nor smaller than natural, and is perfectly regular.

In degeneration of the muscular tissue the pulse in the advanced stages is small, weak, irregular, and unequal, sometimes slow, and the impulse of the systoles fails to be propelled.

In pericarditis with copious liquid effusion, the pulse presents somewhat similar characters.

During the formation of fibrinous concretions within the cavities of the heart the pulse suddenly becomes small, weak, intermittent, and irregular.

SECTION IX.—USE OF THE SPHYGMOGRAPH.

The *Sphygmograph* was originally devised by M. Marey to determine various points in the physiology of the circulation of the blood; and, as an instrument of the greatest accuracy, it could not fail to become of great value as an aid in determining the nature of diseases of the heart and arteries. The instrument and its practical use was first shown me in the autumn of 1863, by my teacher, the late John Goodsir, the distinguished Professor of Anatomy in Edinburgh, who predicted for it an extensive field of usefulness in diagnosis. Dr. Anstie (who was the first to show the instrument to a medical society in this country), and Dr. B. Foster (who was the first to publish an account of any considerable number of observations made with the instrument), and Dr. B. Sanderson, have since been the most active exponents of the methods of appreciating the characteristics of the pulse by the sphygmograph. From the writings of these men the following account of the sphygmograph is given; but more especially from those of Dr. Foster, of Birmingham, who kindly lent me his MS. notes for the last edition of this text-book, and has not less kindly revised this section, and added much valuable matter to it.

In the conventional routine of "*feeling the pulse,*" the unaided sense of touch is quite unable to distinguish many of the finer features which the sphygmograph enables us now to appreciate; and a study of its practical application cannot be dispensed with. In place of its being able to take the place of *"feeling the pulse,"* it will not only extend our practical knowledge, derived from this ancient method of observation, but it will define and greatly extend our knowledge of the more delicate characteristics of the pulse which the sense of touch fails to discover. To *"feel the pulse,"* and to *"look at the tongue,"* all patients consider as most essential duties on the part of the physician; and the sphygmograph does not aim at superseding either of these essentials. It aims at adding to our knowledge by writing down, as it were, for our inspection, the traces of hardness or softness of the pulse, thus measuring the *arterial tension*—that is to say, the greater or lesser *pressure of the blood within the blood-vessels.*

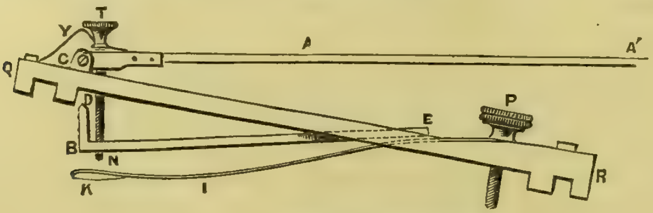

Fig. 17.*—Marey's Sphygmograph.

The value of the instrument at present mainly rests on the exactitude and precision which it gives to our notions regarding "the *practical significance of various forms of pulse*"—a kind of knowledge only acquired by great and long practical experience, and a kind of knowledge very difficult, if not impossible, to convey by a teacher to a pupil.

In the use of the instrument great care and much patience are required. The shape and development of the forearm, the position, size, and depth of the radial artery, all affect the ease of application. When the tactile spring, K (fig. 17) has been placed accurately on the radial artery, the pressure must be varied until the maximum rendering of the pulse-movements is obtained. This variation of pressure is a matter of much importance. All observers agree that a pressure *too great* will destroy the finer features of the pulse-trace, and manufacture a flat-headed curve. A pressure *too slight*, on the other hand, results in the record of a small and mean tracing, in which all the finer features are lost. Some of Marey's followers in France still dispense with any special means for adjusting the pressure on the artery, other than those furnished by the instrument in its original form, and contend that the maximum rendering of the pulse can always be obtained by careful manipulation (LORAIN).

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* The wood-cut (fig. 17) shows in the interior of the frame Q R the essential part of the instrument, which consists of a flexible steel spring, I, covered on its under surface at its free extremity with a convex plate of ivory, K. This ivory plate rests upon the artery to be examined, and, by virtue of the elasticity of the spring I, exerts a certain pressure upon it. Each pulsation of the vessel raises the spring slightly at K, and the multiplication of this movement is obtained by means of a very light lever, A, which moves upon a pivot, C. The elevation of the spring is transmitted to the lever, very near to its centre of movement, by means of a bar of metal, B E, which moves round the point E; this bar terminates in a vertical plate, B D, and is pierced by a screw, T. When the screw acts upon the spring, the connection is established between the spring and the bar, and the movements of the spring are transmitted to the bar, and through its vertical plate to the lever. In order to insure the transmission of the movement, the plate B D must be in contact with the under surface of the lever; by means of the screw T we can arrange this, and regulate the interval between the point of the plate B D and the under surface of the lever. In order that the lever should not be projected too much upwards by sudden movement, and also that it should overcome any slight friction experienced in the paper at its terminal point A, a small spring, Y, rests upon its fixed extremity, and presides over its descent. The screw, P, enables us to regulate the amount of pressure exercised upon the artery by the spring, I.

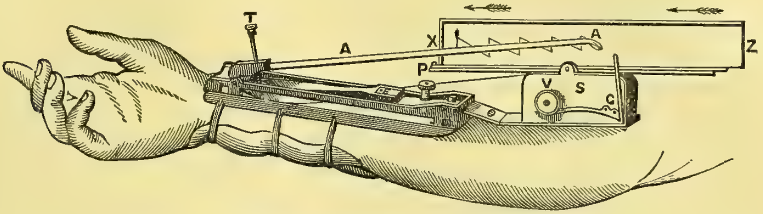

Fig. 18.*—Sphygmograph applied to the arm.

In this country several plans have been devised for measuring the pressure exerted on the pulse, viz.—(1.) By having a dial-plate described round the screw, P (figs. 17 and 18), on which an index attached to the screw would point out the amount of pressure in grammes (FOSTER); (2.) By fixing weights on the head of the screw, T (figs. 17 and 18), and so adding the extra pressure directly over the artery (FOSTER); (3.) By the addition of a screw and brass block to the carpal end of the sphygmograph, by means of which the distance between the under surface of the spring K (fig. 17) and that of the frame at D can be increased or diminished, and the pressure exerted by the spring on the artery modified in either direction (SANDERSON).† Each observer should graduate his own instrument, so as to be able to calculate the pressure exerted by the spring in any particular case. The instrument thus improved becomes a valuable though rough method of estimating the arterial tension. It is true that, as the French observers contend, the maximum rendering of the pulse can be obtained without any of these modifications; but, nevertheless, it is only by some such methods as those described that the results obtained under different conditions, at different times, and by separate observers, can be made comparable, and its full scientific value given to the record.

Another practical point of importance consists in the reduction to a

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[*] The wood-cut (fig. 18), modified from Marey, shows the instrument placed upon the arm over the radial artery in the position for use. The lever, A, is here seen to carry at its free extremity a little pen, which, filled with ink, registers its movements upon the paper which covers the plate X Z; this plate is moved at a uniform rate in the direction indicated by the arrows by means of watch-work placed beneath in the case, S. Ten seconds are occupied by the passage of the plate. The button, V, enables us to wind up the watch-work; and the small regulator, G, starts the plate, or stops its motion, as desired. The application of the instrument Dr. Foster has found much facilitated by the use of elastic bands, instead of a silk lace, as recommended by Marey. These bands embrace the arm, and are hooked on to the small projecting points on the metal framework, as seen in the diagram. The addition of a pad, suggested by Mr. Waters, to the under surface of the arm, renders the instrument more easy to the patient, and prevents any pressure from the bands.

[†] Vide *Medical Times and Gazette*, March 21, 1871.

minimum of the friction between the receiving plate and the point of the pen. Spurious tracings may be manufactured if this be not carefully attended to; and when the record is marked by ink on glazed paper, such worthless tracings are not uncommon. To obviate this difficulty, the pen at the end of the lever should be made of very flexible metal: it can then be easily adjusted. For collecting the trace, smoked glass is much more convenient than glazed paper and ink; because (1.) the friction can be reduced more effectually; (2.) the pen scarcely ever fails to leave its record; and (3.) because the tracings, when fixed (as they easily can be by photographers' varnish), are much more convenient for reproduction by photography. The plates of glass may be prepared by smoking over the flame of a paraffin lamp.

In the selection of a sphygmograph the following points must be attended to:—(1.) The instrument must be of full size; (2.) The spring must be strong; (3.) The lever long enough to amplify the pulse movements fully; (4.) The clockwork should give a rapid rather than a slow passage to the recording plate. The last point is very important as a rapid transit gives a clear development to all the features of the line of descent.

The sphygmograph, when in action, gives an exact representation of the pulse-form—the frequency of the pulsations, and their regularity. It enables us to see at a glance any pecularity in the entire series, or in any single pulsation. A trace, as below (fig. 19), is composed of a series of curves, each of which corresponds to a complete revolution of the heart, and is called a *pulsation*.

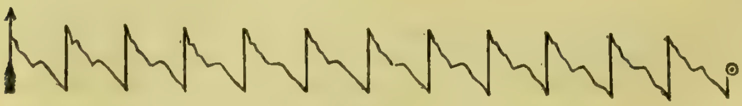

Fig. 19.

For purposes of description the pulse-curve, which corresponds with each pulsation of the radial artery, may be devided into three parts—the line of ascent, the summit, and the line of descent. It is important, however, to regard the pulse-curve as consisting of a systolic and a diastolic part, coinciding with the two periods of each cardiac revolution.

The wood-cut (fig. 20) represents—A typical radial pulse-trace, enlarged (after Dr. Foster); (*a* to *b.*) Line of ascent; (*b.*) Summit wave; (*c.*) First secondary wave; (*d.*) Great secondary wave, or true dicrotism; (*e.*) Third secondary wave, not generally seen. The notch in which *e* is placed corresponds to closure of the aortic valves. The first secondary wave, *c*, should be situated at the junction of the *upper third* with the *lower two-thirds* of the line of ascent, *a*, *b*, about the level of the dotted

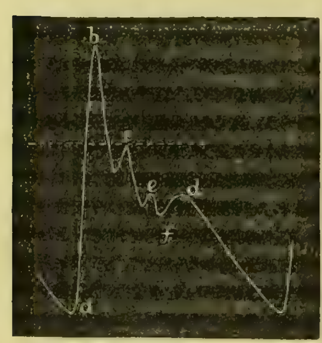

Fig. 20.

line. From *a* to *f* the ventricle is contracting or contracted, and *f* marks the closure of the aortic valves. The third wave, *e*, is seldom marked; but the three others can generally be distinguished, except when the tension is very high. The line of ascent corresponds to the ventricular systole, and marks the flow of blood into the aorta and great vessels.

The line of descent is generally broken by the occurrence of several secondary waves, which correspond to the vibrations in the blood-column, alternately to and from the heart. The summit of the trace is usually followed by a small notch, and then the trace rises again to form the first secondary wave, *c*. The relative position of this wave to the summit part is of most practical importance to observe.

The deep notch in which the *third* secondary wave, *e*, is situated, and which precedes the *second* secondary wave, *d*, is of great importance, as it marks the closure of the aortic valves. It corresponds with that reflux of blood towards the heart which forces together the lappets of the valves; and the *second* or great *secondary* wave (or dicrotism proper) corresponds to the vibrating motion towards the periphery given to the blood by the sudden flapping together of the aortic valves, and the prevention of regurgitation into the left ventricle.

From *a* to the deep notch preceding *d*, corresponds to the period of the heart's systole, and measures the duration of each cardiac contraction. From the deep notch preceding *d* to the beginning of the next line of ascent marks the period of the heart's diastole (Dr. Foster's *MS. Notes*).

From the beginning of the line of ascent, *a*, (fig. 20) to the notch, *f*, in the line of descent, is the period of the ventricular systole. The notch, *f*, separates this systolic part of the curve from the remainder of the line of descent, which corresponds to the period of the ventricular diastole. Having made this distinction between the two parts of the pulse-curve, it is now necessary to explain each of its finer features. These consist of elevations called waves, *b*, *c*, *d*, *e*, with intervening notches. The line of ascent, *a*, *b*, terminates in the first or summit wave. This line is produced by the vibration imparted to the blood-column in the arteries, by the opening of the aortic valves at the very commencement of the ventricle's contraction. When the arterial tension is low, and the ventricle acting with its usual vigour, this line is lofty and nearly vertical, and ends in a pointed summit wave. When the arterial tension is high or the ventricle weak, the vibratory element is not so well developed, and the line of ascent is consequently not so lofty nor so vertical; nor is the summit wave so sharp. The summit, *b*, wave is sometimes rounded and prolonged, but this only occurs when the first secondary wave, *c*, is blended with it. This first secondary wave, *c*, is due to the wave of distension or pressure wave, which is produced by the passage of blood into the aorta from the heart. When the arterial tension is high, this wave is proportionally more developed, and is nearer to the summit of the trace; in cases of very high tension it is blended with the summit wave, and gives the summit of the trace a rounded form. This is perceived in the pulse by the finger, in the qualities of fulness and hardness. There is another small secondary wave, *e*, which is not often seen except in pulses of low tension, it is most probably vibratory in its origin. The two notches remain—the first precedes the wave, *c*, and represents the collapse of the arterial wall after the sudden vibration in the blood column, consequent on the opening of the aortic valves. The great notch, *f*, which marks the end of the systolic part of the curve is called the aortic notch, and is due to the centripetal reflux, which effects the closure of the aortic valves. The moment these valves are closed, the blood column rebounds from them as from a spring-board, and sends towards the periphery the *great* secondary wave, *d*, or true dicrotism. When the arterial tension is high, and the aorta consequently very full, the aortic valves are closed quickly, and the reflux and the great secondary wave are faintly marked. On the other hand, when the tension is low, the reflux is more decided, and the aortic notch and the great secondary wave are more developed. After the great secondary wave or dicrotism, the line of descent falls gradually as the blood flows onwards, and by its obliquity marks the celerity of the fall of the pressure within the vessels, and indicates the facility with which the blood passes on its course. The line varies in form; sometimes it is purely oblique, at others it forms a curve convex upwards, and occasionally one or more undulations may be seen in it. These are seen in states of low tension, and are most probably vibratory in their origin. A state of low tension, with great elasticity of the arterial walls, such as we have in slight febrile conditions when the capillaries are dilated and the passage of blood easy, favours the production of all these vibratory waves. When these waves are distinctly seen, the pulse trace is sometimes called *polycrotous*, just as the normal pulse trace is called *tricrotous*, from possessing three principal waves—the summit, the first secondary, and dicrotism, or great secondary waves.

Senile change (fig. 21) in the vessels, and consequent loss of elasticity, is indicated by,—(1.) The diminution of *dicrotism;* (2.) By the great dimensions of the curve; (3.) By the closer proximity, not only of the first but of all secondary ascensions to the apex of the curve; (4.) By the great development of the first secondary wave as compared with the dicrotism. In near relation to this pulse of old age stands the radial pulse of people not far advanced in life, suffering from hypertrophy of the left ventricle; and next, that which accompanies insufficiency of the aortic valves.

The great points to be noticed in the *senile pulse* are the high positions of all the secondary waves in the line of descent, and the enormous size of the first secondary wave as compared with the second secondary wave or true *dicrotism.*

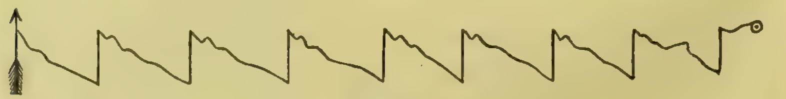

Fig. 21.*

In examining a pulse-trace, one should note, in addition to the form of each pulsation, whether the summits of all of them can be joined by a straight line, and whether the bases can be also connected by a similar line parallel to the former. In some instances this ceases to be the case, and a series of pulsations cannot be contained between such imaginary lines. The pulsations become irregular, and the line to join their summits or bases must cease to be horizontal. The line joining the summits of a series of pulsations is the line of the maxima of *arterial tension.* Its value as an indication is not absolute; it only tells us the variations that the arterial tension may undergo during the period of the observation; and it enables us to judge of the relative pressure within the vessels during any of the cardiac contractions registered. This line of greatest tension is of much value, and, with the corresponding line of least tension, should be observed in all cases; as these lines generally undergo parallel deviations, and a glance at either usually suffices to inform us of any change.

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* *Senile pulse.*—The arrow head indicates the top of the tracing.

Particular febrile diseases cannot be recognized by pulse forms peculiar to them, but the changes which the pulse-curve undergoes are intimately connected with the temperature variations. In the healthy pulse-curve the *tricrotous* form exists, but the febrile pulse ever tends to become *dicrotous,* and may become *monocrotous.* These changes are chiefly effected by the deepening of the aortic notch. When the notch has not sunk down to the level of the curve-basis (the line of least tension), and has not quite annihilated the first secondary wave, but has retarded the great secondary wave or dicrotism, the pulse is called *hypo-dicrotous* (Fig. 22). With this form of pulse the temperature of the body rarely exceeds 100° Fahr.

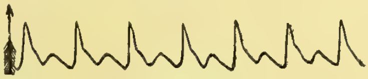

Fig. 22.*

When the aortic notch sinks to the level of the curve-basis, the first secondary wave having disappeared, or nearly so, and the dicrotism being still more retarded, the pulse is called dicrotous (Fig. 23). This form corresponds with a temperature of about 103° Fahr., and with a pulse-rate of over 100 per minute.

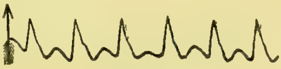

Fig. 23.†

When the aortic notch sinks below the level of the curve-basis, and the *dicrotism* appears partly blended with the line of ascent of the next pulsation, the pulse is called *hyper-dicrotous* (fig. 24), and the temperature usually ranges above 104° Fahr.

Other signs of importance also occur. A short and non-vertical ascension-line with a square or blunt summit indicates failing heart-action. The occurrence of irregularity in the pulse-curve at the height of the pyrexia is another grave sign. In its mildest form the irregularity consists in a want of exact similarity in the successive pulsations which affects the systolic portion more particularly, and tells of varying rigour of ventricular systole. When, however, there is an undulatory irregularity of the general line of the pulse trace, we have a sign of still graver import which informs us that the power of the ventricle is momentarily changing. When at an advanced stage of the fever the hyper-dicrotous pulse changes to the *monocrotous* or imperfectly *monocrotous* form (Fig. 25), it is an almost certain indication of death (Wolff).

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* *Hypo-dicrotous pulse.*—The arrow head indicates the top of the tracing.

† *Dicrotous pulse.*—The arrow head indicates the top of the tracing.

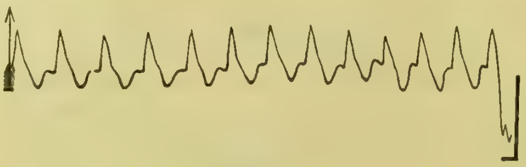

Fig. 24.*

The *frequency* of the pulse may be studied by means of the sphygmograph; for, as the plate moves at a uniform rate, and occupies exactly ten seconds in its passage, we can with ease calculate the pulse-rate. Slight variations in frequency, and irregularities that would most probably escape appreciation by the unaided touch, are by this means revealed. The frequency of the heart's action, according to the French physiologist, depends very much upon the state of the circulation in the vessels of the periphery—an easy passage of blood favouring the increased action—a difficult passage (by reason of the greater arterial tension) causing diminished frequency of the ventricular systole. The law is thus laid down by Marey that, in the majority of cases, "*the frequency of the pulse*—i. e., *the number of contractions of the heart—varies inversely to the arterial tension;*" but Onimus and Viry maintain that "*the number of contractions of the heart varies directly with the initial force.*"

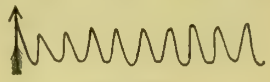

Fig. 25.†

Even if a pulse-trace fails to indicate any specific lesion, the sphygmograph is the most exact measurer of tension and index of a *hard* or *soft* pulse. When we find the pulse-trace without any well-marked notch *before* the great secondary wave, and the line of descent forming an almost unbroken oblique line, it indicates *high arterial tension*, with arteries unusually full, giving the full incompressible hard pulse. On the other hand, when the line of descent falls suddenly, the arteries are insufficiently filled, and the pulse is known as the soft compressible pulse.

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* *Hyper-dicrotous pulse.*—The arrow head indicates the top of the tracing.

† *Monocrotous pulse.*—The arrow head indicates the top of the tracing.

The *force* of the pulse is indicated by the height of the pulsations. The greater the elevation of the lever, the greater the energy of the pulse-beat; and we may say that, in many cases, the strength of the ventricular contraction is expressed by the force of the pulse. This law, however, has many exceptions; and we find that the altitude of the pulse-trace depends on several other conditions. For example:—

(1.) The volume of the artery greatly influences the amplitude of the trace. This can be well seen in traces collected from old persons. In senile changes (fig. 21) the volume of the vessels is increased considerably, and the trace betrays great fulness. Marey believes this to be due, not solely to the hypertrophy of the ventricle which exists in the old, but also to the dilatation of the artery.

(2.) The state of arterial tension modifies greatly the force of the pulse; and, as the tension is dependent on the state of the capillary circulation, it may be said that in most cases "the force of the pulse is not in relation with the energy of the ventricular systole, but that it is regulated by the state of the circulation in the ultimate ramifications of the vascular system." By means of the manometer, in a great number of experiments, this law has been proved to hold good—a feeble state of arterial tension giving to the finger and the instrument the sensation of increased amplitude. The following diagrams (figs. 26 and 27) illustrate this (Marey).

The difference in the amplitude of the traces is very distinct. In the state of feeble tension, or easy passage of the blood onwards, the lever falls quickly to the point of least tension, and is elevated considerably at each pulsation. In the case of difficult passage of the blood through the capillaries, and consequently of great arterial tension, the lever descends slowly by a line convex upwards; and, long before it has reached a minimun tension equal to that in the former case, the lever is raised slightly by the next pulsation. While the lines of the *maxima* of arterial tension are the same in both cases, the lines of the *minima* are very different. On this depends the amplitude of the pulse-trace.

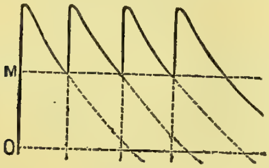

Fig. 26.*

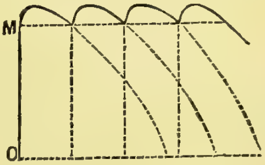

Fig. 27.†

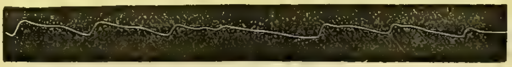

Fig. 28.

(3.) The duration of the interval which separates the pulsations has also a distinct effect on the amplitude of the trace. This is due to the fact that, during a long interval, the blood flowing continually onward lessens the pressure in the vessels, and thus favours the greater amplitude of the next pulsation. This is well seen in a trace (fig. 28) taken from a patient of Dr. Foster's in the Queen's Hospital, Birmingham.

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* The form of pulsation in a state of feeble tension.

† Under a state of strong tension.

The condition of the vessel itself, as to permeability below the point observed, influences the force of the pulsation by altering the pressure within the artery. Marey has also pointed out that in some cases, where the pulse-beat is almost imperceptible to the finger, the sphygmograph records a considerable amplitude of trace; and *vice versâ.* Such cases have been associated with a very slow distension of the vessels.

The discrimination of increased arterial resistance, as measured by the exaggeration of the systolic expansion, is the element of diseased action, whose detection and estimation, according to Dr. Sanderson, are the most important purposes to which the sphygmograph can be put. "Anatomical researches lead us to believe it probable that the earliest beginnings of what we may call degenerative disease consist in such structural alterations of the minutest arteries as, by rendering them less pervious to the circulating blood, must inevitably lead to increased arterial resistance estimable by the sphygmograph; and if by such an examination," continues Dr. Sanderson, "we can ascertain that the heart is over-taxed long before any change can be detected by auscultation or percussion, it is obvious that we have made a step forward in practical utility." Thus, he considers it likely that the *sphygmograph* is to be of greater use as an aid in forming an opinion as to the probable duration of life than in any other department of medical practice. There are many persons in whom, in the absence of any other trace of ailment, the pulse-curve indicates that the arterial resistance is excessive. The question is, "Are such persons sound?"

Drs. Anstie and Foster on the other hand, are of opinion that the diseases in which the *sphygmograph* will prove of the greatest diagnostic value are—(*a.*) In aortic regurgitation, by estimating the amount of valvular imperfection; (*b.*) In discovering unsuspected commencing cardiac hypertrophy, senile disease of arteries, or capillary disease, dependent on degenerative processes in the ultimate tissues; (*c.*) Above all, in discovering the existence of intrathoracic aneurisms, and in deciding the locality of an aneurism; (*d.*) In aiding prognosis and decisions as to treatment in the course of acute diseases. Dr. Anstie has already made a series of observations in fever, pericarditis, pneumonia, and delirium tremens, which show that in this direction the sphygmograph promises to be of great value, as the best indication of the use or otherwise of certain remedial agents.

SECTION X—GENERAL SYMPTOMS OF THORACIC DISEASE.

The elucidation of some of the topics, of which a short outline has been given in the previous sections, claims for the name of Laennec an immortal fame. He discovered how, *by means of auscultation,* disease might be detected. He not only accurately described the sounds heard in diseased states, and compared them with the sounds in health, but by morbid anatomy, in connection with careful clinical observation, he traced the mechanism of those sounds to anatomical and physical conditions of the organs with whose functions they were connected. The immediate effect of this inestimable discovery was to divert the attention of the physician from the study and observation of those vital symptoms and general states of the constitution so pregnant with information when correctly observed and properly appreciated. It was the close observation of these vital symptoms, watching the order and the periods of their manifestations, and the modes in which they were combined, that distinguished the successful practice of our forefathers in medicine—such men as Cullen and the two Gregorys of former days. Disastrous results of treatment were the consequence of the circumscribed study of so-called physical diagnosis; "but what was lost in lives was gained in pathology." The physician of the present day now knows better. He is a close observer of symptoms—*subjective* and *objective*—of constitutional states, and of physical signs; and with all the delicate instrumental appliances to appreciate those signs, he forms his judgment from the combined evidence of them all.

The general symptoms which express derangement of the pulmonary organs and their functions are *dyspnœa, cough, expectoration, tenderness,* and *pain.* The general symptoms which indicate derangement of the cardiac apparatus are *palpitations, sinking,* and *fainting,* combined sometimes also with *dyspnœa, cough, pain,* and *tenderness.*

The sensation of *dyspnœa* is brought about by an embarrassed or laborious breathing, amounting in severe cases to a sense of suffocation, expressed by the common English phrase, "*want of breath;*" or by the meaning conveyed by the Latin term "*anxietas,*" when the dyspnœa is at the point of greatest intensity. It is aggravated by exertion, some positions of the body, and a full stomach. The act of speaking is frequently arrested "*to fetch a breath;*" and the patient who suffers from dyspnœa cannot hold the breath, or refrain from the attempt to inspire, as a person in health can. The dyspnœa may occur in paroxysms, and the acts of respiration may be painful. The difference between the dyspnœa of asthma and that of cardiac disease has been already given.

The number of respirations performed in a given time is greatly increased, and often unequally so, when the paroxysm is aggravated. In health, from eighteen to twenty acts of respiration are unconsciously completed in a minute, according as a person is lying, sitting, standing, or walking; and the ratio of the acts of respiration to the pulse varies in the proportion of one to four (WATSON), or one to nearly six (WILKS); that is, about one complete act of respiration for every *four* or *six* beats of the heart. But so intimately are the functions of the heart and lungs dependent upon each other, that any deviation from these proportions, in the acts of the one or other set of organs, immediately influences the actions of the other.

Cough, expectoration, and the nature of the *sputa,* furnish valuable indications of thoracic disease.

Microscopical Elements of Sputa consist of—(1.) Young epithelium-cells—*i. e.*, of mucous corpuscles; (2.) Of mature epithelium in the form of pavement, cylindrical or ciliated bodies; (3.) Cells containing granules, or bodies like cells made up of granules; (4.) Pus-cells; (5.) Coloured corpuscles of the blood; (6.) Fibrin, either in the form of flake-like membranes or in the form of casts of the smaller bronchi and pulmonary air-cells, as in the expectoration of pneumonia. They are sometimes seen as dichotomous cylinders with rounded enlargements, composed of fine filaments, generally covered with granules. They may be met with from the third to the seventh day in pneumonia. (7.) Fat occurs in granules or globules; (8.) Tubercle matter, earthy, calcareous, amorphous, and crystalline particles also occur; (9.) Substances derived from the food; (10.) Carbon and true pigment, free or contained in cells; (11.) Fragments of pulmonary tissue.

Chemical Characters of Sputa.—Much attention has not yet been given to the chemical characters of the sputa. Professor Laycock, of Edinburgh, had a chemical analysis made of very fetid expectoration in bronchitis, which demonstrated the existence in it of *butyric* and *acetic* acids. The odour was characteristic of the *butyrates of ethyl*, resembling the smell of may-flower or apple blossoms, combined with an odour of fæces. Chemical investigation may thus demonstrate the cause of the excessive fetor in those cases which resemble gangrene of the lungs, if the smell alone is considered (*Med. Times and Gazette*, May, 1857, p. 480). A much more extended analysis has been recently made by Bamberger into the chemistry of the sputa—(1.) In chronic bronchial catarrh; (2.) In bronchial dilatation; (3.) In chronic pulmonary tuberculosis; (4.) In the infiltration of acute tuberculosis; and (5.) In pneumonia. In sputa which is chiefly catarrhal the salts vary but little—the organic matters vary considerably. The insoluble salts form about 4 to 5·5 *per cent.* of the whole saline contents; the chief amount consists of *chloride of sodium* and *phosphate of potash.* Puriform matter predominating, causes the sputa to contain a greater quantity of organic and inorganic substances; there is considerably more *phosphoric acid* in the ash, considerably less *chlorine*, and less *sulphuric acid.* The ash of pneumonia sputa differs from that of catarrhal in several respects. The alkalies, combined with phosphoric acid (which amount to 10 and 14 *per cent.* of the saline constituents of catarrhal sputa), are almost entirely absent in pneumonia during the inflammatory period, but the sulphuric acid is remarkably increased. The quantity of chlorine (37 *per cent.*) is nearly the same as the average in the catarrhal (36 *per cent.*); and there is not much variation in the insoluble salts, except in the phosphate of iron derived from the blood. In the period of resolution the sputa of pneumonia become more similar to the catarrhal; the *phosphoric acid* increases, the *sulphuric* diminishes, and the chlorine reaches a very high amount, while the potash and soda are present in the same relative proportion as in the catarrhal, whereas during the inflammatory period this was inverted. Sugar has been detected during the height of the inflammation; and Dr. Beale has shown that an excess of the chloride of sodium is constantly present in pneumonic sputa. In the sputa of bronchiëktasis, sulphureted hydrogen, acetic, butyric, and probably formic acids were detected (*New Syden. Society Year-Book*, 1860, p. 128; Schmidt's *Jahrb*, band 114, p. 3).

Sputa Typical of Pneumonia is characterized by its viscidity, semi-transparency, and tenacity, adhering strongly to the vessel containing it. So tenacious is it, that the vessel may be turned upside down without the sputa becoming detached from the sides. This rusty-coloured sputa consists of mucus intimately mixed with blood—not streaked with it, as in bronchitis, but thoroughly mixed and amalgamated with it—so that it acquires a yellowish, or reddish-yellow, or even a red colour, according to the quantity of the blood. If the disease be not very intense, the expectoration never attains the degree of viscidity or the depth of colour above referred to; but though still tenacious and adherent to the sides of the vessel, moves from one part to another as the vessel is tilted. If the disease progresses to a favourable termination, the sputa become more abundant, less adhesive, and less highly coloured, passing through the various shades of orange, until at length they become greenish or whitish, and resemble the expectoration of ordinary catarrh. If the disease be hastening to a fatal termination, the expectoration becomes scanty, less tenacious, and of a darker or dullish-brown hue, resembling the juice of prunes. If the type of inflammation be typhoid, or adynamic, or connected with tubercles in the lungs, the mucus may be tinged, or even streaked with blood; or it may consist throughout of nearly colourless, stringy, and more or less frothy mucus (Fuller *On Diseases of the Chest*).

Sputa Typical of Gangrene of the Lung, at first of a muco-purulent character, sometimes tinged with blood, begins to emit a very disagreeable odour; and as soon as a free communication is established between the air-passages and the sloughing tissue of the lung, they not only acquire an intensely fetid gangrenous odour, but assume an appearance more or less characteristic of the disease. They lose their muco-purulent character, and become extremely liquid or sero-purulent, and of a dirty greenish or ash-grey colour. At the same time the breath acquires an offensive putrid odour, the pulse feeble and rapid, with evidence of great and increasing prostration, (FULLER, l. c.)

Sputa Typical of Acute Bronchitis appear, after a few days, as a thin, saltish, frothy mucus, sometimes streaked with blood. They increase in quantity, and soon become glairy, semi-transparent, and of a faintly yellowish colour. Subsequently they assume a greyish or greenish-yellow tint, and become opaque and viscid. If the attack is severe, they become muco-purulent, and in some instances may even lose their glairiness, presenting the character of thoroughly opaque mummillated sputa.

In *chronic bronchitis* the sputa may be of the following characters:—either (1.) The expectoration of a greyish, or greenish, or yellowish-white muco-purulent matter; or (2.) The expectoration being difficult, the sputa are comparatively scanty, consisting of stringy, tenacious mucus, of a greyish or yellowish-white colour, occasionally streaked with blood; at another time, expectoration being easy, the sputa are more copious, muco-purulent in character, of a yellowish-green colour, having a faint unpleasant odour; at another time the sputa are profuse, almost wholly purulent, of a nauseous and sometimes a fetid odour, usually running together into one mass, but often remaining separated, and forming distinct mummillated masses; or (3.) There is a profuse expectoration, sometimes to the extent of half a pint in an hour, of a thin, watery, ropy fluid, which varies in opacity, but is usually somewhat transparent, resembling gum-water (FULLER).

Sputa Typical of Plastic Bronchitis consist of ordinary bronchitic sputa, or blood-tinged mucus, with fragments of white fibrinous matter, or white fibrinous casts of the bronchi, which are ejected during violent paroxysms of cough. These concrete masses vary from mere fragments to large pieces of from *one* to *four* inches in length, and may be either tubular or solid, their ejection being preceded and often accompanied by spitting of fluid blood. These casts consist of concentric laminæ, found at different periods in successive layers, and consist of amorphous granular matter intermixed with mucus-corpuscles, compound granular cells, oil-globules, and ovoid cells containing dark colouring matter, such as exists in ordinary bronchial mucus (FULLER).

Sputa Typical of Acute Phthisis consist—(1.) Of frothy mucus, often speckled with blood; and when the tubercle softens, the sputa become muco-purulent or purulent; or (2.) The sputa may be scanty, consisting of little more than frothy mucus; (3.) Expectoration, at first scanty, thin, colourless, and transparent, somewhat resembling saliva or gum-water, of a greyish colour, and more or less frothy. After a time the thin colourless sputa lose some of their transparency, and are seen to contain specs of opaque matter, which gradually subside and form a deposit resembling the sediment in barley-water; or they remain suspended by the more ropy part of the secretion, and float in the transparent mucous fluid in the form of striæ. Gradually becoming less aërated, they become more glairy and more tenacious, lose their pearly-grey colour, and are seen to be mixed with specks or streaks of an opaque white or buff colour, and not unfrequently with specs or streaks of blood. (4.) As the malady progresses, the characters of the sputa change again. They become opaque, of a whitish or yellowish hue, and are coughed up in more distinct and homogeneous masses. Sometimes they form rugged pellets of a yellowish-white colour, resembling boiled rice, which sink or partially float in a colourless, semi-transparent, ropy, non-aërated mucous fluid; or, accompanied by little mucous fluid, the sputa form large masses of a buff or yellowish-green colour, floculent in appearance, but perfectly smooth in outline, which do not coalesce, but remain distinct and separate from each other if expectorated into a vessel of water.

All these forms of sputa occasionally occur in *chronic bronchitis* as well as in *phthisis* (FULLER).

Cough.—The severity, the frequency, or paroxysmal nature of the cough must be ascertained; also the circumstances which excite it most; and whether it is attended with pain, or followed by expectoration or vomiting. The ease or difficulty of the expectoration must be noted, and whether it is accompanied or not by pain. The quantity of the sputa ought to be measured in the day and night, the form of the masses spat up, their transparency or opacity, colour and viscidity, tenacity or adhesive property. The special characters of the sputa ought in every case to be closely observed, noting particularly its thin, serous, or frothy character; whether it contains any membranous or concrete exudation-masses or blood; and it should be examined microscopically.

Pain.—The exact locality of pain in the chest should be ascertained, its severity, and the direction it tends to take. What particular circumstances aggravate it, and the effects of breathing, coughing, pressure, and postures should be ascertained.

Palpitation.—When *palpitation* occurs, its severity ought to be estimated by laying the hand over the region of the heart of the patient. It is desirable to ascertain its constancy; the circumstances which aggravate its existence or produce it—such as the influence of exertion going up a hill or up stairs, and the influence of mental emotion (*What to Observe*, pp. 39-44).

In the following table the more prominent characters of the palpitation which depends upon organic disease of the heart are contrasted with those of palpitation arising independent of disease of this organ (Bellingham, *op. cit.*, p. 172):—

PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.	PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.
1. More common in the male than the female.	1. More common in the female than the male.
2. Palpitation usually comes on slowly and gradually.	2. Palpitation usually sets in suddenly.
3. Palpitation constant, though more marked at one period than at another.	3. Palpitation not constant, having perfect intermissions.
4. Impulse usually stronger than natural; sometimes remarkably increased, heaving, and prolonged; at others irregular and unequal.	4. Impulse neither heaving nor prolonged; often abrupt, knocking, and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.
5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.	5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion, not increased.
6. Palpitation often accompanied by the auscultatory signs of diseased valves.	6. Auscultatory signs of diseased valves absent; bruit de soufflet often present in the large arteries, and a continuous murmur in the veins.
7. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.	7. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.
8. Palpitation often not much complained of by the patient, occasionally attended by severe pain, extending to the left shoulder and arm.	8. Palpitation often much complained of by the patient; readily induced by mental emotion; and frequently accompanied by pain in the left side.
9. Lips and cheeks often livid; countenance congested; anasarca of lower extremities common.	9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.
10. Palpitation increased by exercise, by stimulants, and tonics, &c.; relieved by rest, and frequently, also, by local or general bleeding, and an antiphlogistic regimen.	10. Palpitation increased by sedentary occupations; by local and general bleeding, &c.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

Expression of the Countenance in Thoracic Disease.—The countenance is often expressive of heart disease. In acute inflammatory affections of the lining or investing membrane, it acquires an anxious and depressed character. An elevation and depression of the *alæ nasi* or *nares* are commonly observed with the respiratory acts; and the occurrence, in children, of these phenomena indicates a greater amount of disease than the general symptoms would lead us to suspect. In chronic cases, when the circulation is impeded, the expression of the countenance becomes almost pathognomonic. The venous system becomes congested, the face becomes bloated and dusky, the eyelids puffed, the eyes staring, the conjunctivæ suffused, the lips and cheeks purple, respiration laborious, the air-passages loaded with mucus, and the jugular veins distended or pulsatile.

SECTION XI.—DISEASES OF THE HEART AND ITS MEMBRANES.

There are few complaints which more surely tend to shorten life, and none give rise to greater suffering and discomfort than diseases of the heart, pericardium, and great vessels. Alike in the young and in the old, they are the chief causes of sudden death, and if not suddenly fatal, "they lay their own hard conditions on the continuance of a man's life, and almost settle beforehand the manner of his death" (SIR THOMAS WATSON). These diseases are to be described as lesions of (*a.*) The *pericardium;* (*b.*) The *endocardium;* (*c.*) The *muscular structure* of the heart; and (*d.*) The blood-vessels.

(a.) *Diseases of the Pericardium.*

PERICARDITIS.

LATIN EQ., *Pericarditis;* FRENCH EQ., *Péricardite;* GERMAN EQ., *Pericarditis*—Syn., *Entzüngdung des Herzbeutels;* ITALIAN EQ., *Pericarditide.*

Definition.—*An inflammation of the fibro-serous membrane containing the heart, and investing it on its external aspect.*

Pathology and Morbid Anatomy.—The normal pericardium is a *membranous bag* containing the heart, fixed at the base to the upper part of the diaphragm. The apex of the bag surrounds the great vessels. It consists of two layers:—

(1.) *A fibrous layer,* of dense, thick, unyielding fibres, interlacing in all directions, is composed of connective tissue, including white and yellow fibres; which are firmly attached at the base of the pericardium to the central aponeurosis of the diaphragm. At the apex they are continuous with the fibrous coat of the large vessels, over which they are continued in the form of tubular prolongations, till their fibres are gradually lost or amalgamated with those of the artery.

(2.) *The serous layer* lines the outer aspect of the fibrous layer, as a *parietal layer,* and is reflected over the heart, as the *visceral layer* of the pericardium. It is reflected upon the arch of the aorta, about two and a half inches from the base of the heart. It is covered by a simple layer of pavement epithelium cells, which rests on the sub-epithelial connective tissue, which is always more compact immediately beneath the epithelium than in the parts farther removed from it.

In the young subject the fibrous layer is generally thin and transparent; in adult age and advanced life it is thicker and more opaque. Injection shows it to be supplied with minute arteries and veins.

Constant motion of the heart upon the serous aspect of the pericardium modifies the effects of inflammation and other lesions of the membrane; and the exact apposition of opposing surfaces is constantly changing.

In tracing the effects produced by the inflammatory process, the changes which take place both in the epithelium of serous membranes, and in the connective tissue elements below, ought to be studied. The examination "should not be confined to an examination of the lymph coating, its free surface, or to the mingled liquid and solid products lying in the serous cavity, but the membrane itself should be carefully inspected, not only at its free surface, but for some distance into its substance" (Turner, *Ed. Med. Jour.*, April, 1864).

The pericardium, like other serous membranes, is liable to inflammation, tending to the effusion of a serous fluid, diffused over a large extent of surface, and which sometimes becomes purulent—*suppurative pericarditis*, and the account here given of its morbid anatomy will, *ceteris paribus*, apply to other serous membranes.

In the early stages the free surface is no longer smooth and glistening, but becomes covered with a soft "lymph," loaded with rudimentary colourless corpuscles, as figured by Mr. Turner at fig. 36*a* of Sir James Paget's Lectures on *Surgical Pathology*, p. 348, 3rd edit., (see also p. 65, vol. i.) These corpuscles are not unlike those which first occur in the fluid of herpes. The changes in the pericardium itself are very early shown in the epithelial cells on its free surface, which become swollen, opaque, and loosened, so as to fall off into the serous cavity. A rapid multiplication of their curtained nuclei takes place, which are set free by the breaking down of the cells themselves. In the corpuscles of the connective tissue beneath the layer of epithelium, proliferation also goes on; small groups of rudimentary corpuscles appear, formed by germinating changes in the nucleated cells; and these multiply with such rapidity that adjacent groups run together and form masses of immature corpuscles—so numerous in the focus of inflammation that the inflamed part seems to be altogether composed of these new products—all trace of its natural structure being lost. The more superficial layers of this corpuscular inflammatory lymph becomes detached, and falls into the liquid exudation, which pours with more or less abundance into the serous cavity, where they mingle with the fibrinous coagula found in that fluid. When the inflammatory lymph becomes vascular, vessels are seen to pass into it from the connective tissue beneath the epithelium (PAGET and TURNER).

The pericardium at the commencement is seen to be reddened by dense capillary conjestion of vessels from the deeper textures, with here and there extravasations of blood. The tissues become infiltrated with serum, and losing cohesion, are easily torn. As the inflammatory lymph increases, the surface assumes a shaggy appearance, so that fine villi, papillæ, folds, or masses of new formation are developed by the proliferation of the material just described—the young connective tissue corpuscles. More often the appearance of the surface is rough and shaggy, like the villi of a large animal's intestine, or the surface of a bullock's tongue, or the lymph may be smooth as a gall-bladder. These changes constitute the first step in the formation of the so-called false membrane, the adhesive lymph which glues the surfaces of the pericardium to each other. The serous fluid thus seems to coagulate upon the opposed surfaces, and becomes vascular.

In the specimens of inflamed serous membranes examined by Professor Turner of Edinburgh, "the membrane was thickened and swollen, and covered by a soft layer of yellowish flaky inflammatory lymph, which could be readily scraped off with a knife. When the lymph was removed, an abundant bright red mottling of the membrane was noted. Examined microscopically, this inflammatory lymph was seen to be composed of small, pale, faintly granular cells, such as are familiar to all pathologists as products of the process of inflammation, and are described sometimes as the corpuscles of inflammatory lymph, at others as exudation corpuscles.

"The epithelium had either been shed from the surface of the membrane into the cavity, during the inflammatory process, or its characters had been so altered that it was no longer recognisable. Two opinions have been put forward," writes Professor Turner, "and supported by observations, of the part which the epithelium takes in the early stage of inflammation of a serous membrane. According to Dr. Julius Cohnheim (*Virchow's Archiv.*, vol. xxii., p. 516, 1861), some of whose observations were made on animals killed the day after he had irritated the peritoneum, the epithelial cells were found to be increased in size, more opaque, with no longer a mosaic arrangement, but lying next each other as large round globules, with a large shining nucleus. In some cells fatty degeneration had begun, and this could be traced in others up to the stage of disappearance of the cell membrane, and destruction and resolution of the cell into granules. Dr. E. Neumann's observations (*Virchow's Archiv.*, vol. xxiv., p. 202, 1862) agree with those of Cohnheim as far as relates to the degeneration and shedding of the epithelium cells. Dr. Rindfleisch (*Virchow's Archiv.*, vol. xxiii., p. 519, 1862), again, regards the epithelium as not necessarily destroyed by fatty degeneration at the commencement of the inflammatory process, but as assisting in the production of the new-formed cells of the inflammatory lymph. He induced artificial peritonitis in a rabbit, and found that the epithelial cells had lost their normal, six-sided, flattened form, that their corners had become rounded, and their shape was more globular. The contents also were more opaque, and commencement of nuclear division was observed. Rindfleisch considers that there is a direct metamorphosis of the epithelial scales into cells which, through their rounded form and divided nucleus, resemble pus-corpuscles. Even if these conclusions of Rindfleisch's be accepted, I cannot but think," says Professor Turner, "that the part which the epithelium plays in the production of the cells of the inflammatory lymph is comparatively unimportant, and that it is the sub-epithelial connective tissue we are more especially to look to as the seat of their formation.

"When sections through the inflamed serous texture were examined, before the addition of acetic acid and glycerine, a confused mass of new-formed cells mingled with the proper fibres of the sub-epithelial connective tissue in apparently inextricable confusion was seen. But after the addition of the above reagents had caused the white fibrous element of the tissue to disappear, the transparency of the textures was promoted, and the arrangement and relations of the new-formed cells could be followed, especially in those parts of the section where the elastic fibres were at a minimum. The new-formed cells were small, pale, and rounded, with faintly granular contents, and closely corresponded in their characters to those existing in the inflammatory lymph coating the free surface of the membrane. In those parts of the sections which lay immediately beneath this free surface, the cells were crowded together in great numbers, and formed closely packed rows or clustered masses. But somewhat deeper they were not so densely set, and here, therefore, their mode of arrangement could be more satisfactorily studied" (Turner, *Edin. Med. Journal*, April, 1864).

The exudation, which more or less distends the cavity, is separable into a fluid and a more or less solid portion. The liquid part may be scanty or it may amount to several ounces, or more. It first accumulates in the upper and anterior part of the sac, the heart occupying the lower portion. The inflammatory lymph also first begins to accumulate in this region, especially on the surface of the pericardium, which fits tightly round the heart, and most in the vicinity of the roots of the great vessels. *Post-mortem* evidence of *pericarditis* may be seen here when not found in any other part of the pericardium. When the serum is in great amount, however, the whole of the sac is distended, and the lungs, especially the left, are pressed backwards, and the thorax may be dilated over the region of the heart, whose cardiac dulness is increased.

The inflammatory lymph and fluid is generally heavily charged with fibrin, forming reticulated and villous masses upon the walls of the pericardium; so that the aspect of the heart's surface on opening the pericardium is like that of the surface of a sponge, or a network-like lace, or of two surfaces which have been besmeared over with a soft, plastic, sticky substance like lard, which, being placed in contact, have been quickly pulled apart. It is this form which is most commonly met with in the pericarditis of acute rheumatism (NIEMEYER).

The inflammatory lymph may be limited to a portion only over the heart, or it may be spread over the whole surface.

The substance of the heart itself may not, in early stages and recent cases, suffer any material alteration; but when the inflammation has continued for some time, the muscular texture of the heart becomes infiltrated with serum, softened and flabby, when dilatation of its cavities may supervene.

The ultimate results upon the pericardium itself are thickening in proportion to the amount of proliferation of tissue, and amount of inflammatory lymph which is not reabsorbed. If the products are small in quantity, the liquid portion may soon be absorbed; and the solids undergoing fatty degeneration, or liquefactive degeneration of some kind, are absorbed also. The greater the amount of lymph the more difficult is the absorption of the products. When thickened parts remain, they present the appearance of tendinous-like spots, and sometimes elongated white adhesions connect opposite surfaces together, which are not found to materially impede the action of the heart. Such terminations may be considered as recoveries for all practical purposes.

If the pericarditis continues for a long time chronic, the thickening of the membrane and new tissue may be very considerable all over the surface, so that permanent damage remains, and exercises an increasingly deleterious influence, even after the main bulk of inflammatory material has been absorbed. A fine fibrous mass of young connective tissue thickens the pericardium, and encloses the heart in a dense indurated capsule.

Partial inflammations continued for a length of time are apt to result in what have been termed "*white spots*," or "*milk spots*," *maculæ albidæ*, or *fibroid granulations.*

The *white* or *milk spot* is so often found in hearts which in other respects are perfectly healthy, that many pathologists doubt their morbid nature (Baillie, Sœmmering, Hodgkin, J. Reid). The anterior surface of the right ventricle is their most frequent seat. Occasionally these spots are observed upon the surface of the left ventricle, or upon the auricle, or upon the prominences of the coronary vessels. Their size varies from a fourpenny piece to a crown or larger. They are more common in adult than in early life; but they have been observed in the infant under three months old. They increase after the age of eighteen, apparently progressively with age. About 33 *per cent. post mortem* examinations from the ages of eighteen to thirty-nine show such *white spots;* and about 71 *per cent.* from ages between forty and eighty. Baillie, Laennec, Louis, and Todd, state that these opaque patches can easily be dissected or peeled off from the visceral layer of the pericardium, leaving the membrane entire. Corvisart, on the other hand, says they are seated on the under surface of the membrane. There seems, however, to be two forms of the white spot—namely, (1.) A superficial, which may be peeled off; and (2.) A deeper spot, which cannot be so detached (Bizot, Paget, King, Hodgkin).

A great difference of opinion prevails as to the cause of these spots. Some may, no doubt, be due to previous pericarditis, but all are not of this origin. The weight of evidence seems to be in favour of attrition being their cause, yet all are not referable to this cause either (Hodgkin, King, Jenner, Wilks).

The circumstances which seem to favour the development of this white spot are those which would increase the rubbing of the part against the pericardium applied to the anterior wall of the thorax. Those circumstances are—(1.) Dilated heart; (2.) Impeded action of the lungs (*a.*) from those diseases which, leading to augmented volume of the lungs, tend to press the heart forwards; and (*b.*) from continuous pressure upon the chest in an antero-posterior direction, commenced at an early age, before the epiphesis of the ribs and the pieces of the sternum are fully grown and united—as in young soldiers who, during great exertions, carry a loaded pack and wear cross-belts.

Those due to inflammatory origin will generally be found associated with other *post-mortem* evidences of inflammation, such as adhesions by distinct filaments or bands of lymph between the heart and pericardium, especially about the roots of the great vessels.

Pericarditis may also terminate in purulent formation (*suppurative pericarditis*). There may also be multiple and scattered accumulations of pus in the sub-serous layers, as in cases of pyæmia. Such local pus formations commence in the sub-serous filamentous tissue, beneath the cardiac fold of the pericardium, and occasionally assume the form of minute abscesses in the sub-serous tissue and on the surface of the cardiac fibres (Craigie, *Path. Anatomy*, p. 706).

In the chronic forms of pericarditis, inflammation may express itself (1.) By attacking the original membraniform exudation not yet removed; (2.) By thickening and pulpiness of the pericardium; (3.) By the formation of a thick, tough, universally unyielding case; (4.) By hæmorrhage; (5.) By purpura; (6.) By tuberculosis; (7.) By osseous concretions; (8.) By cysts from parasites and sacculated conditions of the pericardium, containing pus-like fluid, the remains of old pericarditis—so-called abscess of pericardium (*Path. Trans.*, vol. ix., p. 89).

The acute forms of pericarditis generally involve the muscular walls of the heart to a greater or less extent. On cutting through them, the muscular substance is seen for a greater or less depth, of a deeper colour than usual, and the cohesion of the tissue is also impaired, the finger readily passing through it.

General Symptoms.—The symptoms of pericarditis vary much as regards their expression, and especially in accordance with the co-existing malady with which it may be associated, thus modifying the symptoms and characters of each. In some instances they are most insidious in their approach; as, for example, when pericarditis follows upon *pleuritis* or *pneumonia, scrofula with tubercle* (*tubercular pericarditis*), *Bright's disease, chronic disease of the heart*, or *aortic aneurism;* and participation of the pericardium in the inflammation often remains undiscovered till revealed by *post-mortem* examination. In other cases the symptoms appear to be violent and unmistakeable from the commencement.

In pericarditis, rheumatism has been found to assume a new and formidable aspect; and rheumatic pericarditis is generally attended with more violent symptoms than non-rheumatic pericarditis. The most marked characteristics are, decided evidence of local trouble in the chest, especially (1.) pain more or less severe in the præcordial region; and from this point it radiates over the whole of the sternum, sometimes extending to the brachial plexus and down the left arm. This pain is accompanied by disturbance of the heart's action, a sensation of constriction over the whole chest, by urgent distress, and by an incapacity to take a long breath, or to cough. From these causes the patient is (2.) restless and anxious, and this anxious expression of the countenance is often peculiar and striking from the first. When acute pericarditis is not the result of rheumatism, the patient may suffer no pain, and the symptoms are often most obscure, general as well as physical. The countenance is pallid, and assumes an aspect of distress, and there is an incapacity or unwillingness on the part of the patient to lie on his left side. The pain in the region of the heart may be acute, severe, and shooting towards the shoulder, augmented by movement and increased by pressure upwards against the diaphragm. (3.) There may be febrile exacerbation, and the pulse, varying from 90 to 110, full and strong, and often intermittent, or otherwise irregular. (4.) Dyspnœa may exist in proportion to the distension of the sac with fluid, pressing on the lungs, and sometimes on the œsophagus, causing dysphagia, pain in the cardiac region, palpitation, and subsequent dyspnœa as the most common signs. This state of things having lasted from three to four days to a week, the patient may die suddenly.

Yet all of these signs may be absent. The pulse may give forth no sign; the breathing may not be changed; and pains may not exist, and yet pericarditis may be there. The action of the heart may get feebler and feebler, weaker and weaker, the circulation becoming irregular. The pulse at the most may get intermittent, the veins of the neck prominent, the skin cold and pale; and, with œdema of the limbs, death may soon follow, with pericarditis—latent, and unrecognized.

Before death, and often throughout the more severe periods of the disease, there is delirium of a peculiar kind—sometimes quiet, but often wild and furious. This delirium is peculiar, and has been noticed particularly by Sir Thomas Watson, Dr. Burrows, and others.

Even when the disease is most unmixed, it has been mistaken for a continued fever, or for pleurisy; and being usually one of the first complications of an otherwise fatal disorder, its mortality is thus far in excess of rheumatic pericarditis, which proves fatal in about one out of every six cases (Fuller).

When the pericardial effusion is great, it tends to impede, by its pressure, the action of the left lung; and hence the patient prefers in such a case to *lie on his* left side, so as to give more free play to the right lung; or he sits up, but bent forward in his bed.

Pure idiopathic pericarditis is rarely witnessed, and very rarely occurs as a severe or clinically important form of disease.

Dr. Stokes, in respect of morbid anatomy, arranges cases of pericarditis into three classes. In the first class are to be placed those in which there is a slight though general effusion of coagulable lymph. In the second, those in which there is supperadded the secretion of serum in abundance, causing distension of the pericardial sac. In the third class are to be placed those cases in which signs of muscular excitement, if not of myocarditis, are added to the preceding conditions. These three classes are thus contrasted, in respect to their symptoms or diagnosis, in the following tabular arrangement, given by Dr. F. Sibson:—

First Form.	Second Form.	Third Form.
Absence of pain or local suffering frequent. No sign of muscular excitement, nor any special character of pulse. No increase of dulness over the heart.	The local and general symptoms more decided, though often very trifling. Irregular action of the heart and pulse, often very manifest in the advanced periods. Remarkable increase of dulness over the heart.	Local distress, often extreme, even at the outset. Tumultuous action of the heart. Irregularity of pulse. Dyspnœa, orthopnœa, œdematous swellings, syncope, death.

As the disease passes from the first to the last of these forms, there is a progressive increase in the violence of the inflammation, denoted in the second form by the occurrence of excessive serous effusion, and in the last by the altered and impaired condition of the muscular substance of the heart itself. Death tends to occur by syncope, induced by paralysis of the left ventricle. Rokitansky thus describes the influence of pericarditis on the heart-tissue:—Its muscular substance is paralyzed, being of a dirty brown or yellow colour, flabby, and easily torn—a condition which speedily leads to passive dilatation of the heart, general cachexia, and dropsy.

The first stage of pericarditis, *before exudation,* is not discoverable by physical signs (STOKES). This period rarely lasts longer than thirty-six hours; and to Dr. Stokes, in 1833, the Science of Medicine owes the description of the most characteristic physical sign of pericarditis;—namely, a double *frottement* or friction-sound. Others also about the same time had noticed such a sound, and had correctly interpreted its meaning. Bouillaud and Collin, on the Continent, and Drs. Watson, Latham, and Mayne, in this country, had all, independently of each other, perceived and appreciated the symptom—a circumstance which, as Sir Thomas Watson justly remarks, gives to the symptom a greater amount of importance. This sound closely resembles a rasping murmur. It has been named a "*to and fro*" sound by Sir Thomas Watson. It is apt to disappear gradually from below upwards with the increase of effusion, and to return with its decrease. It may disappear from the apex to the base with the progressive formation of firm adhesions. It is usually limited to the region of the heart, but changes its character and its seat from day to day. It is sometimes remarkably modified by local bleeding, passing from a loud rough sound to a soft bellows murmur—most rough and intense during inspiration. The hand applied over the cardiac region will sometimes detect a rubbing sensation, which ceases with the cessation of pericarditis.

In diagnosis, however, it is necessary to bear in mind that *friction-sound is not necessarily present in pericarditis.* During the progress of a case, friction-sound may be absent or it may be present for long periods —its presence or its absence bearing no appreciable relation to the intensity of the disease. The amount of fluid effusion has much to do with this. A really considerable effusion of fluid generally at first muffles, then renders barely audible, and finally removes the sound, the friction-sound becoming indistinct as the heart's sounds are gradually extinguished. Like the heart's sounds, the friction-sound continues audible longest, and is recovered soonest towards the base. When the lymph is rapidly condensed into firm granulations, and the parts of those granulations most exposed to attrition have become polished and rubbed away, so that the points gradually receding from each other present fewer and fewer points of contact, then the friction-sound may subside, although no further effusion of fluid takes place (W. T. GAIRDNER).

Again, *the presence of friction-sound is not necessarily a proof of the existence of pericarditis.* There are permanent exocardial murmurs, probably associated with the well-known "*milk spots*" on the anterior aspect of the heart, as pointed out by Professor Gairdner. When it is remembered how frequently slight, short, and ill-defined murmurs, especially with the first sound of the heart, may be discovered, in persons otherwise healthy, about the left border of the sternum, at the level of the third and fourth intercostal spaces, or lower, the existence of "milk spots" may be their cause. *It is only when the murmur arises for the first time under observation, or when it accurately coincides with the development of symptoms, or where it corroborates and explains the symptoms, and the other physical signs already existing, in such a manner as to leave no doubt of its nature, that we are justified in assuming that a friction-murmur over the heart is pathognomonic of acute pericarditis.*

These pericardial or exocardial friction-sounds or murmurs may be, and are often, mistaken for endocardial murmurs (TAYLOR, STOKES, GRAVES, SKODA, SIBSON), and the distinction between exocardial and endocardial murmurs is not always easy, nor is it to be effected by the ear alone. The following distinguishing signs are condensed from Dr. Sibson's interesting review of the works of Stokes and Bellingham on diseases of the heart (*Medico-Chirurgical Review* for 1854):

The exocardial may be distinguished from the endocardial sounds by the nature and nearness of the exocardial sound—by its existence with diastole as well as systole—its limitation to the region of the heart—its non-existence over the great vessels—its variations over different parts of the heart—its rapid and frequent change in character, or its disappearance from day to day—its want of correspondence with the rhythm of the heart, while it seems to follow upon its movements (SKODA), or to precede and follow the impulse (WUNDERLICH)—its co-existence with tactile vibration, and where there is much effusion, with an extensive cone-shaped region of cardiac dulness. The apex of this dull region points to the top of the sternum, its broad base extending downwards to the right, and far to the left of the epigastrium. In addition to these signs, which, when they exist, serve to establish the existence of pericarditis, and separate pericardiac from endocardial murmurs, we have other very characteristic signs by which to distinguish every case of pericarditis, especially in the earlier stages. By the aid of pressure, as first demonstrated by Dr. F. Sibson, applied gently over the region of the heart, we have a test decisive as to the cause of these sounds, when we are in doubt as to whether it is endocardial or exocardial. If the noise is that of a valve-murmur, pressure from without does not increase or modify it, except in some anæmic persons, over the aorta. If, on the other hand, it is that of a friction murmur, soft and bellows-like, of exocardial origin, pressure intensifies the noise, and converts the sound into a rustle or rub. By pressing gently on the costal cartilage or end of the sternum with the stethoscope, the intermediate fluid is displaced, and the roughened surfaces are brought into contact. This method of diagnosis is most valuable, especially in the early stages, when it is of real importance to arrive at a correct diagnosis (*Provincial Med. Trans.*, vol. xii., p. 540).

"A really refined and intellectual diagnosis," and not one founded on the mere *aural* recognition of acoustic characters, is necessary to guard against serious mistakes. In the majority of cases where the friction-sound of pericarditis is recognized, it is known to be such by the circumstances in which it occurs rather than by the mere character of the sound itself (STOKES, WALSHE, FULLER, GAIRDNER). Increased extent of dulness in percussion, and marked prominence over the cardiac region, are also two characteristic signs.

When extensive effusion takes place, the heart is pushed upwards to the fourth, third, or second intercostal spaces; consequently the seat of the heart's impulse, of the rubbing sounds, and of tactile vibrations, all are correspondingly raised (SIBSON, LATHAM, WALSHE).

A valuable distinctive sign of pericardial effusion, when contrasted with pleuritic effusion, is, that when the left side is dull in front and resonant behind, it is a pericardial, and not a pleuritic effusion (STOKES). Any large increase of fluid at once betrays itself, especially in the young, by the protrusion of the left cartilages and ribs, the widening of their interspaces, prominence of the ensiform cartilage, and, in some extreme cases, by an epigastric fulness or even tumor. When the fluid increases, the pulse becomes feebler, and more disposed to falter and to flutter. It becomes irregular and excited; and often the patient is so fixed in one position that he fears to move, lest he may aggravate by exertion the dyspnœa and action of the heart from which he suffers so intensely. The jugular veins not unfrequently become distended, and this distension does not lessen during inspiration when the effusion is great. A significant sign is thus furnished of the greatness of the obstruction which exists to the thoracic circulation. Œdema and great coldness of the extremities are also apt to supervene with such a state of things. When, however, the products of inflammation become solid, and little serum remains, the pericardium, by the opposed serous surfaces, becomes attached to the heart throughout, and the pulse then resumes its force and regularity; and, if the patient survives, this adhesion remains for life.

The occurrence of acute pericarditis apart from vital phenomena or symptoms is not now believed in, and among the most characteristic general symptoms is the occurrence of a dry short cough, not explained by any morbid state of the lung.

The physical signs of pericardities may be summed up as follows:—

(1.) *Inspection* often discloses a distinct bulging of the cardiac region, especially in children and the young. The thoracic walls must be yielding, and the effusions large to produce this effect; and ossification of the costal cartilages, as after the adult age, is apt to hinder its production.

(2.) *Palpation* so as to feel the position of the heart's beat, as to whether it is in its proper place, and also as to the force of its impulse.

The more copious the exudation the weaker is the impulse, unless in cases of hypertrophy, or violent palpitation. Different positions of the patient influence the results. Standing upright, the impulse may be felt; lying down, the heart recedes behind the fluid which separates it from the thoracic walls. Friction also is said to have been felt by the hand laid over the region of the heart in some cases. (NIEMEYER).

(3.) *Percussion* may reveal nothing if the lung covers the pericardium, even although exudation may be present to the extent of half a pound

(NIEMEYER). On the other hand, the *form* which the dulness assumes over the heart is one of the most characteristic and important signs of the disease. At first the increase of dulness is towards the root of the aorta and pulmonary vessels, and extends upwards to the second rib, or even higher, and passes beyond the right edge of the sternum. When the effusion is very copious, the dulness forms a triangle with the base to the diaphragm and the obtuse apex alone. As it extends lower down it gets broader, and may pass beyond the right border of the sternum on the one side and the left mammary line on the other. Extension of the dulness in this direction to the left side, beyond the point at which the apex beats, is a positive sign of the existence of a collection of fluid in the pericardium (NIEMEYER).

(4). *Auscultation* brings out the remarkable disproportion between the extreme dulness and the extremely feeble impulse and the inaudibleness of the heart's sounds. Friction-sounds like those of pleuritis may be heard before the surfaces are separated by fluid, and may again return as the fluid disappears (see p. 555).

Prognosis.—While the consequence of pericarditis "is sometimes, though rarely, the speedy extinction of life, in nineteen cases out of twenty the disorder proves fatal at a remote period, destroying the subject of it more slowly, indeed, but almost as surely." Such is the opinion of Sir Thomas Watson; and again he writes, "I am certain it is a fertile, but often unsuspected source of chronic disease of the muscular substance of the heart, and of its consequences, asthma, dropsy, sudden death." Sometimes death occurs suddenly when least expected; and the mode of death tends to be by asthenia. A fatal termination may occur, although rarely, in less than forty-eight or thirty hours. In ordinary cases which progress favourably, the disease generally begins to yield in the course of a week or ten days, and sometimes sooner under active treatment.

It is not now, however, generally believed that when the disease terminates by adhesion, dangerous or fatal lesions of the heart tend to supervene, as Dr. Hope believed. It is consistent with the experience of Drs. Stokes, Sibson, Wood, of Pennsylvania, W. T. Gairdner, Smith, and other observers, that hypertrophy and dilatation of the heart do not commonly follow on pericardiac adhesion. Sir Thomas Watson, however, with many others, regards adhesion of the pericardium as a suspicious prognostic of future evil—"that other structural changes will soon or slowly develop themselves, and first render life burdensome and full of suffering, and then consign the patient to an earlier grave than might else have awaited him."

Among the patients of a general hospital, from 6 to 8 *per cent.* die of pericarditis, while a proportion, varying from 3 to 8 *per cent.*, have had the disease at a previous period and recovered from it (W. T. GAIRDNER).

Generally it may be stated that there are differences in the results of pericarditis in the healthy compared with the results in the unhealthy, sickly, weak, "worn out," pale, or scrofulous. A person otherwise in fair health may survive a first shock of such a disease, but with a damaged organ for life. The sickly, weak, and scrofulous linger on, victims to palpitation, dyspnœa, often approaching to suffocation, and after some weeks may die suddenly. The worst class of cases are those in which pus is found with the serum; and the greater the amount of effusion the more unfavourable the case. The more serum there is with lymph the more unfavourable is the case, especially if no indication of absorption exists. Where there is only inflammatory lymph occurring in an otherwise robust person, the case is not unfavourable.

When pericarditis accompanies *pneumonia, pleurisy,* or *acute articular rheumatism,* the result, so far as pericarditis is concerned, is generally favourable;—not so, however, when it complicates *Bright's disease,* disease of *the heart, aneurism,* or *scrofula* with *tubercle,* or in the *purulent* form, as in *septicœmia.* But even in the cases that do appear to recover, remote effects may still result, especially chronic pericarditis—the inflammation sooner or later breaking out afresh—which rarely terminates in complete recovery, and which of itself does not directly prove fatal, but generally indirectly through œdema of the lungs and slow suffocation. The exudation in such chronic cases is generally extremely profuse, and the dyspnœa severe. Relapses of inflammation seem to follow relapses, and the substance of the heart itself becomes extremely soft, relaxed, and discoloured. The circulation then begins to indicate distress, the pulse becomes small and irregular, the venous system overloaded, and general dropsy sets in. But depression of the cardiac action is generally most intense in the cases of suppurative pericarditis, a result which may be not less due to the constitutional disease than to the local lesions.

The most common sequelæ of pericarditis are as follow:—

(1.) Adhesion of the oppressed surfaces of the pericardium, with more or less complete obliteration of the pericardial sac.

(2.) Dilatation of the heart, the more likely to result in proportion to the length of the attack, and the amount or extent of adhesion.

(3.) Hypertrophy of the heart, if its texture be sound;—or,

(4.) Atrophy and fatty degeneration, from constant chronic infiltration of the heart's texture, as well as from the pressure of the surrounding morbid pericardium.

Cause.—Pericarditis is generally the local manifestation of a disease which pervades the system. There is scarcely a form of constitutional malady which may not be accompanied by this lesion; but those with which it is most apt to be associated are *rheumatic fever, Bright's disease, pyœmia, scarlatina, small-pox, scurvy, pneumonia, pleurisy, peritonitis, hydatid cysts, abscess in liver* or *mediastinum, inflammation of lungs* or *pleura, scrofula, cancer.* These diseases are mentioned in the order of the frequency with which they give rise to pericarditis, as consistent with the observations of Drs. Taylor, T. K. Chambers, A. W. Barclay, and Dr. Fuller. In cases of *acute rheumatism,* pericarditis occurs once in every 5·97 cases (Fuller).

The muscular substance of the heart sometimes suffers in cases of pyæmia consequent on the vitiated state of the arterial blood, and pericarditis has been known to arise in this way (Kirkes, *Med. Times and Gazette,* October 25, 1862, p. 432). Such cases have usually been described as cases of carditis, or acute inflammation of the muscular substance of the heart depositing pus. Dr. Kirkes, however, believes that the muscular tissue of the heart is not primarily at fault; but, in common with other parts of the body, it is by accident the seat of secondary formations in consequence of contaminated blood; and the attendant pericarditis is an accident of the proximity of some of the deposits to the surface, and it may be of their bursting into the cavity, just as an ulcer in the wall of the intestine may burst into the *peritoneum* and cause *peritonitis.* It has also occurred by extension of other diseases from neighbouring organs—*e. g.*, cancer of œsophagus, and the like (J. W. Begbie). (For more details regarding pericarditis, the student is recommended to study the excellent treatises of Dr. Fuller *On Rheumatism* and *On Diseases of the Chest;* also Walshe and Stokes *On Diseases of the Heart.*)

Treatment.—The results of recorded treatment are unsatisfactory in the highest degree. Sir Thomas Watson thus writes, that "in a large proportion of the cases, whether they be treated well or ill, or not treated at all, the patients will seem to recover." Although blood-letting and calomel have hitherto been regarded as two most efficient agents in accomplishing the indications of cure, yet Taylor's cases show that pericarditis may come on during salivation. Dr. Parkes informs me that he has seen this occur two or three times. It is, then, very doubtful if calomel ever does any good in pericarditis, notwithstanding that its use, so as to affect the gums, is advised by Watson, Graves, Stokes, and Fuller. Professor W. T. Gairdner, Drs. John Taylor, of Huddersfield (*Med. Times and Gazette,* 1849), and J. Risdon Bennett, of London (*Lancet,* Dec. 6, 1851), are all unfavourable to the use of mercury. In all the *constitutional diseases* in which pericarditis occurs as a complication, mercury is certainly contra-indicated. In rheumatism and in Bright's disease, which furnish by far the larger number of cases of pericarditis, mercury is decidedly objectionable; and it is now well known to be productive of most injurious consequences in Bright's disease. Its use tends to weaken the heart and the system at large, and thus proves a source of additional irritation to the cardiac tissue, by undue re-action on the system.

Dr. Fuller, however, gives his opinion very strongly in favour of mercury as a remedy in the acute and sthenic form of pericarditis, as it presents itself in persons of a strong and healthy constitution. "No such case," he says, "can be treated safely without its administration." Such idiopathic cases are indeed rare, the causes which induce pericarditis being for the most part constitutional.

"A single bleeding from the arm," writes Dr. Stokes, "appears on the whole justifiable, but its repetition will be a matter for careful consideration." It is a remedy which has been clearly shown by the late Dr. Taylor to shorten the duration of pericarditis, and to do so the more effectually the earlier it is performed (*Med. Times and Gazette,* 1851). Niemeyer, on the other hand, is of opinion that general blood-letting is never required in pericarditis as such, and its employment is to be confined to the very few cases in which the repressed outflow from the veins into the heart causes symptoms of pressure upon the brain, and demands a reduction of volume in the circulation. The treatment by bleeding may also be further carried out by leeches over the præcordial region, or by cupping there. In cases of rheumatism, Drs. Stokes and Gairdner hold that "it cannot be too strongly impressed upon the mind that, valuable as the discovery of the signs of an inflamed pericardium may be, it is not for these alone that he is to look, but rather for the indications of excitement of the heart, whether attended or not by the signs of exocardial or endocardial disease." These indications may demand local depletion, "even though no friction-sound or valvular murmur whatever be present." Sir Thomas Watson gives a most judicious opinion when he says,—"I think there is a peculiar risk in frequently bleeding to syncope in this affection." Blood may be drawn from the arm till some effect upon the pulse is produced; but he gives the preference to repeated relays of leeches over the præcordial region, or to free cupping. Dr. W. T. Gairdner advises that when the pain is very marked, where it has strongly the characters of angina, leeches are to be applied in moderate numbers. From *four* to *six* leeches so applied, followed by fomentations, very commonly relieve the pain, and rapid improvement follows. Local blood-letting, says Niemeyer, moderates the pain somewhat, and is indicated where it is troublesome. The effect of applying from ten to twenty leeches, according to circumstances, to the left edge of the sternum, is astonishing in most cases.

To judge as to the repetition of blood-letting, the force of the heart must be observed, as indicated by the pulse at the wrist, by the actual strength of its impulse, and by the character especially of the first sound. If the impulse continues vigorous, and the first sound is undiminished, blood-letting may be repeated by the further *local* abstraction of blood; but if the impulse has manifestly declined in force, while the first sound is lessened, great caution is required in the further abstraction of blood. Fomentations, sometimes plain and sometimes medicated with opium, friction with *camphorated* and *iodureted* liniments, and, in obstinate cases, the use of blisters, are the chief local remedies to be relied on besides leeches.

In the second stage of the disease, when liquid effusion distends the pericardium, some reliance may be put in treatment by a blister of a large size over the præcordia.

"Stimulants," writes Dr. Stokes, "are often imperatively called for. The weakened heart requires to be supported and invigorated. This may be effected by the use of wine, and by the use of opium." If depletion has been excessive—if signs of muscular weakness supervene—if there be evidence that the heart, previous to the attack, was in a weakened state—if a tendency to collapse or to a typhoid state manifests itself—"we must give wine, quite irrespective of the physical conditions of the heart" (STOKES).

Niemeyer recommends *digitalis* as a suitable remedy in cases where the beat of the heart is very frequent and insufficient, causing cyanotic and dropsical symptoms. Its effect is then very markedly beneficial.

Opium, in doses of one grain (*i. e.*, stimulant doses) every third hour, seems "to expend itself solely on the disease," and its beneficial effects are seen to result when it does not produce contraction of the pupil, nor headache, hot skin, furred tongue, nor constipation (CORRIGAN, STOKES, GRAVES, SIBSON). Battley's solution is perhaps the most useful preparation. This remedy must be watched closely.

There are two more important circumstances to be attended to, as stated by Dr. Sibson—namely, that our treatment of pericarditis must depend upon the stage of the disease in which it is first discovered to exist; and it is important to know that pericarditis from acute rheumatism calls for a totally different line of treatment from that associated with Bright's disease, or diffused inflammation of a low type.

Niemeyer considers that the use of cold applications, such as ice-bladders, applied over the cardiac region, is deserving of great reliance.

In the rheumatic form, colchicum, in the form of a draught, and the administration of alkaline remedies, are indicated by the constitutional state. Warmth, especially moist warmth, and flying blisters are the best agents to promote absorption.

Paracentesis is recommended by Dr. Sibson in all those cases in which the effusion is so great as to cause alarming distress, orthopnœa, obstruction to the venous circulation, and serious interference with the heart's action. In such cases a fine exploring trochar and canula is to be introduced (not plunged), so as to make a valvular opening below the heart, either to the left of the xyphoid cartilage, or through the fifth intercostal space, close to its anterior extremity, and the fluid drawn off by means of a syringe (Sibson, in *Med.-Chir. Review*, July, 1854). The result may be merely palliative; but, as Niemeyer justly remarks, "even to afford the sufferer opportunity, after the operation, to pass the night in his bed (perhaps for the first time in a long period) and to enable him to sleep a little, is a great gain."

The general principles of safe treatment are summed up by Dr. W. T. Gairdner as follow:—(1.) To make large allowance for the insignificant and spontaneously healing class of cases revealed more by physical signs than by symptoms, and to regard them as demanding little active treatment; (2.) To consider rheumatic pericarditis in general as a disease susceptible, to a great extent, of cure under palliative local remedies and fitting constitutional treatment; (3.) To hold the general treatment as subordinate to the constitutional treatment of the disease with which the pericarditis is associated.

SUPPURATIVE PERICARDITIS.

LATIN EQ., *Pericarditis suppurans;* FRENCH EQ., *Péricardite suppurée;* GERMAN EQ., *Eiterige pericarditis;* ITALIAN EQ., *Pericarditide suppurativa.*

Definition.—*An accumulation of pus in the pericardium.*

Pathology.—When acute inflammation results in an accumulation of pus in the pericardium (which it does slowly), it is generally associated with a constitutional cause, and the pus may be of a laudable healthy character, though sometimes of a greenish hue. The quantity may be only a few ounces, or so abundant as to fill the pericardium.

The pericarditis which attends *septicœmia, puerperal fever, variola, scarlet fever*, and the like, is generally a result of the morbid conditions established by these diseases, manifesting itself by a series of inflammatory disturbances (*hyogenic fever*), attacking especially the *skin*, the *joints*, and the *pericardium*, where the effusion is generally purulent.

As soon as the young cells—the pus corpuscles of the inflammatory exudation—mingle with fluid effusion to a visible amount, so as to become yellow and opaque, the fluid of pericarditis assumes the character of thin pus. The fibrinous deposits that occur are then remarkably yellow, rotten, and pasty. It is a purulent lymph, which rises up like an empyema, generally in cases of protracted pericarditis, with sero-fibrinous exudation abundant. Sometimes the exudation is purulent from the first, as in *septicœmia* and *puerperal fever*. The muscle texture of the heart is generally very much disorganized in such cases,—discoloured, flabby, and softened, the epicardial layers undergoing fatty degeneration (VIRCHOW).

Pyæmic cases of suppurative pericarditis are sometimes the results of accidents to the pharynx, giving rise to purulent infection, by phlegmonous inflammation of the areolar tissue at the back of the mouth, descending between the pharynx and spine. Destruction of the sub-serous tissue of the pericardium and pleuræ takes place, and purulent pericarditis. I dissected such a case in Glasgow Infirmary many years ago, where the back of the mouth was injured by a dirty spoon; and Dr. W. T. Gairdner also records a case, p. 16 of his *Clinical Notes*.

Treatment.—Nothing special can be said regarding such cases, more than has been already stated in the previous pages. Depression of cardiac action is most intense in such cases, and such threatened heart-palsy demands stimulants.

TUBERCULAR PERICARDITIS.

LATIN EQ., *Pericarditis tuberculosa;* FRENCH EQ., *Péricardite tuberculeuse;* GERMAN EQ., *Tuberculöse pericarditis;* ITALIAN EQ., *Pericarditide tubercolase.*

Definition.—*Pericarditis in a scrofulous constitution, with miliary tubercles developed in the young membranous tissue of chronic pericarditis, and attended with repeated outbreaks of inflammation.*

Pathology.—In the scrofulous cachexia, where *acute miliary tuberculosis* of the lungs exist, pericarditis with minute tubercle on the pericardium may be also found. It is a common form of pericarditis in the army, and its invasion is very insidious, so that, without physical examination its recognition and diagnosis may not be possible. It is also often rapid and acute in its results.

The greyish nodules of tubercle on the pericardium do not undergo farther change to softening, but the patient dies rapidly of the fever, often without betraying any symptoms of the existence of tubercles in the pericardium, or of pericarditis.

It is one of the frequent forms of chronic pericarditis, so that inflammatory attacks are frequently repeated. There is generally also a hæmorrhagic effusion in the sac in such cases. In cases where there is a disposition to purpura or scurvy, or during the progress of some malignant febrile disease, it occasionally happens that blood as well as serum or pus is effused within the sac (*Hæmorrhagic pericarditis*). The source of the blood is from the new-formed vessels of inflammatory exudation, which, being yet tender, delicate, and thin-walled, give way (see vol. i., p. 77); or it may be from the highly congested vessels of the serous membrane, and the softened tissues which prevail in scorbutus. The lymph of such cases is of a spongy red colour and shaggy appearance, exactly similar to that which exudes from the large intestine into the small gut in cases of scorbutic dysentery.

If the blood is small in amount, the serum of the pericarditis has a reddish colour, but if the flow be considerable, the effused mass may resemble a pure extravasation, and assume a blackish colour. The lesion sometimes occurs in recent pericarditis,—occurring in cachetic patients, such as those suffering from *chronic alcoholism*, *scrofula*, and *Bright's disease.* The walls are studded with translucent prominences of the pericardium, which may afterwards become yellow and cheesy.

Treatment.—Nothing more special can be said regarding the treatment of this form than has been said under *scrofula*, and in the preceding pages on pericarditis.

ADHERENT PERICARDIUM.

LATIN EQ., *Pericardium adhærens;* FRENCH EQ., *Péricarde adhérente;* GERMAN EQ., *Verwachsung des Pericardiums;* ITALIAN EQ., *Pericardio aderente.*

Definition.—*More or less adhesion of the opposed surfaces of the pericardium by inflammatory exudation (adhesive lymph) becoming organized, and with more or less complete obliteration of the pericardial sac.*

Pathology.—When lymph has been effused in such cases, it is then commonly found organized, so that the pericardium is often partially or universally adherent all over the heart. In some instances the lymph effused, instead of forming adhesions, becomes converted into cartilaginous and even osseous patches, which are readily detached from the surface of the heart by the scalpel.

Parasitic cysts may also establish adhesions, as well as fatty and fibroid growths. The adhesions are thus sometimes partial and sometimes total; sometimes consisting in a firm agglutination of the opposed surfaces, or merely in a few long bands and fibres.

In some cases the remaining pericardial tissue is thus transparent, atrophic, and dry, so that the heart almost appears as if it were naked; in other cases the pericardium is converted into a dense, indurated, unyielding case, or into a cartilaginous mass. The adhesion may be so indurate, that remains of effusions may still exist, in a sacculated condition, sometimes purulent or so-called abscesses, as already noticed.

Symptoms.—The subsequent degeneration of the heart, which an adherent pericardium entails, brings about functional difficulty in its action, which may be still farther aggravated by valvular disease or other cardiac lesions. The more dense and unyielding the enclosing pericardium is, the greater is the functional difficulty of the heart. The pulse becomes extremely small and always very irregular, dyspnœa, cyanosis, and dropsy very soon appear. A sinking of the intercostal space with the heart's beats, and a rising again during diastole at the region of the apex impulse, is considered a sign of value, as indicating an adherent pericardium; but the symptom is also often wanting (NIEMEYER). No symptoms are distinctive of adherent pericardium (see also Wilks in *Guy's Hospital Reports*, vol. xvi., p. 196, 1871).

Treatment must be regulated by the functional difficulties of the circulation, caused by the adherent pericardium, and by the source to which those disorders of circulation may be traced as due.

DROPSY OF THE PERICARDIUM.

LATIN EQ., *Hydrops-pericardii;* FRENCH EQ., *Hydro-péricarde;* GERMAN EQ., *Herzwassersucht*—Syn., *Hydro-pericardium;* ITALIAN EQ., *Idro-pericardio.*

Definition.—*An increase of the normal liquor pericardii, containing little albumen, and analogous to the fluid of chronic hydrocephalus.*

Pathology.—Inflammation of the pericardium may terminate by resolution; but more commonly serous effusion results, besides the solid lymph, and the quantity of serum effused varies from a few ounces to a few pints. The solid lymph is then often found in shreds.

Louis has given one case in which the effusion amounted to four pounds, and Corvisart another in which the quantity was still more considerable. Walshe records sixty ounces—and when such great effusions occur, the diaphragm is depressed below the end of the sternum, by the great amount of fluid contained in the sac of the pericardium.

Inflammatory dropsy generally contains fibrin.

Increased amount of fluid in the sac of the pericardium also occurs in cases of wasting, with shrinking or diminution in the bulk of the heart (*Hydro-pericardium ex vacuo*).

The veins of the heart being obstructed also lead to increase of fluid. It is thus seen in diseases of *mitral valves, emphysema,* cirrhosis of the lungs,—diseases in which the right side of the heart is working under abnormal conditions.

In other cases it occurs as a part of general dropsy, as in *Bright's disease, chronic affections of the spleen,* or *cancerous conditions* of the system.

When the fluid is large in amount, the pericardium is dull, white, and without lustre; the fat has disappeared from the surface of the heart, and its connective tissue is œdematous.

Symptoms.—The condition is always secondary, as already indicated in its pathology; and, therefore, symptoms beyond the physical signs of effusion are to be sought for in connection with those lesions of which it is a more or less constant result.

Treatment must be regulated by the attendant circumstances of the case, and in accordance with what has been already written on the subject of pericarditis.

(b.) *Diseases of the Endocardium.*

ENDOCARDITIS.

Latin Eq., *Endocarditis;* French Eq., *Endocardite;* German Eq., *Endocarditis;* Italian Eq., *Endocarditide.*

Definition.—*An inflammation of the lining serous membrane of the heart, covering the valves and lining the chambers of that organ.*

Pathology and Morbid Anatomy.—By a species of preference (the reasons for which are in a great measure speculative), the coverings of the orifices and valvular structures of the heart are by far the most frequent seat of lesion in the internal inflammation of that organ.

The frequency with which these parts suffer may in some measure be explained if their minute anatomical and histological relations are connected with the morbid states of similar tissue. The peculiarity of the minute structure of these parts, as influencing the arrangement assumed in the first instance by morbid deposits, seems to have been first definitely stated and illustrated by Sir Thomas Watson, (Lect. lxi., p. 275, 3d edit.) There is found to exist, enclosed between the reduplications of the endocardium, a quantity of fibrous tissue. An increase in its amount takes place at the centre of each aortic valve, forming the *corpora Arantii,* and at each of the extremities or angles of the segments. The minute exudations which are formed as the result of the inflammatory process in the endocardium may be seen to arrange themselves in double festoons, suspended as if from the *corpus Arantii,* often in a row, like a string of beads, along the line of union of the thick portion of the valve, with the inner convex margin of its two thinner crescentic portions. The repeated attrition of the opposed surfaces of the valves pushes aside the exudation as fast as it is deposited, and while yet plastic, from the thin crescentic portions of each valve, and so heaps it up along those boundary lines of contact; "just as a thin layer of butter on a board would be displaced and heaped up in a little curvilinear ridge by the pressure of one's thumb."

While, doubtless, the structure of the serous membrane is the same throughout the heart, yet its mixture with white fibrous and elastic tissue, in great abundance at the valvular reduplications, renders it prone to disease in those constitutional states of the system in which the fibrous textures are more particularly involved, as in rheumatism. There can be no doubt that the great majority of cases of *endocarditis* arises in the course of *acute articular rheumatism,* and all the more readily the greater the number of joints attacked. About 20 *per cent.* of the cases of acute articular rheumatism are said to be complicated with *endocarditis* (Bamberger).

At the same time, it is not improbable that an *idiopathic chronic* endocarditis is not uncommon (Niemeyer).

Bright's disease is the morbid state next in frequency, which is complicated with *endocarditis*, especially the acute form developed after scarlet fever.

Acute infectious disorders, especially puerperal fever and measles, are also prone to be complicated with *endocarditis*. Wunderlich, indeed, regards *measles* as the most prolific cause of endocarditis next to *rheumatism*. In all such cases it seems not improbable that the irritant which sets up the inflammation, acting mainly on the tissue of the narrow passages through the heart—its orifices and valves—is the super-heated blood of the fever-patient, as marked by the intensity of the fever (BILLROTH, WEBER, NIEMEYER).

The parts affected in endocarditis, whether constitutional, as from rheumatism, or idiopathic and local, are also the parts on which the great tear and wear on the action of the heart is expended, and thus they are probably the first to suffer, owing to the mutual friction of the valvular edges upon each other. They are those parts of the endocardium which are especially prone to strain and friction from the constant action of the heart. "It is the narrow places, the outlets, which are most often diseased; but especially those portions of the valves which strike against one another inclosing the auricular surfaces of the mitral and tricuspid, and the convex surfaces of the semilunar valves" (NIEMEYER).

Diffuse inflammation of the endocardial membrane has been known to follow the application of a ligature round an artery, and Bamberger is stated to have seen only two cases of traumatic origin.

The morbid appearances of inflammation of the endocardium are similar to those in other serous textures—namely, a silvery opacity, and more or less thickening of its tissue. Inflammatory lymph is often found strongly adherent to the valves, as already described, and forming fringe-like or fibrinous warty out-growths, or excrescences, as they are termed. By its agency the segments or lappets of the valves become variously distorted in shape, or soldered together, and insufficient to perform their functions. An orifice, naturally large, may thus be reduced to the condition of a mere slit, or to the diameter of a goose quill. One segment of the aortic valve may, for example, be turned up and bound to the aorta, or it may be turned down and bound to the inner surface of the heart, or it may be curled up like a shell. The lappets of the mitral valve may be similarly altered. The *chordæ tendineæ* may be similarly fixed by adhesions—and those lesions are of grave importance—orifices are contracted, and their closure rendered impossible; and how the parts come to adhere, seeing that they are in constant movement, is one of the most interesting questions in the pathology of endocarditis (NIEMEYER).

Virchow regards the hypothesis of the formation of a *free* inflammatory exudation in endocarditis as at least not proven, and even doubtful. The inner *arterial tunics* and the *endorcardium*, when they suffer from inflammation, suffer from the *parenchymatous form* (see vol. i., pp. 37 and 60), namely, that form in which active disturbances of nutrition are provoked by an irritation; but which, instead of producing an exudation *between and amongst* the elements of the tissues, causes a *swelling* of the individual normal elements themselves, resulting in proliferation of their cells and increased volume of the part. Thus, the general result arrived at from the most recent observations on the inflammatory process by Virchow, Lister, Turner, Moxon, and Wilks, especially tend to confirm the statements of the late John Goodsir, that "*an explanation of its phenonema is not to be looked for by referring them to actions of the extreme vessels, but to a disturbance of the forces which naturally exist in the extra-vascular portions of the inflamed part*" (*Anat. and Path. Observations*, 1845, p. 43).

The inflammation of endocarditis does not originate in the deep layers of the endocardium, but in its extra-vascular and superficial minute elements of tissue. The minute elements of the tissue become swollen—enlarged individually, by the infiltration of a liquid, the chemical properties of which resemble *mucin*—a *mucinous* inflammation—the fluid of which coagulates into the form of threads upon the addition of acetic acid. A vast formation (proliferation) of new cell-growth at once commences, each new growth, as the inflammation advances, rising up like a Phœnix, out of the ashes of the old.

These new cells become organized into connective tissue, which often develops into reddish or greyish-red delicate villi, as the disease advances, giving the endocardium a finely granular aspect, or covers it with coarse granular wart-like papillæ, firm and hard at their base, with round, bulbous, soft and gelatinous free ends. These constitute the valvular vegetations, to which deposits of fibrin from the blood adhere, which must be distinguished from the vegetations themselves.

In some rare cases ulceration takes place, of which Virchow gives a drawing (*Cell. Path.*, p. 208—*Chance's Translation*), in which the *mitral* valve is implicated, and beneath its free smooth surface, the connective tissue corpuscles are seen enlarged and clouded, with dense tissue intervening. Enlargement, cloudiness, and proliferation of cells goes forward with such activity that the tissue softens and breaks up, producing loss of substance and ulceration of the endocardium—a condition of great danger, and often the starting-point of *true metastasis.* The minuteness and friability of the parts favour the penetration of the softened and separated particles into the smallest capillary blood-vessels of distant parts, such as the *spleen, kidney, brain,* and *heart* itself (*capillary embolia*) and hæmorrhagic infarction, fibrinous accumulation, or metastatic abscess, which is rare, may be the result.

It is rare for the *liver* or the *lungs* to be so affected on account of the direction and mode of their *arterial* connections. If particles of fibrinous clots, and such like *debris* of ulceration, pass into the *carotid* or *vertebral* arteries, then according as the artery of the brain is totally or partially occluded, will be the formation of hæmorrhagic foci (*capillary apoplexy*), with their consequences, or partial anæmia may result, and consequent death of the anæmic part of the brain (*yellow softening*). Similar occlusion of a large blood-vessel of the extremities may lead to gangrene of the fingers or toes.

Short of such ulceration, the surface of the *endocardium* and *inner arterial tunics,* which have undergone this process of inflammatory exudation in their minute textural elements, presents the white patches, dots, marks, and cicatricial contractions (from partial absorption), known as *atheroma,* or *atheromatous degeneration.*

The inflammation hardly ever affects the whole lining of the heart, usually the lesion is limited to patches of varying size; proceeding, in the first instance, mainly from the valves and textures composing the orifices of the heart. At *post-mortem* examinations, reddening of the texture, the result of imbibition merely of the colouring matter of the blood, must not be mistaken for the redness of inflammation, which it is only possible to see in rare instances (ROKITANSKY). Redness from imbibition is quite superficial, and no individual vessels (*vasa vasorum*) are to be seen; but reddening from inflammation exists also in the deep as well as superficial parts, and the hand lens may show the larger capillaries filled with blood to bursting (FORESTER).

The prolonged existence of the inflammatory state ultimately thickens and hardens, by interstitial deposit, the tissue enclosed between the folds of the serous membrane constituting the valves, so that their action is much impaired. These changes may be limited to the fibrous zone which forms the base of the valves, surrounding the aortic orifice with a sort of collar, contracting its diameter, as well as impeding the play of the valves. In other cases the thickening may affect the free edge, or the central portion of the valve. The most remarkable circumstance, however, connected with chronic adhesive inflammation of the left side of the heart, is the excessive tendency which the valves have beyond all other serous tissues to become cartilaginous or ossified. This transformation commences in the substance of the serous tissue, but more commonly in the tissue connecting the duplicature of the valvular fold. This ossific deposition is not necessarily preceded by a cartilaginous formation, but is most frequently an original abnormal secretion, often containing a good deal of earthy matter. In all the instances in which I have examined such deposits, they did not exhibit the histological appearances of true bone-tissue. It is simply a calcification. The hardening element is deposited in various forms: sometimes in layers, at others in points, and at others in large masses, covering the shapeless valves, in knobs or pyramids, occasionally acquiring a size as large as a pigeon's egg. Sometimes the tendons, or the *chordæ tendineæ* attached to the mitral valve, participate in these indurations, and Corvisart met with one case in which they were entirely ossified. The irritation of these deposits often leads to their destruction, and the whole exudation, softening and breaking down, may mingle with the current of the blood, and produce results of a most serious description, to be afterwards described.

Laceration of the relaxed and softened endocardium sometimes also results. The *chordæ tendineæ* give way very frequently, obviously and suddenly interfering with the tension and function of the valve (see some cases of this kind, with drawings, in the records of the Naval Medical Department for 1870). Sometimes the lappet of a valve, or one surface of it, is torn, or the endocardium giving way over the muscular structure of the heart, the blood may be forced continuously through the rupture so as to tear asunder, more or less, the cardiac muscular fibres, and so produce a *true* or *acute aneurism* of the heart. Some examples of this may be seen in the Museum of the Army Medical Department in the Royal Victoria Hospital at Netley. These aneurisms are circumscribed sacs, varying in size from a filbert to a walnut, in the substance of the heart, bounded at its entrance by a torn and ragged endocardium, its walls formed by the forcibly separated fibres of the muscular substance compressed against the pericardium.

Dr. Latham and Dr. Hope were of opinion that endocarditis is more frequent than pericarditis. Dr. Stokes, Dr. Sibson, and others entertain a different opinion, consistent with the evidence of *post-mortem* inquiries, as recorded by Drs. T. K. Chambers, A. W. Barclay, and Taylor.

The tendency of endocarditis is—

1. To produce those affections of the heart which are also described respectively as "*valvular disease of the heart*"—"*hypertrophy*," and "*dilatation*"—morbid conditions more or less simple or combined.

2. Associated as it often is with pericarditis, and acknowledging rheumatism as a most frequent exciting cause, we have the *muscular* substance of the heart itself sometimes affected, constituting what Sir Thomas Watson terms "*rheumatic carditis.*"

Symptoms and Results of Endocarditis.—A more extensive, forcible, and abrupt impulse of the heart than natural, combined with endocardial murmurs, of a soft, low pitch tone and blowing sound, in a febrile state of the system, and with cardiac uneasiness, are signs suggestive of the probability of endocarditis. A careful study of the development, order of occurrence, and combination of the general symptoms and physical signs, can alone convert that probability into a certainty.

1. **General Symptoms.**—The patient is observed to prefer to lie on his back (*dorsal decubitus*), and he may perhaps incline to toss about with his arms. Pyrexia may prevail of a specific kind, as when *rheumatism, Bright's disease,* or *typhus fever* is present, or it may be *idiopathic inflammatory fever,* associated with the endocarditis. So long as the cardiac orifices are not seriously obstructed, and no obstruction exists in the lungs from pneumonia or bronchitis, no special sensation of dyspnœa is complained of. The pulse ranges in frequency between 80 and 120; and it has been stated by Dr. Taylor even to lose in frequency and energy at the outset of the affection, a result which may be due to the infiltration of the heart's texture with serous fluid. More or less headache may prevail. Generally, however, the patient may not complain, even when special inquiry is made regarding the condition of the heart.

2. **Local Symptoms.**—Discomfort and uneasiness at the heart are most common symptoms, and more or less palpitation may be present.

The extent and power of the impulse of the heart ought now to be examined carefully and repeatedly; and the conditions which tend to subdue or to aggravate these phenomena ought, if possible, to be ascertained. The areas of the heart's dulness, both superficial (in breadth) and deep-seated, undergo increase (BOUILLAUD, SKODA, WALSHE). Its impulse is almost always more extended and stronger than natural at the commencement, till infiltration of tissue takes place, when the pulse and force of the heart become small and soft.

The heart's sounds undergo modifications as soon as the tissue of the valves become changed in texture, substance, and shape, by the inflammation.

The murmurs which accompany purely acute endocarditis are thus arranged in the order of their frequency by Dr. Walshe:—(1.) Aortic obstructive; (2.) Mitral regurgitant; (3.) Aortic regurgitant; (4.) Aortic obstructive and mitral regurgitant together. (The student is referred back to page 527, *et seq.*, for the account of the sites of maximum intensity where these murmurs may be listened to.)

The comparative frequency of aortic and mitral valve disease has been determined with more accuracy by the results of the combined observations of Drs. Barclay, Chambers, and Ormerod, as tabulated by Dr. Sibson. From these records it is seen that the *mitral valves* are more subject to disease than the *aortic;* that the disease is often limited to one set of valves, but that it is more often common to both valves than limited to either. When associated with *acute rheumatism,* the endocarditis affects both valves in the greater number of cases; and the *mitral* more frequently than the *aortic.* In the young, who are subject to *acute rheumatism,* disease of the *mitral* valve, and in the old, who are subject to *atheroma,* disease of the *aortic* valve predominates. In the more severe cases in which the valve disease is itself the cause of death, the *mitral* valve disease is shown to be the most prone to go on to a fatal issue (*Med.-Chir. Review,* Oct., 1854, p. 431).

As there is no difference in character between the murmur of endocarditis and that which attends established valvular disease, it is necessary, in order to appreciate the existence of endocarditis more certainly, that the *murmur should be developed under observation at the early period of an acute attack* (WALSHE): and if a mitral or aortic murmur supervene while a case of *acute rheumatism* is being watched, especially if there be congestion and an expression of anxiety in the face, with distress in the region of the heart, *not caused by pericarditis,* there is a strong probability of endocarditis (SIBSON). The substitution of an abnormal murmur at the apex for the *first* cardiac sound is the most frequent and important sign of endocarditis (NIEMEYER). But the symptoms are often exceedingly insidious in their origin and progress, and the disease is rarely simple, being generally combined with pericarditis; and, moreover, as the *general* constitutional symptoms of these two diseases do not differ, the detection of endocarditis, *per se,* is one of the most difficult in practice. Like pericarditis, it is often latent, as in rheumatic fever, and the practitioner is surprised by his patient showing symptoms of valvular disease after an apparently perfect recovery from fever (STOKES). A murmur, *per se,* is no sufficient evidence of endocarditis. Stokes, Sibson, and Graves have each of them recorded cases where *mitral,* and still more often *aortic* murmurs have been generated, when no valvular disease existed,—in cases of fever, especially in a case of fatty degeneration of the heart, and in a case of pericardial adhesion. These murmurs have also been recorded to exist during life, in cases in which no trace of valve disease was observed after death, by Drs. Barclay, Markham, Chambers, and W. T. Gairdner (Sibson, in *Med.-Chir. Review,* Oct., 1854).

As in pericarditis, it is important to recognize the *friction-sound* pathognomonic of its existence, apart from any endocardial murmur with which it might be confounded; so in endocarditis it is, if possible, still more important to detect endocardial murmurs when masked by pericarditis, for the grazing sounds of the latter disease may altogether mask those of the valve murmurs. The principles on which the diagnosis is to be effected are involved in the facts that friction sounds of pericarditis are limited to the heart's region (STOKES); while, as Dr. Walshe so clearly describes, the sounds of the heart, and the murmurs which attend the lesions of its valves, are propagated in certain determinate directions; and while they are heard in maximum intensity at certain points, more or less defined, they may be detected by following the line of propagation at points beyond the mere limits or region of the heart itself. Upon these grounds data are furnished by which to distinguish the murmurs of endocarditis. (See *ante*, p. 523, *et seq.*)

For this purpose the murmurs of endocarditis must be looked for in suspicious cases from day to day *beyond the region of the heart;* and if a *systolic mitral murmur* is heard extending an inch and a half beyond the nipple, it is most probably due to *mitral regurgitation* (SIBSON). The detection of an *aortic murmur* with pericarditis is much more difficult, because the *friction-sound, frottement*, or " to and fro" sound often mounts to the top of the sternum. The *aortic murmurs* are therefore to be listened to in the line of the natural propagation of the aortic sounds; and if an *aortic murmur* exists, it can only be distinguished in the neck, the best point for examination being just above the sternum, a little to the right, over the *innominate artery.* If, after listening to the first sound, the second sound be observed to follow clearly and distinctly, the chances are that there is no affection of the aortic valves, even if there be a loud systolic murmur. If, however, the second sound be indistinct, inaudible, or prolonged, or be replaced by a diastolic murmur, aortic endocarditis may be suspected or detected (SIBSON). (The student is requested to contrast p. 523, *et seq.*, with these statements.)

As far as the immediate practical value of the information is concerned, it seems to be really unimportant where the exact seat of the murmur is. It is of no practical importance, for example, in the first instance, whether the murmur proceeds from a " mitral, a tricuspid, or a semi-lunar valve, or whether it may be due to a contraction, or a dilatation, an ossification, a permanent patency, or a warty excrescence." The practical points to be determined, in the first instance, reduce themselves to two—namely, *First,* Do the murmurs proceed from an organic cause? *Second,* What is the vital and physical condition of the texture of the heart itself with which they are associated?

For subsequent practical purposes the limits of inquiry may also be very much circumscribed—namely, to the recognition of the occurrence of contraction or of dilatation of the orifice, because both of these conditions are attended with a permanently open state, and permanent valvular disease.

While, therefore, the occurrence of murmurs, and their nature, and the circumstances under which they are developed, are of the utmost importance to establish the existence of an endocarditis, the condition of the muscular substance of the heart must be the great guide in prognosis and treatment. The vital and mechanical state of the heart's cavities must be ascertained. The action of the heart must be carefully noticed at different times, as to whether its force and vigour are above or below the natural standard—whether it is liable to excitement from slight causes—and whether it tends to regular or irregular action, as regards rhythm or frequency of revolutions. An extremely feeble pulse with scanty filling of the arteries implies implication of the texture of the heart. The duration and origin of the disease must be ascertained; and how far the brain, lungs, or liver suffer from the mechanical or vital effects of the lesion (STOKES).

The symptoms, therefore, of acute endocarditis being detected, the immediate treatment of the disease must be proceeded with; and the physician requires also to look before him in anticipation of the results which are likely to ensue if valvular lesion is established, such as hypertrophy and dilatation (which see farther on).

Another class of symptoms and results is apt to be associated with endocarditis and valvular disease—as when products of inflammation are apt to poison or spoil the blood. Rigors, heat of skin, profuse perspiration recurring irregularly, dull, earthy-yellow discoloration of the skin (not of the conjunctivæ), diarrhœa more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, are the symptoms which Dr. Walshe describes as announcing this untoward occurrence. Secondary deposits in the lungs, the liver, or the brain are the records of its morbid anatomy. But, again, these secondary deposits may not be the direct result of existing endocarditis. Virchow, Kirkes, Simpson, and Rokitansky have shown how the fibrinous coagula, which have become permanently attached to the valves, covering the vegetations upon them, may become worn away superficially, and taken into the blood in fine particles, thus leading to secondary coagula, as shown at p. 568, in the capillaries of the spleen and kidneys, to obliteration of these vessels, and in the capillaries of the brain leading to softening, and sometimes to sudden death. The great tendency to the formation of these coagula on the valves, in rheumatic pericarditic attacks, must be specially remembered when it is determined to abstract blood, as the slowness of the heart's action which may ensue greatly favours the tendency to coagulation of the blood and to the deposition of such fibrinous deposits on the valves.

Causes of Endocarditis.—The inner membrane of the heart (exposed as it is to the action of many morbid poisons, and also to many substances which may be taken up by the absorbents and introduced into the circulation) is not found so frequently diseased as we might expect. Of all substances, however, alcohol has the most striking effects on this tissue. It is not only proved to be absorbed and actually to circulate in the blood, but there are few drunkards the inner membrane of whose heart and large vessels is not more or less diseased; so that alcohol probably acts as a specific poison on the endocardium. The morbid conditions associated with Bright's disease, with rheumatism and gout, and with syphilis, appear to act especially upon this tissue, and many who suffer from these diseases often ultimately die of some form of endocarditis often extending along the lining membrane of the aorta.

Prognosis.—So far as the endocarditis is concerned, the immediate prognosis is very similar to that stated under pericarditis; but the future chances of life being prolonged depend upon the lesions which remain permanent. Lesions of the valves are the most common result. The valves at first thickened, gradually begin to shrink, or the *chordæ tendineæ* or edges of the valves adhere, or rupture of some of their textures takes place. After an attack of endocarditis, there may be no defect of valves capable of physical recognition. The valve-texture, however, may be nevertheless damaged, so that shrinking, atrophy, and retraction of the damaged tissue, commencing gradually, progresses very slowly, so that no valvular disease may appear for months after an attack of endocarditis; and any pain in the region of the heart which may have attended such an attack may be quite forgotten. If valvular lesion is fully established, and remains persistent, hypertrophy is certain to follow, and the danger will be the greater in proportion as dilatation of the cavities of the heart predominates over hypertrophy. "Under all circumstances," writes Dr. Walshe, "dilatation is a most serious disease; and the danger increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months. The usual termination of *endocarditis* is death from the resulting lesions of the valves; and it is rare for a patient to die suddenly from endocarditis alone, unless in the course of *Bright's disease.* The fatal results of *endocarditis* do not usually ensue until a lapse of years, when the valves, though acting normally and efficiently for a time, at last begin to undergo changes, and become so deformed that their functions are at last impaired and valvular disease is confirmed. An extremely feeble pulse, with scanty filling of the arteries, is a bad sign in such cases, showing that the muscle texture of the heart itself is implicated. Rigors are also an unfavourable sign; also sudden swelling of spleen, pain in that region, vomiting and albuminuria, or hemiplegia, the signs of true metastasis. If neither aortic lesion, hypertrophy, nor dilatation results, the tissue of the heart itself may be so impaired as to lead to softening, like what occurs in typhoid and typhus fevers, scurvy, or purpura. Fatty degeneration of the cardiac tissue is also a result to be apprehended.

The valuable medical reports of St. George's Hospital, London, prepared by Drs. A. W. Barclay and Rogers, contain the following statistics relative to the *percentage* of mortality *among hospital patients* from *diseases of the heart* during a period of six years—namely, from *pericarditis,* 34·8; from *endocarditis,* 9·19; from *hypertrophy,* 60·5; from *dilatation,* 52·1; from *valvular disease,* 24·5.

Treatment.—From whatever cause arising, endocarditis is one of the most intractable diseases with which we are acquainted. No defined and uniform line of treatment can be laid down suitable for all cases; because, as has been seen, the morbid conditions and circumstances under which endocarditis occur are extremely varied.

What has been written relative to the treatment of *paricarditis* applies equally to acute *endocarditis;* but the management of cases in which the patients suffer from the valvular lesions and their immediate consequences demands the adoption of various lines of treatment, according to the causes or circumstances producing the endocarditis.

When endocarditis seems lapsing into the chronic stage, Dr. Walshe recommends the use of *iodide of potassium* and *liquor potassæ*, combined with bitter tonics.

(CHRONIC) VALVE DISEASE.

LATIN EQ., *Morbis valvarum;* FRENCH EQ., *Maladies des valvules;* GERMAN EQ., *Klappenkrankheiten;* ITALIAN EQ., *Malattie delle valvole.*

Definition.—*Lesions of the valves of the heart, or of its orifices, coming on for the most part insidiously, and which, persisting, induce obstruction or regurgitation, tending to hypertrophy and dilatation of the heart, with congestion of the pulmonary and systemic capillaries, œdema, anasarca, and dropsy.*

Pathology.—The term "chronic valvular disease" is sometimes used in order to distinguish chronic lesions of the valves of the heart from "chronic endocarditis;" for it has been already shown that the valves and orifices of the heart are often damaged by this affection.

But valvular disease does not always owe its origin to endocarditis, and is not always chronic. Such valvular disease is often very insidiously established as a local expression of chronic Bright's disease, or chronic rheumatism, and in gout, as well as in such forms of constitutional derangement of the system as are associated with imperfect nutrition of the body. A very slow deposition of fibrin on the substance of the valves, or degeneration of their structures, is the usual lesion they present. The valves ultimately become thickened, opaque, and puckered, and may be rigid by the presence of atheromatous or calcareous matter. Yielding under pressure, they are apt to become thinner than natural, or they become perforated or cribriform—a condition which may also be referable to congenital deficiency. Sometimes they rupture, and present all the phenomena of ulceration, or their surfaces are irregular and their edges beaded by the deposit of fibrin from the blood. One segment of a valve may be found adherent to another; and in each of these cases the disease may be of such a nature as to obstruct the onward flow of blood, or to permit regurgitation through the diseased valves, when an obstructive or regurgitant murmur will be the result (see *ante,* under "Cardiac Murmurs"). The orifices of the heart may be the parts diseased, while the valves are sound. In such cases the orifices are usually roughened by calcareous or atheromatous matter, or they may be so much dilated that the valves are insufficient to shut them.

The lesions to which valve disease may be referred are of the following varieties:—(a.) *Vegetations;* (b.) *Fibroid thickening;* (c.) *Atheromatous* and *calcareous degeneration;* (d.) *Aneurism;* (e.) *Laceration;* (f.) *Simple dilatation of orifice;* (g.) *Malformations.*

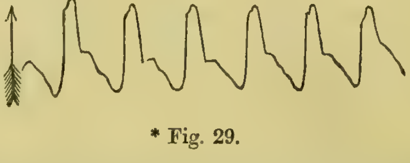
The exact lesion which causes the morbid state of the valves or orifices must generally, however, remain a matter of conjecture; and it is not of so much practical importance to determine the lesion as it is to determine whether it is of such a nature as to cause obstruction to the flow of blood, or to permit of its regurgitation. One or other of these conditions is indicated by a persistent endocardial murmur, and the persistence of such murmur assuredly points to a condition which must lead to cardiac hypertrophy and dilatation. Nevertheless, disease at the various orifices operates very differently the one from the other in many particulars, a resumé of which is here given from the admirable writings of Drs. Fuller, Foster, Gairdner, Hilton Fagge, and Fothergill.

Aortic obstruction, one of the most common forms of valvular disease, has little effect in producing engorgement of the pulmonary capillaries, or general systemic congestion and dropsy; except when it induces dilatation of the left ventricle, and so causes the *mitral valve* to become so inefficient as to permit of regurgitation.

The dilatation of the left ventricle is the turning-point in these cases. So long as the hypertrophy is sufficient to overcome the obstruction at the aortic orifice all goes on well. When the obstruction is no longer compensated for in this way, the ventricle begins to dilate. Each systole leaves a little blood in the left ventricle, the left auricle is no longer able to empty itself completely, and dilates under the accumulated blood, and gradually pulmonary and systemic obstruction begin to appear. Aortic obstruction is the least rapidly fatal form of chronic valvular disease, because its mode of compensation—hypertrophy of left ventricle—is the simplest (FOSTER). It is simply a question of increased power; and as the contracting action of interstitial inflammation of the valves and orifice is very gradual, the muscular hypertrophy can keep pace with the changes. The hypertrophy here is usually, therefore, the most perfectly compensatory, and the compensation lasts longest.

The character of the pulse is not materially altered, except in well-marked cases, when it becomes slower; but when the action of the heart is forcible, and the obstruction is rough, excessive eddying of the blood may be produced, causing a thrill at the base of the heart, and in the track of the aorta and its branches.

The peculiarity seen in the pulse-tracing from the sphygmograph consists in an obliquity or break of the line of ascent, which marks the greater duration of the ventricular systole, and the gradual entry of the blood into the vessels. In the following tracing (fig. 29) from Dr. B. Foster these characters are shown :—



* Fig. 29.

Mitral obstruction, although less common than mitral regurgitation, is, now that its physical signs have been elucidated, recognized much more frequently than formerly. The lesion necessarily induces dilatation of the left auricle; and hypertrophy of this cavity and of the right ventricle more especially, are the means of compensation. This compensating hypertrophy, although it may defer for a long time systemic serous

* Pulse-trace of aortic obstruction (DR. B. FOSTER).

engorgement, cannot prevent a certain amount of pulmonary congestion. Hence, in this form of chronic valvular disease, severe cough, dyspnœa, attacks of cardiac asthma, and severe palpitations are common. Congestion and œdema of the lungs, and not unfrequently pulmonary apoplexy, are the ordinary modes of the approach of death. In some cases, especially in younger patients, the lesion may be tolerated for years. In such cases the compensation effected by the right ventricle is nearly perfect, the pulse remains regular, but soft and of low tension, easily altered in form, or suppressed by any excessive pressure on the spring of the sphygmograph (fig. 30). In these cases the symptoms most complained of are shortness of breath, troublesome cough, and occasional severe palpitations, and great inability for any muscular exertion. In the later and more developed stages of the disease the pulse is markedly irregular, (Fig. 31), and all the general symptoms are worse.

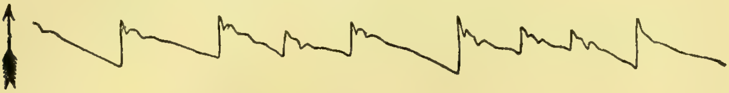


Fig. 30.*

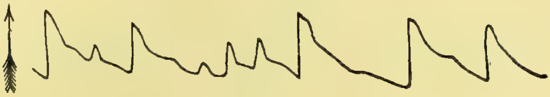


Fig. 31.†

The pathognomonic indication of contraction of the mitral orifice is a "*presystolic*" or "*auricular systolic*" *murmur* (Fauvel, Gairdner, Hilton Fagge).

Of the characters of this murmur Dr. Fagge gives the following account:—

"(1.) The first and most important, although not an essential quality of a direct mitral murmur, is its *place in the cardiac rhythm*. It is '*pre*systolic.' The 'first sound of the heart' is no longer the first audible sign of the heart's waking up from its quiescence during the pause; it is *preceded* by the morbid sound or bruit. The relation can probably be expressed in no way so well as by a diagram, after the manner first suggested by Dr. Gairdner (see fig. 32).

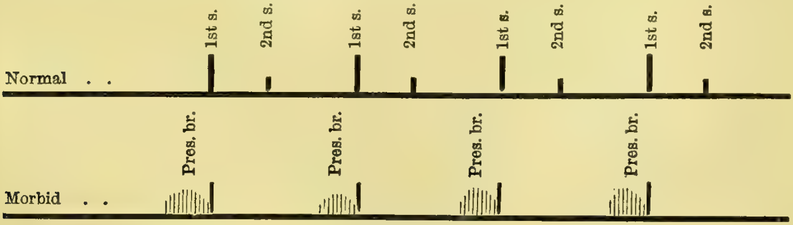


Fig. 32.‡

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* Pulse-tracing of mitral obstruction (Dr. B. Foster).

† Pulse-tracing of mitral obstruction (developed stage), in which the orifice only admitted the top of the little finger (Dr. B. Foster).

‡ Diagram to show position of "*presystolic*" or "*auricular systolic*" murmur (Gairdner, Fagge).

"(2.) The direct mitral murmur has a special *seat.* It is loudest over the apex of the heart, and is generally confined to the region of the apex. It is inaudible or scarcely audible at the base, and is very rarely carried round the axilla to the back. If the stethoscope be placed over the third left costal cartilage the natural cardiac sounds are perceived (unless there be co-existing aortic disease).

"(3.) The *quality* of a direct mitral murmur is, in most cases, peculiar. Almost all those who have written on this subject have remarked that the 'presystolic bruit' has a *rough, churning* (or, as Dr. Salter calls it, '*grinding*') character, which of itself enables the auscultator to suspect its nature and origin.

"It is frequently accompanied by a markedly palpable thrill, or '*frémissement cataire*'" (*Guy's Hospital Reports*, xvi., 3rd Series, 1871).

Obstruction of the pulmonary orifice is a very rare lesion. It leads to hypertrophy and dilatation of the right ventricle, and ultimately to regurgitation through the tricuspid orifice, with turgescence and pulsation in the large veins of the neck.

Tricuspid orifice obstruction is very rare; and leads to hypertrophy and dilatation of the right auricle, with excessive congestion of the venous system, unaccompanied by any visible pulsation in the neck.

Aortic regurgitation, from incompetency of the sigmoid valves, is one of the most common forms of chronic valvular diseases. The heart finds compensation for the valvular imperfection in hypertrophy and dilatation of the left ventricle. By these changes an extra quantity of blood is thrown into the aorta at each systole, and the regurgitation of a portion of this extra charge can consequently take place without materially disordering the normal balance between the arterial and venous blood. For in aortic regurgitation, as in all other valvular lesions, the tendency is to empty the arteries and overfill the veins. The occurrence of too great dilatation of the left ventricle and degeneration of its muscle is, however, almost inevitable; for, as the coronary arteries are mainly filled in health during the diastole of the ventricle by the rebound of blood from the closed aortic valves, insufficiency of these valves has a direct influence in impeding the coronary circulation and diminishing the blood supply to the hypertrophied muscle: hence gradual degeneration of the walls and dilatation of the cavity follow. After a time, the mitral valve becomes incompetent, and the pulmonary and systemic circulation become rapidly embarrassed. It is in this affection we meet with the true "*cor bovinum*"—an excessive enlargement of the left ventricle, both in thickness and cubic capacity of the cavity (Fothergill, Foster).

Aortic regurgitation is accompanied by a peculiar and very characteristic pulse-beat. The prolonged swell imparted to the blood at each systole is not sustained by the perfect closure of the valves. The waves of blood are short and abrupt; the pulse jerks and leaps, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. These peculiarities are consequences of the low arterial tension which the regurgitation of blood into the ventricle during its diastole produces. The traces of aortic patency given by the sphygmograph are marked by abnormally great amplitude. The vertical line of ascent marks the sudden ventricular contraction; and is often abruptly terminated by a sharp pointed process (fig. 33, DR. FOSTER). The summit of the pulsation-trace is in many cases very short; but in others it presents a horizontal or curved line, especially if any constriction of the aortic orifice exists, or other cause producing delay of the passage of the blood into the vessels.

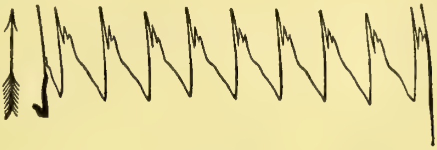

Fig. 33.*

The distinctive features of aortic regurgitation are, however, to be found in the line of descent. There are often one or more extra secondary waves. The first secondary wave occurs earlier than in the normal trace, and the notch which precedes it is often very much exaggerated. But the main characteristic *is the suddenness of the fall of the line of descent, and the comparatively small size of the second secondary wave or true dicrotism* (DR. FOSTER).

Dr. Sanderson gives a somewhat different explanation of the same lesion and pulse-tracing (*Brit. Med. Journal*, July 20, 1867, p. 40). "Fig. 34 represents the pulse of a middle-aged man, who had acute rheumatism eight years ago, and several times since. When the tracing was taken he was suffering from extreme orthopnœa and præcordial pain. The impulse of the heart was to the left of the mammary line, and occupied a space as large as the palm of one's hand. A loud diastolic *bruit* was heard at the fourth cartilage, and a systolic bellows-sound over the aorta. Posteriorly, there was dulness at both bases, and abundant subcrepitant *râles* in inspiration. There could, therefore, be no doubt as to the nature of the case, which soon terminated fatally. After death, it was found that the aortic valve was so altered that the most copious regurgitation of blood must have taken place during each diastolic period. In this case the pulse exhibits characters which Dr. Sanderson believes are not met with excepting in connection with aortic incompetence. These do not consist, as is often supposed, in the unusual verticality of the expansion; for, as has been already seen, this peculiarity may be produced by merely functional disorder. The distinctive peculiarity consists in the collapse. The tracing shows that the artery becomes completely emptied during the interval between each beat and its successor; so that the diastolic expansion is no longer indicated. The explanation is simple. Immediately after the heart has ceased to contract, the blood injected into the

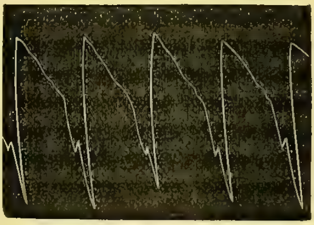

Fig. 34.†

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* Pulse-tracing of aortic regurgitation (DR. B. FOSTER).
† Pulse of aortic regurgitation.—H. R., aged 36.

aorta rushes back into the relaxed ventricle; so that, although the arterial equilibrium is for a moment disturbed, it is almost immediately re-established, the excess of pressure in the great arteries being at once relieved. In other words, the elastic force, which is naturally expended in producing what is called the diastolic expansion, is wasted in regurgitation."

Mitral regurgitation is the most common of all forms of chronic valvular disease, and depends for compensation mainly on hypertrophy of the right ventricle. Hypertrophy of the left ventricle and dilatation of the left auricle also occur, leading to systolic auricular impulse at the second intercostal space, by admitting of the transmission of the impulse from the ventricle. When excessive, it causes a vibration, a thrill, or a purring tremor, perceptible on the chest-walls in the region of the heart, but which is not transmitted to any extent along the aorta or great vessels. The pulse-tracing is seen to differ from the normal dotted line principally in its great frequency, and in the depth and amplitude of the diastolic notch (fig. 35). This pulse closely resembles the undulating pulse of typhus (fig. 36). In the one disease the contractile force is *weakened*, in the other it is *wasted*. The effect is the same: the systole is ineffectual (SANDERSON).

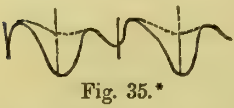

Fig. 35.*

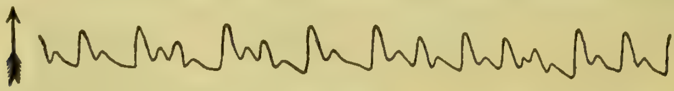

Fig. 36†.

When the regurgitation is also combined with obstruction, from contraction of the auriculo-ventricular orifice, the left auricle usually becomes hypertrophied as well as dilated; and this additional lesion always diminishes the irregularity of the pulse characteristic of pure mitral regurgitation; and the tracing from the sphygmograph is as follows (figs. 37 and 38, B. FOSTER):—

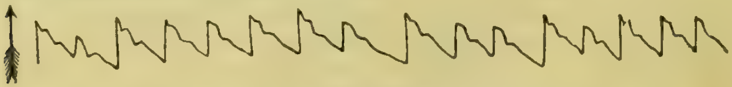

Fig. 37.‡

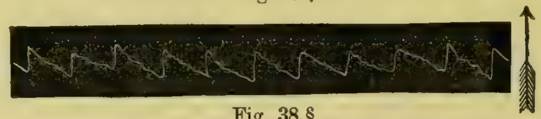

Fig. 38.§

Fig. 39 shows a pulse often seen in rheumatic valvular disease, with large mitral regurgitation. The heart acts very irregularly. At times the ventricle contracts effectually; but at other times the systolic expansion of the arteries is imperfect and abbreviated, in consequence of which the mean arterial tension declines. Whenever this is the case, the pulse assumes a form which is as distinctly dicrotic as that of typhus; so much so, indeed, that its double character can be readily recognized by the finger (Dr. Sanderson).

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* Dicrotic feeble pulse of mitral regurgitation.

† Typical mitral regurgitant pulse trace (DR. B. FOSTER).

‡ Less irregular form of mitral regurgitant pulse-trace when some obstruction is associated with the regurgitation (DR. B. FOSTER).

§ Irregular pulse of pure mitral regurgitation (FOSTER).

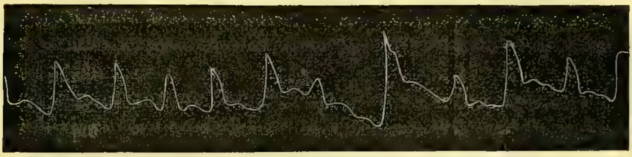

Fig. 39.*

Mitral regurgitation primarily interferes with the circulation through the lungs, producing cough, dyspnœa, and other symptoms of pulmonary congestion; and pulmonary apoplexy is common. The pulse is characteristic. It is irregular in rhythm, and unequal in force and fulness.

Regurgitation through the pulmonary orifice is so rare that its effects have scarcely been verified by clinical observation.

Regurgitation through the tricuspid orifice, although rare as a primary disease, yet is not uncommon as a consequence of dilatation of the right ventricle. The right ventricle then becomes hypertrophied, the right auricle dilated, the *venæ cavæ* distended, and there is a strong tendency to congestion of the systemic and cerebral capillary circulation.

Symptoms.—The symptoms produced by chronic valvular disease mainly depend on the impediment offered to the pulmonic and systemic capillary circulation. In the more advanced stages all forms of valvular mischief result in a certain amount of capillary engorgement; and therefore all forms of chronic valvular disease have many symptoms in common. Oppression at the chest, breathlessness, speedy exhaustion on exertion, a general sense of lassitude, headache, restless and disturbed sleep, with frequent starting and frightful dreams, cough, palpitation, dropsy, occasional pain in the region of the heart, and sometimes severe *angina,* are amongst the earlier phenomena. The cough is due to pulmonary engorgement, and varies with the amount of *mitral* obstruction or regurgitation. Dyspnœa, while it is an early and constant symptom of mitral lesions, is, on the other hand, frequently absent in affections of the aortic valves until the later stages. The mechanism of compensation in the two cases explains this; for, while in mitral affections this compensation depends on the right ventricle, and thus entails a certain amount of pulmonary engorgement, in aortic affections it depends on changes in the left ventricle, and consequently the lungs escape (Foster). On the other hand, the headache, restlessness, disturbed sleep, and dropsy, are proportioned to the amount of systemic capillary congestion, and vary with the extent of tricuspid obstruction or regurgitation. Albuminuria is not an uncommon occurrence in the later stages; it is generally preceded by scanty urine, of a high colour, high in gravity, and loaded with urates. The scanty urine indicates the low arterial tension, causing insufficient pressure in the capillaries of the malpighian bodies, and the albumen, which is afterwards superadded, points to the venous engorgement of the kidney. In the characters mentioned above, the urine of cardial albuminuria differs from that depending on chronic renal disease.

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* Pulse of mitral valve regurgitation (Sanderson).

The dyspnœa of cardiac disease is peculiar and characteristic, as the late Dr. Hyde Salter has ably shown:—It is rather a breathlessness than a difficulty of breathing. It has a panting, gasping character. Oppression, rather than tightness, is complained of, and there is a strangling, choking-throat feeling about it. The breathing is always accelerated. The dyspnœa is extremely intolerant of movement, or of any exertion whatever, and is often the only circumstance under which dyspnœa is felt. As long as the patient remains at rest there may not be the slightest appearance of dyspnœa, but the moment any exertion is made the breath is gone. The dyspnœa of heart disease is also intolerant of the recumbent posture—hence the name "*orthopnœa*," which signifies "upright breathing"—the patient being compelled to sit erect in order to breathe. In extremely severe cases the patient may not lie down for many days and nights; and should he momentarily fall off into a dose, he is instantly awoke by a sense of impending suffocation, and is in a death-struggle for breath. No suffering can compare with this, and it is not wonderful that the sufferer longs for the sleep of death. "It were indeed a sad story," writes Dr. Ormerod in his admirable Gulstonian lectures, "to tell how patients with disease of the heart die—the tragedies, so to say, of the medical wards of our large hospitals. . . . How some, wrung with pain, have struggled in the week-long agony of death. How some have for days together fixed themselves in the most fantastic postures, the only way in which they could find relief; some leaning forward, resting their hands on a stool, to catch a few minutes' sleep; some on their hands and knees, till the approach of death, blunting their sensations, allowed them to lie down —a sure sign of coming dissolution."

The dropsy of cardiac disease is usually a late occurrence. It makes its appearance as *œdema* or *anasarca*, very partial and slight at first—a puffiness merely of the eyelids, or more frequently slight œdema of the ankles. By slow degrees it ascends towards the trunk, and ultimately involves the upper extremities and the face; the scrotum in men and the labia in females becoming enormously swollen. Towards the close, effusion is apt to occur into one or both pleura, but ascites is not common (BELLINGHAM). The first appearance of the dropsy, as Dr. Latham observes, marks an eventful period in the progress of cardiac disease. It indicates that a new law takes effect in the circulation, and gains the mastery over the law of health, which has hitherto been able to retain the watery elements of the blood within the blood-vessels. Now, the serum escapes from the blood-vessels, and accumulates in the areolar tissue of the body.

The forms of valvular disease, in the order of the frequency in which dropsy is met with, are,—(1.) Considerable contraction of the left auriculo-ventricular orifice; (2.) Dilatation of the right auriculo-ventricular orifice, with hypertrophy and dilatation of the right ventricle; (3.) A state of the mitral valve and orifice permitting free regurgitation;

(4.) Considerable contraction of the aortic orifice. As a general rule, it supervenes earlier, the earlier that general venous congestion ensues. A varicose condition of the blood-vessels in the air-vesicles of the lungs is very soon established, which essentially aggravates the dyspnœa.

Prognosis.—It is most unfavourable in cases of mitral and tricuspid regurgitation, and least so in cases of aortic obstruction; and, generally, it may be said that the form of disease which is most rapidly fatal is that which is most rapidly productive of systemic or pulmonic capillary congestion. If the heart be healthy at the date of the occurrence of valvular disease, if the extent of the lesions be not excessive, if the blood be of a normal character, if the viscera be healthy and the secretions free, if the patient's mode of life be regular, temperate, and sedentary, life may be prolonged; but if the heart be hypertrophied and dilated, and if these conditions progress, and if the blood be spanæmic, and the lungs or liver unsound, if the secretions be irregular or defective, or if the patient leads a laborious life, the disease will run a comparatively rapid course, proving fatal probably within two or three years. In either case death is apt to occur suddenly from syncope. This is especially true of aortic regurgitation. In mitral valve lesions the approach of death is more commonly through pulmonary complications and general dropsy. The occurrence of albuminuria is a prognostic sign of great value, as it shows that the general venous stasis has so affected the small vessels of the kidney as to produce a local dropsy, and hence the albumen in the urine. The albuminuria marks the thorough development of the systemic venous engorgement, and the prognosis is generally the worse the longer this symptom has existed, or the more frequently it has occurred.

Treatment.—All the bad symptoms in valvular affections arise from defective compensation; and as the compensation in all cases depends on the integrity of the heart-muscle, the maintenance of its nutrition is the first great indication. The lesions themselves being incurable, palliative treatment is necessarily directed to aid the mechanism by which the ill effects of the valvular incompetency are met.

In affections of the aortic valves, when the heart-muscle is well nourished, an almost perfect compensation may be maintained for years. In such cases a nutritious (albuminous) diet, with fluids in small quantity—tonics, especially preparations of iron—are indicated. In the earlier stages a moderate and steady amount of exercise is beneficial.

In *mitral* affections, on the other hand, the compensation can never be so complete as to do away with the tendency to pulmonary complications. In these cases, therefore, while the same nutritious diet and tonics are indicated to promote the nutrition of the cardiac muscle, exercise must be taken more cautiously; the object being to regulate and moderate the action of the heart, controlling the tendency to local congestion, and mitigating or removing the symptoms which result from the cardiac derangement. If the patient be plethoric, the heart's action tumultuous, and its impulse forcible, cupping between the shoulders may afford immediate relief to palpitation and oppression at the chest. Repeated or profuse venesection is dangerous, and is calculated to excite irritability of the heart, to impoverish the blood, and to induce dropsy.

Repetitions of blood-letting ought therefore to be effected by the application of a few leeches to the *præcordial* region, and very general relief will be obtained by hydragogue purgatives, aided by dry cupping, mustard poultices, and turpentine fomentations. When, however, active congestion of the lungs exists, venesection, cupping, blisters, and sinapisms are required.

When the heart's action is tumultuous and irregular, one of our most powerful remedies is *digitalis.* As a general rule, however, the drug is only useful in mitral affections, and is contra-indicated in aortic valve disease, except in cases in which the hypertrophy has exceeded the limits of compensation, and become the chief cause of the symptoms.

Digitalis slows the pulse and increases its tension, while strengthening and regulating the heart's action. The slowing of the pulse increases the period of ventricular diastole—that is to say, the period during which regurgitation takes place when the aortic valves are incompetent; consequently digitalis does harm in this lesion by augmenting the regurgitation.

In mitral obstruction it is, on the contrary, most valuable. In this condition the rapid, irregular pulse tells of the varying quantities on which the ventricle contracts. Sometimes the interval between the ventricular contraction is so short, and the ventricular charge is so small, that the systolic wave does not reach the wrist. Under these circumstances the auricle must have more time to fill the ventricle. This, to say nothing of the increased power given to the cardiac muscle, is exactly what digitalis effects. By slowing the action of the heart, the period of time during which the blood from the distended auricle can flow into the ventricle is increased; and as the extra time allows more blood to pass through the narrowed mitral orifice before the final effort of the auricle is made, that effort is made on a smaller quantity of blood, and is consequently more effective; for the smaller the quantity of blood which the auricular muscle has to push before it, the greater will be the velocity given to the current. The ventricle, though contracting less frequently, contracts more effectually. Instead of eighty or ninety irregular contractions per minute—no two succeeding ones equal in force, and some so valueless that they are not perceptible at the wrist—we get some sixty steady equal beats. The pulse grows in force, fulness, and regularity; the arterial tension rises; the pulmonary congestion diminishes; the kidneys, before inactive, wake up to their work; and the advancing dropsy recognizes its master, and beats a sullen retreat (Foster, *Med.-Chir. Rev.*, July, 1871).

Digitalis is most useful in mitral regurgitation also; for, to keep up the compensation, there must be good steady muscular action, no wavering in the contraction, no inefficiency in the sphincter-like narrowing of the auriculo-ventricular orifice, and no unsteadiness in the action of the papillary muscles. Any extra effort soon disturbs the artificial equilibrium, and confused muscular action follows. It is this which digitalis corrects. In place of a large number of ineffective contractions, it concentrates the power of the ventricle on a smaller number of well-directed steady beats, each throwing a larger charge of blood into the arteries, and so diminishing, beat by beat, the over-distension of the right heart. The right ventricle so aided, is also aiding, by the more vigorous efforts which the digitalis enables it to make. In this form of disease all turns on the healthiness of the cardiac muscle; the remedy will do no good—nay, rather will do much harm—if muscular degeneration has occurred.

Digitalis should not be given when there is fatty degeneration of the cardiac muscle. The best test of its beneficial action is the quantity of the urine. As long as the flow of urine increases or keeps up to a maximum, which the digitalis has produced, the drug is acting beneficially. The diuresis is the outward and visible sign of its beneficial action, and indicates a restoration of the normal balance between the contents of the arteries and the veins, an increased arterial tension, and consequently a refilling, under pressure, of the empty capillaries of the Malpighian bodies. The high-coloured scanty urine, loaded with urates, is replaced by a clear and copious stream, which tells of a steadily beating heart and a firmer pulse (FOSTER.)

The action of the heart is also markedly influenced by the internal administration of *veratrum viride*. The dose of *the tincture* is from *five* to *twenty minims*, which should be gradually increased from the smaller to the greater dose, till some obvious effects are produced. If the pulse is reduced, or nausea occurs, no increase of the dose should be made; and if vomiting occur, it should be suspended; and when resumed, the dose should be diminished. *Veratria* is to be given in doses of from *one-sixteenth* of a grain to *half a grain*. When the pulse is sufficiently reduced, the doses should be diminished one-half. *Morphine* or *laudanum*, with brandy, is an antidote for an overdose of this *veratria*, which is an exceedingly powerful remedy as a cardiac sedative, and requires to be used with great caution, the patient being constantly watched. Its depressing effects on the circulation and nervous system are often very striking—a pulse of 75 or 80 being subdued in the course of a few hours to 35 or 40 (*Record of Pharmacy and Therapeutics*, No. v., p. 35, J. C. Braithwaite). *Aconite and hyoscyamus* are also remedies which exert a sedative influence over the heart. Aconite is chiefly useful in excessive hypertrophy. *Hydrocyanic* acid and *caffeine* are also good remedies in such cases.

Sleeplessness being one of the most distressing symptoms, opium in some form might be considered advantageous; but, as the late Dr. Hyde Salter justly observes, "To give sedatives in such a case would be the refinement of cruelty. What keeps this poor man awake is not a want of tendency to sleep, but a condition that makes sleep impossible. Relieve him of his orthopnœa and he would be asleep in ten seconds, and so dead asleep that it would take a great deal to rouse him, like a half-asphyxiated child on whom tracheotomy had just been performed. His great struggle, as it is, is the struggle between sleep and life; with opium thrown into the scale of sleep, the struggle for life would only be so much the harder. In one way, and only in one way, would opium give him ease: the narcotic of opium, added to the narcotic of the carbonic acid already circulating in his veins, might accelerate by some hours, or even days, the final coma, and make him sooner sleep the sleep of death. But the euthanasia that is purchased by anticipating the natural process of death comes very near to homicide, and is an alternative that few would adopt" (*Brit. Med. Journal*, Feb. 8, 1862).

It is a very interesting fact, first pointed out by Dr. Clifford Allbutt, that morphia given hypodermically acts like a charm in relieving the dyspnœa and sleeplessness of valvular affections. It is most useful in mitral valve disease, but may be administered in the other forms. The dose to commence with is one-eighth to one-sixth grain of the *bimeconate* of morphia. In one instance of mitral obstruction in which there was general dropsy, pulmonary engorgement, albuminuria, with sleeplessness and orthopnœa, which had lasted for weeks, the first dose gave the patient some hours of gentle sleep. In this case the hypodermic injection of morphia was continued in gradually increasing doses for over twelve months—the patient having his dose several times weekly at 5 P.M., and sleeping quietly till 4 A.M. The remedy, as the patient expressed it, " robbed his disease of its worst terrors, and not only prolonged, but rendered life tolerable and even pleasant." Aided by the morphia, the other remedies began to act, and all the worst symptoms were gradually subdued.

Careful regulation of the patient's mode of life is above all things necessary in chronic valvular disease. Excitement of all kinds must be avoided; and the diet should be light, nutritious, and of moderate quantity; the clothing warm, and cold should be carefully avoided, especially by those affected with mitral disease.

When anasarca supervenes, the hydrogogue cathartics are required to insure copious watery discharges from the bowels. *Electuary* of *bitartrate* of *potash*, *gamboge*, *elaterium*, *podophyllin*, and *compound jalap powder* should be given on alternate days. Stimulants also may be required, the most suitable being Hollands gin, or whisky.

(c) *Diseases of the Muscular Structure of the Heart.*

MYOCARDITIS.

LATIN EQ., *Myocarditis;* FRENCH EQ., *Myocarditê;* GERMAN EQ., *Myocarditis;* ITALIAN EQ., *Miocarditide.*

Definition.—*An inflammation of the muscular structure of the heart (extremely rare as an idiopathic disease), which becomes softened, flabby, and finally degenerates.*

Pathology.—Our knowledge of the anatomy as well as the pathology of the heart and large blood-vessels may be said to begin with Harvey; but the subject can hardly be said to have taken a scientific form till the beginning of the present century, when the work of Corvisart appeared, followed by those of Burns in England, of Testa in Italy, of Kreysig in Germany, and by the works of Bertin, and more especially of Laennec, Bouillaud, Senac, and Collin in France. A large school has since been formed in Europe by the labours of these eminent pathologists.

Nineteen years ago Dr. Williams and Dr. Hope, in their respective treatises, showed how it was possible to make an accurate and minute diagnosis in almost every case of cardiac disease; and the subsequent labours of Stokes, Graves, Bellingham, Forbes, Walshe, Davis, Sibson, Latham, Fuller, and Gairdner, in this country, have brought the pathology of diseases of the heart and lungs to their present advanced state of perfection.

Although *myocarditis* is rare as an idiopathic affection, still it does occur not seldom as a consequence of *acute rheumatism* or of *pyæmia.* It is also a condition generally associated with *endocarditis* or *pericarditis,* or both; and its effects are especially obvious in the strata of fibres nearest the inflamed membranes. It is accompanied by *proliferation* of the connective tissue of the sarcolemma, and absorption of the primitive fasciculi, leading to cicatricial-like contraction of the affected parts. *Post-mortem* signs of its existence are also to be found in cases of valvular disease of the heart resulting from endocarditis. Thus it is most frequently seen in circumscribed spots, as cicatrix-like scars of portions of the cardiac walls. When portions of a large extent are involved, the result may take the form of an abscess, or give an opportunity for the formation of an aneurismal dilatation. Syphilis is sometimes a cause of the disease.

Morbid Anatomy.—The left ventricle, especially towards its apex or in the septum of the heart, just below the aorta, is the most commonly affected parts. The papillary muscles are also often affected (NIEMEYER, DITTRICH).

The lesion is recognized chiefly by discoloration of the muscular fibres, which assume a greyish colour, and are softened. Microscopically, the transverse and longitudinal striation characteristic of the heart's muscles have disappeared, and the fibrillæ are generally broken down into a finely granular ditritus, with fat globules and granules. When this condition has passed away, a cicatrix-like scar remains, which ramifies in various directions as the inflammation did, of which it represents the effect.

Indurations of the walls of the heart may be thus also explained as an occasional result of *myocarditis.* Bouillaud has collected a series of cases in which this change of structure has been observed. In one, the walls of the heart were almost tendinous. In another, the *carneæ columnæ* of the ventricle were so increased in density as to split. In a third, the walls of the right ventricle seemed to be undergoing a cartilaginous transformation; and Broussais has seen them as hard as a cocoa-nut. Chronic congestion of the heart leads to this condition, and then to defective contractility, leading to dilatation, (Jenner, *Med.-Chir. Trans.,* vol. xliii.) The more usual mode of induration is by ossification—a change which usually begins in the coronary arteries, and frequently stops there; but in some rare cases this ossification extends so that the walls of the auricles, of the ventricles, or of both, and sometimes also of the cardiac partition, become converted into bone. There are specimens in the museum of St. Thomas's Hospital, and many other hospital museums, which make it remarkable how life could have been continued, looking to the unyielding nature and great extent of the ossification of the walls of the heart and great vessels.

Symptoms and Course.—Few authors have met with a case of myocarditis unless complicated with pericarditis, and no distinction has hitherto been observed between the symptoms of these two diseases. Corvisart says it is impossible to distinguish between these affections. M. Laennec affords us no assistance in this dilemma, for he considered that no incontestable example of carditis existed; while Bouillaud says he knows of no symptom which is especially characteristic of carditis (FULLER). It is therefore not to be diagnosed during life, except as a matter of probability, in connection with *rheumatism*, tedious and malignant *scarlet fever, septicæmia, valvular disease, endocarditis, pyæmia, embolism*, or *syphilis;* especially if rigors set in, with swelling of the spleen, vomiting, or pain in the region of the kidneys, and albumen or blood in the urine.

Treatment, under such circumstances, cannot be indicated beyond what has been stated in endocarditis.

ABSCESS OF THE HEART.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abscès;* GERMAN EQ., *Abscess;* ITALIAN EQ., *Ascesso.*

Definition.—*A collection of yellow purulent liquid, surrounded by the softened and discoloured muscular substance of the heart.*

Pathology.—M. Simonet has recorded a case of *suppuration* of the heart in which the disease appeared to result from rheumatism. The patient, a woman, was brought to the hospital labouring under most tumultuous action of the heart, with a pulse irregular and contracted, her breathing oppressed, and her extremities cold. She was bled, but died in a few hours in a fit of syncope. Several purulent collections were found in the substance of the heart, and especially in the interventricular partition. The internal surfaces of the cavities were red in several places; the muscular structure was of a yellowish hue, softened, and easily torn with the least effort.

Dr. Graves was once consulted by a gentleman, fifty-five years of age, complaining of palpitation, dyspnœa, and finally anasarca. He suffered from severe pain and oppression at the region of the heart. Hypertrophy and dilatation of the ventricles were detected. The patient died suddenly a few weeks afterwards; and, besides the hypertrophy and dilatation, an *abscess* was found in the walls of the heart which contained about two ounces of pus.

Another case in proof of suppurative inflammation taking place in the heart is one that was examined by the late Mr. Stanley. In this instance the vessels were loaded with venous blood, and the muscular fibres were of a very dark colour, of a very soft and loose texture, and easily torn by the fingers. On a section of the ventricles, numerous collections of dark-coloured pus were seen among the muscular fasciculi. Some of these were seated near to the cavity of the ventricle, while others were more superficial, and had detached the pericardium from the heart. The muscular parietes were softened, and loaded with dark blood.

Suppuration of the muscular substance of the heart and of the coronary artery, in a case of pyæmia, is recorded in the sixth volume of the *Pathological Society's Transactions*, p. 151.

Such abscesses rarely become encapsuled or dry up; but perforation nearly always takes place, unless death occurs beforehand (NIEMEYER). The results vary according to the direction in which the abscess points and bursts. If into the pericardium, *pericarditis* will follow; if into the cavity of the heart, the *debris* of the abscess will pass into the circulation, and may lead to the phenomena of *embolism* as metastatic abscesses; or the abscess opening in the septum, communication between the two sides of the heart may be established; the whole substance of the heart's wall may rupture; or, the insertion of a valve may be torn away.

Ulceration of the heart has been occasionally seen, from an abscess in the walls of the heart having opened either into one of its cavities or into that of the pericardium. It has also resulted from the softening of a cancerous tumor, or from a suppurating tubercle. Cloquet has given the case of a man, aged seventy-nine, subject to frequent syncope, who died suddenly, in whose heart there was an ulceration of the left auricle, through which about two pints of blood had escaped into the pericardium. No means exist of ascertaining the existence of such conditions during life; suspicion may be awakened by a feebler impulse, a slower beat, and greater dulness of the sounds of the heart than normal. Patients suffering from this affection are usually hypochondriacal, liable to palpitation on the least exertion, and often die from the ventricle rupturing.

HYPERTROPHY.

LATIN EQ., *Hypertrophia;* FRENCH EQ., *Hypertrophie;* GERMAN EQ., *Hypertrophie;* ITALIAN EQ., *Ipertrofia.*

Definition.—*An abnormal growth of the muscular substance of the heart, increasing its volume by thickening of the cardiac walls.*

Pathology and Morbid Anatomy.—The bulk or volume of the normal heart I have found to range from 12·5 cubic inches to 19·8 cubic inches.

The anatomical description of the condition known as hypertrophy is in reality a compromise between two opinions. By some it is regarded as a multiplication or increase in the number of the muscular fibres or primitive fasciculi (FORESTER)—an *Hyperplasia* (see vol. i., p. 88). By others the condition is due to a thickening of existing fibres—an increase in bulk of the primitive muscular bundles—a true *hypertrophy* (BAMBERGER, ROKITANSKY). Very rarely an idiopathic disease—for "spontaneous uncalled-for hypertrophy" is less and less believed in—it is more commonly a secondary affection; and, in general terms, hypertrophy of the heart is always a compensating growth to overcome some obstacle, except, perhaps, when it may be the result of plethora. The hypertrophy may be general or partial—that is, may affect the whole heart, or one side of the heart, or one ventricle, or one auricle, or the ventricle of one side and the auricle of the other, or both ventricles or both auricles, or, indeed, every possible combination of the four cavities. The auricles, however, are much less frequently affected than the ventricles.

Two forms are recognized by the College of Physicians, namely:—

(a.) *Of the left side.*

(b.) *Of the right side.*

A hypertrophied heart may weigh from sixteen to thirty-two, or even over forty ounces.

The natural thickness of the walls of the left ventricle is in the adult male from *five* to *six and a half lines;* the female *four and a half lines.* The thickness of the right ventricle in the male measures *two lines,* in females *one and two-third lines.* The right auricular wall *one to two and three-fourth lines;* the left *a line and a half* (BIGOT). But Laennec has seen them, in cases of hypertrophy, to measure an inch and a half, or *eighteen lines,* in thickness at the base, or triple the healthy standard. An hypertrophy of *fourteen lines* in the left ventricle and *seven lines* in the right may be attained; and the increase seems most marked in the external layers of fibers and *columnæ carneæ.* This thickness generally diminishes towards the apex, which latter is often natural; but in other cases even the apex is thickened, and instead of *two lines* it may measure *four. The columnæ carneæ,* and likewise the cardiac partition, are proportionably hypertrophied in these cases.

In hypertrophy of the right ventricle the walls are more uniformly thickened than in hypertrophy of the left ventricle: still, however, the increased thickness is always more marked about the tricuspid valves, and at the origin of the pulmonary artery. The greatest thickness observed has been seldom more than four or five lines, which, taking the natural thickness at two and three-quarter lines, is scarcely a twofold increase. In malformations of the heart, however, it has been found much greater; and both Bertin and Louis have each seen a case in which the *foramen ovale* was open, and in which the thickness varied from twelve to sixteen lines. Besides an increase of thickness, the walls of the right ventricle, when hypertrophied, acquire a greater firmness, so that on cutting through the walls they do not collapse.

The increase in volume is sometimes most marked in the muscular walls of the left ventricle, and sometimes in the trabeculæ and papillary muscles of the right ventricle.

These forms of hypertrophy are distinguished mainly by the *capacity of the chambers* of the heart, which accompanies the hypertrophy, namely:—

(*a.*) Where the capacity of the cavities is normal—*i. e.,* retain their relative capacities to each other, although the walls are increased in thickness—*simple hypertrophy.*

(*b.*) Where the cavities enlarge or dilate, and the muscular walls thicken—i. e., *eccentric hypertrophy.*

(*c.*) Where the capacity of the cavities is diminished and the walls thicken—i. e., *concentric hypertrophy.*

The capacity of the cavities may be enlarged during life; but the hypertrophy of the heart may be so great, and the contraction at or after death so powerful and energetic, that all dilatation has disappeared. This is especially apt to be the case in the hypertrophy of the heart, which complicates *Bright's disease.*

Hypertrophy of the heart, however, seldom takes place without an alteration in the size and form of the chambers. These may, indeed, be natural, but more commonly they are increased; so that supposing the chamber of the natural size to hold two ounces, when thus diseased it will often contain the larger portion of a pint. This state of parts has been termed *eccentric* hypertrophy; and admitting the normal heart to weigh from eight to eleven ounces, according to age, the weight in this form of hypertrophy is often double or triple that amount; and Bouillaud speaks of eighteen, twenty, and twenty-two ounces being not uncommon; and Peacock records even more than forty ounces—the ox's heart—the *cor bovinum.* On the contrary, hypertrophy sometimes takes place *concentrically*, or at the expense of the cavity of the heart, and from this cause the ventricle has been found so reduced in size as to be not larger than an unshelled almond. This form—namely, *concentric* hypertrophy—is exceedingly rare, except as a congenital malformation; and its existence is doubted by Cruveilhier, and disproved by Dr. Budd as a result of disease; while, on the other hand, Rokitansky and Bamberger believe that, although rare, it sometimes does occur. A normal heart very strongly contracted at the moment of death has often been mistaken and described as an instance of concentric hypertrophy.

An analysis of ninety-six cases, collected from various authors, and tabulated by Dr. Sibson (*Med.-Chir. Review*, October, 1854, pp. 434, 435), shows that by far the larger proportion of cases of valve-lesion tend to thicken the walls and enlarge the cavities of the heart; that aortic regurgitation with narrowing of the aperture, and still more without such narrowing, induces active dilatation of the left ventricle, followed consecutively by enlargement of the left auricle and the right ventricle and auricle; that disease of the pulmonic valves causes dilatation of the right cavities; that mitral narrowing with regurgitation leads to enlargement of the left auricle, followed in succession by dilatation of the pulmonary veins, congestion in the lungs, enlargement of the right ventricle and auricle, distension of the venæ cavæ, engorgement of the liver, congestion in the systemic capillaries, and at length, and in nearly one-half of the cases, enlargement of the left ventricle itself; that combined disease of the aortic and mitral orifices causes enlargement of the left ventricle, and to a less, but nearly to the same extent, of all the other cavities. Thus there is established a great pathological fact—originally stated by Senac, and confirmed by Morgagni—that *dilatation*, with *hypertrophy*, acknowledge as their cause a force acting *a tergo*, attempting to overcome an obstacle in advance. But there are also other morbid states which, upon the same principle, tend to these results, and are independent of valve disease—namely, *bronchitis*, *emphysema*, and any lung disease in which there is an obstacle to the flow of blood through the lungs. Disease also of the arterial trunks, such as *atheroma* (*endarteritis deformans*) or morbid states of the blood, like anæmia, dilatation or narrowing of vessels, whether of the lungs or of the system, may induce dilatation of the right and left cavities of the heart (ROKITANSKY). The retrograde influence of the systemic capillaries, as shown by Dr. Sibson, tends to exercise a similar influence, as shown by the effect of sudden fright and despair, causing rupture of the left ventricle, or by the influence of *Bright's disease*, altering the quality of the blood, tending thereby to retard its progress through the systemic capillaries. Thus hypertrophy and dilatation ensue from increased resistance to the exit of blood from the cavity of the heart, and from the necessarily increased efforts to expel it, and to propel it onwards.

Increased arterial resistance may be connected with a variety of causes. Thus, it may arise either from a contracted state of the capillaries, from diminished elasticity of the arteries, or from narrowing of the aortic valve. Figs. 40 and 41 serve to illustrate these points. An example of the most simple case of resistance to the contraction of the left ventricle—namely, that which occurs in aortic stenosis—is given in fig. 40. This is the pulse-tracing of a patient in whom the heart, with the adherent pericardium, weighed thirty ounces. The left ventricle was both thickened and dilated, and the aortic valve so deformed and beset with vegetations, that the orifice would scarcely admit the tip of the index finger. The other valves were healthy. All the other morbid appearances found were distinctly referable to the cardiac lesion as their cause. The tracing scarcely needs explanation. The second event, which, in the pulse of aortic regurgitation, and indeed in all atonic pulses, is either entirely suppressed or difficult to distinguish, is here extremely well marked; and the form of that part of the pulse-curve which represents it shows that the systolic expansion of the artery is of nearly equal duration with the diastolic interval (SANDERSON).

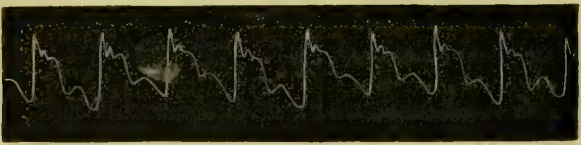

Fig. .40*

The patient whose pulse is represented in fig. 41 was complaining, at the time when the observation was made, of dyspnœa on the slightest exertion, and præcordial pain. He was subject to nocturnal paroxysms of cardiac distress, which, he stated, always came on with pain at the scrobiculus, followed by palpitation and nausea, with violent respiratory efforts. On examining his chest, Dr. Sanderson found that the cardiac dulness extended from the sternum to the mammary line, and that the præcordial impulse was diffused and expansive. The systolic sound was prolonged, but no abnormal murmur could be made out. The man died several months afterwards, when it was found that the heart weighed twenty ounces, and that the left ventricle was both hypertrophied and dilated,

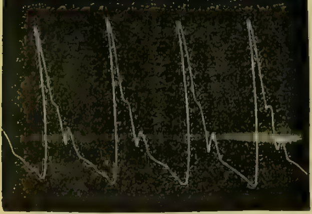

Fig. 41.†

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* G. M., aged forty. Pulse of aortic obstruction, with hypertrophy of the left ventricle (SANDERSON).

† W. S., aged fifty-nine. Hypertrophy of the left ventricle, without valvular disease.

without valvular disease. (See under "Bronchitis" how collapse of the lung may also produce dilatation without valvular disease.)

Just as with the external muscles, so with the internal, whenever they are subjected to constantly recurrent and vigorous action with sufficient supply of nutritive material, so will they increase in growth and volume. The well known instance of the blacksmith's arm, the leg of the ballet dancer, and the mountaineer, are cases in point. It is now also a matter of demonstration that hypertrophy of the heart occurs whenever the function of the organ is permanently or repeatedly over-tasked, and when the resistance which it should normally encounter is increased; and anatomically the nerve arrangements are such as to regulate its action, so that its energy is adjusted to meet the wants of the system in such a way that activity increases as obstacles arise.

The beautiful dissections of the heart by Mr. Pettigrew, in the University of Edinburgh and in the College of Surgeons of London, show how the heart consists of several folds of organic muscular fibre-tubes folded on each other, associated with a system of motor and co-ordinating innervation capable of rhythmical contraction and distension. The motor power of the heart seems to be under the control of minute nerve ganglia, "each with a morsel of muscular fibre under its direction" (FOTHERGILL). Under the influence of these ganglionic cells, acting directly on the muscular substance, the action of the heart is carried on and excited in a tumultuous manner, if the connection of the heart with the vagus nerve be severed. The vagus nerve is thus shown to exercise a co-ordinating (VON BEZOLD) or an inhibitory action, by spinal fibres passing to the ganglionic cells (BIDDER), and acting indirectly through them. The application of a stimulus, such as electricity, to the vagus, retards the cardiac contractions, and may even arrest its action in diastole. When the cavities of the heart are filled by blood, such distension by its contents leads to more or less rhythmical contractions. These contractions the vagus nerve seems to regulate, so that they only occur when the stimulus of distension is sufficient, and then contraction, truly peristaltic, takes place so swiftly that it seems a simultaneous general contraction of the whole heart.

Thus, to some extent, not yet determined exactly, the vagus nerve regulates the rhythmical contractions of the heart; and whatever may influence the vagus nerve may produce irregularity of the heart's action.

The condition next in importance to the nervous influence in hypertrophy is that of nutrition. There must be the necessary amount of nutrition and growth to meet the demand. If increased strain be thrown upon the heart to overcome obstacles to the passage of the blood through its several orifices, as occurs with the simple growth and development of the body, increased growth and bulk of the muscular tissue takes place, so that the balance is always maintained between the blood to be driven and the power to drive it (FOTHERGILL).

But if there is a deficiency, from any cause, in this compensatory nutrition, dilatation of the cavities sets in, from over distension of the heart's fibres. In fact there seems to be *a system of innervation of the heart*, not yet well understood, which can be compared only to that of the *iris*. The innervation is complex, so as to involve contraction and reduction of the capacity of the cavities on the one hand, and distension on the other; but how these compensations are brought about is not yet fully known, even if MM. Cyon and Claude Bernard's dissections of nerves in the rabbit were verified, to show an *acceleration nerve* as stimulating increased action against obstruction, and a *depression nerve* of the heart regulating the normal distension of its walls.

This involves a power of accommodation which seems similar to that of the power of accommodation possessed by the texture of the eye connected with the *crystalline lens*, the *iris*, and the *ciliary structures;* so much so that the cardiac chambers may for a time become blood-reservoirs, from which a portion only of the blood may be expelled.

A summary of the conditions which thus give rise to obstruction of the heart's action, and therefore to hypertrophy in its efforts to overcome the resistance, is given by Niemeyer as follows:—

(1.) Hypertrophy of the heart almost always accompanies abnormal enlargement of its cavity (*dilatation*). When so dilated, its capacity is increased; and, as the organ cannot discharge its normal load without expenditure of a certain degree of force, the effort requisite for the expulsion of its abnormal increase of contents must be proportionately greater, even though the resistance at the orifices and in the arteries be normal.

An adherent pericardium induces dilatation of the heart, and that again hypertrophy; the obstacle being the adherent pericardium. So also defective closure of valves induces dilatation in the first instance, from which hypertrophy follows in the portion of the heart concerned with the deficient valves, caused by the greater effort required to expel the increased amount of blood which the heart contains.

(2.) Hypertrophy of the heart accompanies stricture of its outlets, congenital or acquired, and contraction of the great vascular trunks—conditions causing resistance to the flow of blood which the heart has to surmount; and *stenosis* of the auriculo-ventricular orifices brings about hypertrophy of the auricles.

(3.) Aneurism of the aorta and pulmonary artery bring about hypertrophy of the heart. The sudden expansion of an aneurism increases resistance to the flow of blood, and more force is required on the part of the heart to propel the blood through the dilatation. Hence the hypertrophy.

(4.) Any obstruction to the blood occurring in the range of the aortic current, or of the current of the pulmonary artery, will induce hypertrophy of the heart.

(5.) General plethora may be accompanied by hypertrophy of the heart.

In overcoming these obstructions, the effort which the heart is excited to make, in the first instance, is attended with increased distension on the one hand, and an accelerated contracting action on the other; the combined exercise of which results in a power of accommodation regulating the action of the heart, according to the circumstances of the case.

The dilatation may be permanently established under any of the following circumstances (FOTHERGILL):—

(1.) When mitral regurgitation leads to enlargement of the left ventricle, pouring in of the blood takes place under increased pressure.

(2.) Muscular failure from defective nutrition, as in fever, atheroma of the coronary arteries of the heart, or pericardial adhesions.

(3.) Obstruction to the flow of blood forwards, as by deposits on the semilunar valves, and diseased arteries, such as atheroma or *endarteritis deformans*.

(4.) Functional disturbance of nerve-influence.

(5.) Excessive exertion and consequent cardiac exhaustion.

(6.) Valvular insufficiency.

The greater the distension, the more frequent and greater are the efforts at contraction. Such increased and frequent contraction tends to increase the arterial distension and tension, and so far relieves systemic symptoms. But increased arterial recoil takes place, propelling more blood into the coronary arteries during the diastole, which thus increases and improves the coronary circulation, greatly increasing thereby the nutrition of the heart, and thus promoting compensatory hypertrophy, enabling the muscular walls the better to resist the distending action of the blood, or overcome obstruction to its forward flow (FOTHERGILL). Such compensatory hypertrophy frequently enables serious lesions to be borne without any great constitutional embarrassment, restoring a balance between the opposing forces of propulsion and obstruction. So perfect is such compensatory hypertrophy that, in a case quoted in a note by Niemeyer, " a huntsman in Griefsvald, who suffered from extensive stenosis and aortic insufficiency, and immense eccentric hypertrophy of the left ventricle, performed all the manœuvres and forced marches of the army without difficulty."

In farther consideration of the subject of hypertrophy of the heart, it will appear that the condition is one which can scarcely be described without a consideration of dilatation of the heart; and although the College of Physicians have separated hypertrophy and dilatation, regarding them as distinct substantive diseases, yet the phenomena of each are here considered together.

Symptoms.—In detecting enlargement of the heart and thickening of its walls, the size and force of the heart, ascertained by *palpation*, *percussion*, and *auscultation*, furnish the principal data. (See page 517, *et. seq.*) It is by the extent and power of the impulse that the heart's muscular condition is ascertained; and so long as the muscular condition is sound, the valve disease has but little influence on health (SIBSON and STOKES). As a rule, however, the persistence of valve disease implies an enlarged heart, with an impulse increased in extent and in power. But there are also cases to be guarded from mistake—namely, those where a murmur exists with a preternaturally strong, troublesome, quick, and smart impulse, *but limited within a diminished cardiac region.* In such cases such a murmur is of anæmic origin, and the heart is usually lessened rather than enlarged.

The symptoms of *hypertrophy* of the heart are local and general. The local symptoms are,—a more powerful impulsion, a wider range of action, and some change in the sounds of the heart. There is also a greater extent of dulness of sound in the cardiac region, and sometimes a bulging out of the left side.

The increased impulsion in hypertrophy of the heart is in proportion to the greater thickening of the walls. Thus, in slight cases, it is only sensible to the hand, while in others the heart "knocks against the ribs," and even raises the hand of the auscultator. This greater impulse not only often causes a vibration of the *præcordial* region, but even shakes the whole of the chest. Besides being sensible to the touch, the abnormal action of the heart in these cases is often sensible to sight, each contraction agitating the patient's dress, and sometimes even moving the bed-clothes. The point of the heart deviates more to the left, and its motions may be sometimes traced from the second or third rib as low as the sixth or seventh intercostal space.

The increased thickness of the walls of the heart is evidently unfavourable to the transmission of sound; and in simple hypertrophy without enlargement of the cavity, the natural sounds will be duller than in the normal state; and if the hypertrophy be concentric, or with smaller cavities, the natural sounds will be scarcely heard. When, however, the cavities are enlarged, as in eccentric hypertrophy, the sounds are often clear, full, and even much louder than natural.

In hypertrophy of the left ventricle the impulse is stronger immediately under the inferior portion of the sternum than between the fifth and sixth ribs. Lavoisi laid it down as a sign of hypertrophy of the right ventricle that there is swelling of the jugular veins, which pulsate synchronously with the carotids. Corvisart repudiated this symptom; but Laennec found it in every case of hypertrophy of the right ventricle. In general this pulsation is limited to the inferior parts of the jugular veins; but in other instances it has been seen to extend to the superficial veins of the arm. He regards this symptom, therefore, as one of the best diagnostic signs of hypertrophy of the right ventricle.

In estimating the general symptoms of hypertrophy of the heart, our knowledge of the influence of the left ventricle over the arteries would lead the inference, *a priori*, to softening of the brain; that one of the effects would be a disposition to congestion and to hæmorrhage; and that apoplexy, hæmoptysis, and hæmorrhage from the bowels would often result. It appears, according to Bouillaud, that out of fifty-four cases of hypertrophy of the heart, eleven, or one-fifth, were attacked by cerebral hæmorrhage, or ramollissement of the central ganglia of the brain. Many suffer from pulmonary hæmorrhage, while a few suffer from hæmorrhage from the bowels. Indeed, on opening bodies that have died of this disease of the heart, the abdominal viscera and mesenteric veins are found loaded with blood. The conjunction of hypertrophied heart is very common in Bright's disease. Besides these concomitants, a pouchy or otherwise diseased state of the aorta often co-exists with hypertrophied heart—the diseased aorta being caused by the abnormal power of the heart; or the hypertrophy of the heart results from a supplemental force being necessary to compensate the functional incapacity of a diseased aorta. Many persons affected with hypertrophy of the heart suffer severely from *angina pectoris*, with palpitation.

Dilatation of the heart implies that the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms—namely (1.) *When dilatation predominates over hypertrophy;* (2.) *Simple dilatation, where the thickness of the walls is normal;* (3.) *Dilatation where the walls of the heart are attenuated, or thinner than normal.*

Practically, therefore, the physician has principally to deal with the diagnosis and treatment of—(1.) *Simple hypertrophy;* (2.) *Hypertrophy with dilatation of one or more of the cavities;* and (3.) *Simple dilatation with or without attenuation of the walls of the cavities.*

Hypertrophy of a sufficiently compensatory nature entails no distress. Accordingly those symptoms are to be recognized which mainly point to insufficient compensation between hypertrophy and dilatation, and to disturbance of the balance of efficient circulation. Inefficient power of the heart's action is first made obvious by the following conditions:—

(1.) *Palpitation,* which indicates increased action or effort on the part of the heart, with inefficient results. It is not accompanied by a bounding pulse or increased apex-beat, as after exertion or excitement. It is generally associated with irregularity of pulse, with chronic valvular insufficiency, and with *Bright's disease.*

(2.) The *irregularity of rhythm* in hypertrophy is a sign of debility. The commencement of the *peristaltic* contraction or heart-stroke is arrested. It is suggestive of a resemblance to the change of rhythm in the sound of a horse's feet when cantering (FOTHERGILL). It is serious evidence of over-taxation of the heart, often the result of over-exertion, and indicative of ventricular distension.

(3.) *Persistent intermittence* in hypertrophy is usually associated with an impaired first sound, defective apex-beat, and other signs of degeneration.

The most perfect action of the heart is when we are unconscious of its labours; but when a combination of the phenomena just noticed renders the patient apprehensive of evil, or causes him distress, then the physician must seek to discriminate whether the distress is arising from the predominance of *dilatation* or of *hypertrophy*—learn in which direction compensation is deficient, and aid the efforts of nature to repair the defects.

Evidences of distress on the part of the heart are to be recognized by the following signs:—

(1.) *Cardiac engorgement,* indicated by a feeling of anxiety about the præcordia, with a general unaccountable anxiousness; a sense of difficulty of breathing, which is notably increased upon exertion; fluttering about the left breast, amounting to palpitation upon effort or exercise; pulse irregular, or if regular, compressible; a dusky complexion and impeded respiration.

(2.) *Palpation* conveys a feeling of diffused impulse, from a large mass apparently being thrown into contact with the thoracic walls.

(3.) *Percussion* shows increased general dulness, frequently in the direction of the right side of the sternum.

(4.) *Auscultation* discloses a short slapping sound, with or without irregularity, or, perhaps, evidence of laboriousness—a heavy swell with obvious effort—not followed by a corresponding effect—in filling the arterial vessels.

(5.) *General phenomena* of distress embrace a tickling cough, shortness of breath on exertion, slight attacks of bronchitis, pulse small and feeble, cardiac excitement, with irregular rhythm—an aggregation of sounds and phenomena demonstrating embarrassment and laboriousness in the right ventricle, which may lead to permanent dilation of the right ventricular cavity, with more or less hypertrophy frequently found in persons advanced in life, and not uncommon in adult life.

"In this condition expulsive power is deficient until hypertrophy can compensate and restore the balance. The heart is distended, and repeated contractions fail to expel *all* the blood, the ventricles remaining more or less full on systole. The heart is more or less full before the distended auricle and veins behind pour in their contents under the increased pressure of distension. It is in the partially filled condition of the ventricles that the difficulty lies essentially. If the ventricles were not partially full, the auricle and veins would be somewhat relieved; but there is what would fill well an empty ventricle waiting to be discharged into one more or less full to begin with. The action may be moderately regular on quiet being maintained; but it is at once disturbed on motion, especially if this be at all active; and then we get palpitation and irregularity, or even intermittency, the regular action being again restored by quiet. There is a constant contest going on between the stimulus of the contained blood and the inhibitory action of the pneumogastric nerve fibres. The distension excites the muscular walls to overcome the restraining influence; for without the stimulus of distension the walls could never overcome the inhibitory action of the pneumogastric, the *vis inertiæ* of the blood to be driven; and action still further deranges the balance by making still greater calls on the muscular walls. In fact, the heart is in a state of over-distension, and in a condition both analogous to, and homologous with, an hypertrophied bladder attempting to overcome the obstruction of an enlarged middle lobe of the prostate. The over-distension goads the organ to such a contraction as shall relieve that over-distension, but only so far, and no further; there is no complete contraction. An incessant play goes on between the condition of over-distension and the restraining fibres of the vagus; the balance between the muscular walls and their work remaining confessedly disturbed (FOTHERGILL).

Such are the phenomena when dilatation predominates over hypertrophy.

When the reverse obtains—that is, *eccentric hypertrophy* of the left ventricle—a certain grouping of the following subjective and objective phenomena declares its presence:—

There is visible pulsation of the carotids, a loud systolic sound in the larger arteries, and a full pulse, visible even in the smaller arteries; an abnormally strong heart-stroke, extending over the length of the heart; a depression of the apex, extension of cardiac dulness, intensification of the heart's sounds in the left ventricle and aorta, and sometimes a metallic click (NIEMEYER).

When eccentric hypertrophy of the right side prevails, there is augmented heart-stroke, often extending along the sternum and left lobe of the liver, dislocation of the apex of the heart, which extends *outwards* rather than downwards, extension in width of the cardiac dulness, intensification of the cardiac sounds in the right ventricle and pulmonary artery (NIEMEYER).

These combined phenomena indicate general eccentric hypertrophy of both sides.

TABLE CONTRASTING THE MAIN SYMPTOMS OF THE FORMS OF HYPERTROPHY AND DILATATION.

A.—GENERAL PHYSICAL SIGNS.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Arching of the præcordial region, with widening and bulging of the left intercostal spaces from the third to the seventh.		No prominence of cardiac region.
Impulse increased in extent to the left of the sternum.	Extent of visible impulse much greater; pulse may be felt in the back, and its character is less heaving, sharper, and more knocking, than in simple hypertrophy.	Apex-beat indistinctly visible or actually invisible, the rounded-off form of the heart destroying the apex form.
Maximum impression felt below and about the left nipple.	Point of the apex-beat carried downwards and outwards beyond the line of the nipple, so as to reach the seventh interspace.	Impulse conveys an undulatory sensation to the hand, and feeble in proportion to the purity of the dilatation.
Heart's impulse slow and heaving, as if pressing steadily against an obstacle—in rhythm regular, in force unequal.	Force increased, so as to shake the head or trunk of the patient, or the bed on which he lies.	Force of successive beats is unequal. Rhythm irregular, to a slight or to the very highest degree. The hand applied to the region where the impulse is felt does not feel the beat always strike at the same place.
Superficial and deep-seated dulness augmented in area, but its shape retaining the triangular form.	The dulness tends to assume a square form in place of a triangular one, and may reach from the second interspace to the eighth rib, and from an inch and a half to the right of the sternum to three inches, or even more, outside the vertical line of the nipple. It may be detected in the back.	Intensity of superficial percussion dulness not increased; and in cases of attenuated walls of the heart's cavities, the resistance is less marked than in health. General areas of dulness widened.
First sound is dull, muffled, prolonged, and weakened, almost to extinction, directly over the ventricle.	Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.	*First sound* short, abrupt, and unnaturally clear at the apex and base, appears near the surface, and its maximum point of expression is slightly lowered.
Second sound full and clanging, *post-systolic silence* shortened.		*Second sound* not specially affected. Upon the quality of the heart's texture, as regards softness or flabbiness, will depend the extent to which the sounds are transmitted. Intracardiac murmur always regurgitant.
Mitral regurgitant murmur, as a clinical fact, exists at one time and disappears at another.	In consequence of the altered direction of the orifice of the aorta to the cavity of the ventricle, a systolic basic murmur may be generated.	

B.—General Functional Symptoms.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Strength unimpaired. Power of walking or of ascending a hill diminished, on account of dyspnœa induced by the effort.	Strength tends to become impaired.	Strength fails, and the patient is habitually irritable and melancholy.
Face florid.	Purpleness and lividity great in proportion to the valvular or pulmonary obstruction.	Lividity and mottling of the face prevails, and of the lower extremities generally, with chilliness of surface. Softly pitting anasarca spreads from the feet to the abdomen, external genitals, thorax, face, and neck. Ascites follows anasarca.
Constipation habitual.		Bowels habitually constipated or alternately relaxed—discharges dark.
Dyspnœa occasional.	Paroxysmal attacks of dyspnœa.	Dyspnœa, sometimes called cardiac asthma, becomes complete and habitual, with asthmatic paroxysms, in which the cough is dry, harassing, and convulsive. Expectoration serous, sometimes streaked with blood.
Radial pulse full, strong, firm, tense, resisting, and prolonged, without jerk or thrill.	Fulness of pulse continues, but strength and power of resistance lost.	Pulse small and feeble, and abnormally late in time after the ventricular systole. It may be regular, or narrow, feeble, fluttering, and irregular.
Pain rare.	Pain not uncommon.	Palpitation and cardiac uneasiness most distressing.
Rarely, and never rapidly, the direct cause of death.	Indirectly, and more or less rapidly, leads to a fatal issue.	Faintness occurs from time to time, and may lapse into syncope and sudden death.

The preceding table (compiled from Dr. Walshe's treatise) exhibits sufficiently the main points in the symptoms of these forms of disease for the purposes of comparative diagnosis.

Dilatation of the cavities of the heart may exist both when the substance of the heart is hypertrophied and atrophied; but it may also exist when the walls of the heart are of their natural thickness. In any case the dilatation may be partial or general. Partial dilatation of the heart sometimes presents many curious phenomena: thus, the walls of the right ventricle have been seen divided into two distinct parts; or, as Laennec has described it, into a sort of hour-glass contraction.

In other cases this partial dilatation is perfectly aneurismal. Corvisart gives the case of a young negro who died suffocated, and in whom the superior and lateral part of the left ventricle was surmounted by a tumor almost as big as the heart itself. The inner surface of this tumor contained many concentric layers of lymph, exactly similar to those of an aneurismal sac. The cavity of this tumor communicated, by means of a small opening, with that of the ventricle. Laennec mentions two cases in which a tumor of a globular form, and the size of a duck's egg, was situated at the point of the left ventricle, and communicated with the ventricle by an opening an inch in diameter. In these cases the left side of the walls of the sac presented a continuation of the muscular fibres of the heart, while on the right side they appeared formed by the two pericardia. Laennec thinks that these aneurismal tumors are formed by ulceration of the internal walls of the ventricle, as in false aneurism of the arteries; others, that it is owing to a separation of the muscular fibres and the protrusion of the inner pericardium. These are examples of aneurism, not of hour-glass contraction.

Prognosis.—As a complication of other diseases of the heart, hypertrophy, when compensatory, is a favourable rather than an unfavourable condition, tending to mitigate the danger of the chief disease. When death occurs, it is generally due to hæmorrhagic effusions into the brain or lungs, for the prevention of which, treatment such as to favour the compensatory powers of the circulation must be had recourse to with great care and judgment, combined with careful management of the diet. When the phenomena of hypertrophy have persisted for a time they are sometimes replaced by another form of lesion—also preservative, as has been shown by Sir William Jenner. A fatty degeneration of the heart commences, from impaired nutrition due to atheroma of the arteries. Its activity is thus impaired and lessened, and there is less risk of its violent action rupturing the atheromatous and friable arteries of the brain or lungs.

The failure of the right side of the heart in asthenic disease or affections of the respiratory organs is often the channel through which death approaches. The right ventricle, taxed to the utmost, becomes gradually paralyzed by the carbonic acid of its contained blood *anæsthetising* its action, and if its action can be maintained the patient may tide on to recovery (FOTHERGILL).

Treatment.—The symptoms of simple hypertrophy may, in the majority of cases, be greatly mitigated by such means as tend to tranquillize the action of the heart. This end may be best accomplished by occasional very moderate cuppings or leeching over the præcordial region. No known drug possesses the power of controlling the growth of the heart. Saline and aloetic purgatives aid the calmative influence of the local abstraction of blood. Diuretics are useful independently of the existence of dropsy. Sedatives of the heart's action are indicated throughout, such as *hydrocyanic acid*, *acetate of lead*, *digitalis*, and *belladonna*. Of all medicines of this class Dr. Walshe considers *aconite* the best, in the form of the alcoholic extract, given in doses of *one-eighth of a grain*. In repeating the doses, the effects must be watched, while they relieve the painful sensations and disquietude about the heart. If anæmia prevails, animal food should be permitted; and the more soluble and less astringent preparations of iron should also be given. Fluids must be taken in small quantities. Months and even years of treatment may be required to produce any impression on the disease.

Like *hypertrophy*, *dilatation* of the heart is not removable by treatment, but judiciously directed remedial measures may render the condition bearable, and even for a time unappreciated by the patient. To improve the tone of the muscle and strengthen the action of the heart, without exciting its irritability, are the objects to be aimed at in the management of cardiac hypertrophy.

The beneficial influence of *digitalis* has recently been most ably shown by Dr. J. M. Fothergill, in his "Hastings' Prize Essay" for 1870, published in the *British Medical Journal,* commencing July 1st, 1871; and also by Dr. Balthazar Foster in the *Medico-Chirurgical Review* for July, 1871.

It is a most efficient agent in helping to co-ordinate, by restoring the regularity of the heart's movements.

The mechanism of compensation in each form of valve disease, therefore, requires to be considered in each case of eccentric hypertrophy, and have been already indicated (see page 575, *ante*).

In hypertrophy it is only of use when hypertrophy exceeds the limits of compensation. It slows the pulse and regulates the heart-muscle, but if the heart-muscle is unsound it will not be of service.

Dr. Foster prescribes it as an infusion, and continues it as long as the quantity of urine increases or keeps up to the maximum which the digitalis produced. Such diuretic effect is the outward or visible sign of its beneficial action. It indicates a restoration of the normal balance between the contents of the arteries and veins, an increased arterial tension and consequent refilling, under normal pressure, of the empty capillaries of the Malpighian glomeruli of the kidneys. The high-coloured scanty urine, loaded with urates, is then replaced by a clear and copious stream, which tells of a steadily beating heart and a firmer pulse (FOSTER).

Dr. Fothergill in his admirable essay considers *the tincture* of *digitalis* as the most convenient form, ordinarily, but it throws down a dark green precipitate with iron, which is objectionable.

The infusion of digitalis is a good preparation for use, along with potash or diuretics, and is conveniently added to vegetable infusions.

Where it is desired to keep up the action of digitalis for a long time, till structural changes are produced, *the powder of the leaves* is the most desirable form.

It can then be given in pill, with the dried sulphate of iron, carminatives, laxatives, or both, twice a day. Half a grain to a grain of *powdered digitalis,* with an equal quantity of *sulphate of iron,* and a small portion (¼ of a grain) of *cayenne* pepper, in extract of gentian or aloes and myrrh pill, is a useful form which may be continued for months. This pill should be taken shortly *after* food.

The addition of *iron* to *digitalis* is of great importance.

The absorption of *digitalis* through the skin, by using poultices of the leaves, or flannel cloths soaked in the infusion and laid over the skin of the abdomen and thighs, is often most beneficial where it cannot be given by the mouth (CHRISTISON, TROUSSEAU, FOTHERGILL.)

The action of *digitalis* on the dilated heart, not compensated by hypertrophy, is well shown by experiment on the heart of the frog (as described by Dr. Fothergill);—"When paralyzed and almost brought to a standstill in diastole by aconite, the heart is distended, globular, and, in every respect but that of chronicity, in the condition of a dilated or distended heart. Then administer digitalis, and watch the result. The distended globe, just pumping painfully a little off the top of the contained blood, and that at long and irregular intervals, begins to contract with more vigour; each ventricular systole is more and more complete; and the bulk of blood remaining unexpelled—and that is the great point—becomes less and less in quantity. Shortly the distension in diastole is shortened, the distension and contraction come gradually back to the norm, the irregularity in time is lessened, and a complete restoration results. But if the experiment be carried still further, spasmodic contraction or the condition of concentric hypertrophy sets in, irregularity again makes its appearance—for the balance is now disturbed in the opposite direction, in fact, the symptoms of digitalis-poisoning are brought out—and ultimately the heart is brought to a permanent standstill in systole. Thus, in a distended heart, only a longer time is requisite, and, of course, the cause of the original distension must be overcome; so that the conditions are scarcely equal, and longer time and an artificial compensatory hypertrophy is necessary to maintain the balance thus temporarily restored. When this condition of distension is only of short duration, as seen in people who have been overworked and overtaxed for a short period only, but presents all the appearances, signs, and symptoms of cardiac dilatation, the restoration of the natural balance by digitalis may be quick and withal permanent" (*Brit. Med. Journal*, July 8, 1871).

Combined with an exclusively milk diet *digitalis* is regarded by Niemeyer as an invaluable remedy in dilated heart, completely removing dropsical effusion of great magnitude.

There are some circumstances, however, under which it is necessary to withhold the administration of digitalis in cardiac disease, namely—

(1.) The presence of *atheroma* (*endocarditis*) to any appreciable extent is a decided contra-indication against the use of digitalis.

(2.) Reith, Gull, and Brunton also object to its use in fatty heart.

(3.) Intermittency of pulse is also a contra-indication; and if it comes on during its use, the farther administration of digitalis must be suspended, more especially if the pulse becomes thready, and the quantity of the urine diminished. On the other hand, where intermittency is shown to be the result of the heart's inability, the quantity of digitalis must rather be increased (Fothergill).

(4.) The occurrence of persistent vomiting or noises in the head under its use, suggest that it be left off.

The groundwork of medicinal remedies consists in the administration of general tonics in the form of *bitters*, *mineral acids*, and preparations of *iron*. Belladonna may be employed to tranquillize undue excitement with greater safety than any other sedative remedy. Sedatives, as a rule, are unsafe, and require the utmost caution in their use. Due action from the bowels must be daily obtained, to accomplish which the aloetic medicines are the best, aided by the gentle action of an occasional mercurial aperient.

The diet should be nourishing without being exciting; and may include animal food, with a moderate allowance of light beer or wine in small quantities to dinner.

When dropsy appears, diuretics yield most relief in the form of *acetate, nitrate, iodide,* and *bitartrate of potass, nitric æther, compound tincture of iodine,* the *infusion* and *spirits of juniper,* or *gin,* may all be employed in successive changes, and variously combined. Occasional small doses of *blue pill* and *squill,* at bedtime, will facilitate their action generally; and so will cupping over the region of the kidneys, if symptoms of congestion of these organs prevail.

Hydrogogue cathartics also aid the diuretics in subduing the dropsical effusions, in the form of *elaterium, gamboge, bitartrate of potass,* and the *compound jalap powder.*

Dr. Walshe prescribes the following formula for the administration of *elaterium:*—

℞. Extract. Elaterii, gr. $\frac{1}{6}$—gr. $\frac{1}{2}$; Extract. Creasotonis, gr. i.; Extract. Hyoscyam., gr. ii.; *misce,* fiat pilula.

(CARDIAC) ATROPHY.

Latin Eq., *Atrophia;* French Eq., *Atrophie;* German Eq., *Atrophie;* Italian Eq., *Atrofia.*

Definition.—*An abnormal wasting and loss of the muscular substance of the heart.*

Pathology.—Care must be taken not to confound this condition with rigid contraction of the substance of the ventricle diminishing its cavity; and, except as a congenital malformation, its idiopathic occurrence is not believed in.

As an acquired morbid condition it results in the course of a variety of diseases, especially the following:—

(1.) In common with the wasting of other parts, as in the course of *tubercular consumption,* of *cancer,* and of cancerous suppuration, and of old age; also in prolonged typhus and typhoid fever.

(2.) The result of unusual pressure, as from chronic pericardial effusion, thickening of the visceral pericardium.

(3.) Imperfect circulation through the coronary arteries, as from disease in them; or in the course of hypertrophy with aortic valve disease, leading to degeneration and atrophy of the muscular texture of the heart.

Dr. Chevers considered that pericardial adhesions led to atrophic diminution of the heart and of the great vessels (*Guy's Hospital Reports*).

Morbid Anatomy.—The walls of the heart may be so atrophied that this organ has been found to weigh only four ounces two scruples, instead of nine and a half ounces, while the thickness of its parietes was reduced to little more than a thin membrane. This atrophy may be general or partial. In some cases the atrophy takes place without any notable alteration of the capacity of the chambers of the heart. This is termed *simple atrophy of the heart.* More commonly, however, when the walls are thinned, the chambers of the heart are diminished also. Again, the whole heart may be atrophied and reduced in size, as is often seen in phthisis. Thus, Bouillaud gives the case of a woman, aged sixty-one, whose heart was no bigger than that of a child twelve years old; and Burns gives the case of an adult whose heart did not exceed that of a new-born infant. This form has been termed *concentric* atrophy. The fat disappears, and serous infiltration fills the connective tissue. The pericardium is opaque, and thickenings, such as white spots, become wrinkled, and the coronary arteries tortuous. Much fluid is usually found in the pericardial sac.

Symptoms.—A feeble impulse of the heart, while its sounds are louder, clearer, and more distinct than in health, the intensity of sound being greater in proportion to the atrophied state of the walls, combined with increase of size of the chambers of the heart, are local symptoms which may suggest atrophy. The general symptoms are,—slowness of the pulse, occasional palpitation, frequent attacks of fainting, difficulty of breathing, and tendency to dropsy. There are also signs of defective nutrition of the body generally; and great muscular debility (HOPE).

FATTY DEGENERATION OF THE HEART.

LATIN EQ., *Degenratio adiposa;* FRENCH EQ., *Dégénérescence graisseuse;* GERMAN EQ., *Fettije entartung;* ITALIAN EQ., *Degenerazisne grassosa.*

Definition.—*A change in the muscular substance of the heart, which results in the elements of the muscular fibres being replaced by molecular fatty particles. The change tends to sudden death by rupture of the heart, or by syncope.*

Pathology and Symptoms of Cardiac Degeneration.—Microscopic observation has revealed certain remarkable changes, of the nature of degeneration (see vol. i., p. 92), in the heart as well as in other organs, of slow and insidious development, most difficult to detect during life, hitherto unassailed by any remedy, productive of most fatal consequences, and the immediate cause of the sudden and unexpected demise of many distinguished men. It has especially cut off the hard-working men of the intellectual class—*e. g.*, the Rev. Dr. Chalmers and Dr. Abercrombie, of Edinburgh; Dr. Pereira, of London; and very recently the distinguished lawyers, Sir Cresswell Cresswell and Sir Frederick Slade.

Improved means of diagnosis have taught us to anticipate such a termination to many cardiac affections with which we are every day familiar; and in convalescence from severe injury, or under chloroform, sudden death is extremely apt to supervene by syncope, in cases where the heart has undergone the degenerations about to be described.

At least two varieties of fatty disease of the heart have been recognized:—

(*a.*) In the one form the fat, composed of oil in nucleated cells—the ordinary fat-cells—grows on the surface of the organ, between its muscular fasciculi and the reflected pericardium, especially at the junction of the auricles and ventricles, in the groove or sulcus between the chambers, along the trunk of the coronary veins, at the edges of the ventricles, at the apex and at the origin of the aorta and the pulmonary artery. The right ventricle is often almost entirely covered with fat. This form of fat accumulation is not degeneration, but merely an abnormal increase of the amount of fat usually found upon the surface of the heart; but it so gradually encroaches on and insinuates itself between the muscular fibres, that it conceals, impoverishes, and ultimately causes them to waste, from its pressure, so that the muscular walls become thin, especially towards the apex and over the walls of the right ventricle. In these parts the fibrous structure almost disappears, and the *columnæ carneæ* appear to spring altogether from the *endo-pericardium.* In this form of fatty heart the muscular fibres may remain healthy, although they sometimes eventually degenerate. It usually accompanies general corpulence, and especially the obesity of advancing age where much alcoholic aliment is taken.

(*b.*) In the other form of fatty heart a degeneration of the fibre ensues. Its muscular element disappears, and its place is taken by fat in a molecular form, and minute oil-globules ultimately come to fill the sheathes which previously contained muscular fibre. It is a fatty metamorphosis of the primitive fasciculi of the muscular substance.

But it is now also observed that various forms of degeneration are capable of microscopic demonstration in the minute fibrillæ of the heart's substance, the result of decay or disintegration.

The recognition of fatty degeneration in the minute tissue of voluntary muscle led to the observation of analogous changes in the tissue of the heart by Corvisart and Laennec. In Scotland the younger Duncan, Cheyne, and Adams were amongst the earliest observers; but the subject has been mainly elucidated by the writings of Smith, Stokes, Andral, Rokitansky, Hasse, Paget, Ormerod, Quain, Begbie the elder, and Handfield Jones. Considerable variety of opinion prevails amongst these observers regarding the nature and the sequence of phenomena associated with this degeneration; and especially as to the symptoms diagnostic of fatty degeneration of the heart, by which its existence may be inferred during life.

I have made an analysis of twenty-nine cases of sudden death associated with such a lesion, the histories of which are scattered throughout the first ten years' "Records of the Transactions of the Pathological Society of London" (*Med.-Chir. Review,* 1858, p. 429). The results of this analysis are incorporated in the following account, illustrative of the pathology of this formidable disease:—

Age.—Of the twenty-nine cases sixteen were females and thirteen were males, in all ranks and social conditions of life. The average age of the females was *forty-six* years, and the average age of the males was *fifty-two.* The youngest patient was a male infant *six months* old; while the oldest male and the oldest female appear each to have been *seventy-six* years of age. The youngest female was *ten* years of age; and the youngest male (exclusive of the infant) was a boy *eleven* years of age.

In the female a tendency to the degeneration seems to have been observed at a much earlier age than in the male; but between the ages of *fifty* and *eighty,* in both sexes, the greatest number of cases have occurred. The Rev. Dr. Chalmers's age was *sixty-eight;* that of Dr. Abercrombie was *sixty-five;* that of Sir Cresswell Cresswell was *seventy.* Among eighty-three cases collected by Dr. Quain, death occurred in fourteen between the ages of *fifty* and *sixty;* in eighteen between *sixty* and *seventy;* and in fourteen it occurred between *seventy* and *eighty.*

The general health and condition of the patients previous to their fatal illness has been variously described. In eight of the cases recorded in the Society's *Transactions* the previous health is described as delicate, weak, nervous, or reduced by previous illness, such as miscarriage, menorrhagia, hæmorrhoids. The patients who are thus described were of short stature, thin, and spare make. Eight other cases are described as strong, stout, fat, muscular, or hearty. Of these some were of sedentary habits, unaccustomed to active employment, but temperate in habit. Others had suffered from slight attacks, such as of bronchitis, "liver complaints," "spasmodic pains of the stomach." Some are described as anæmic and pale, although at the same time stout and well-grown.

A third class of cases had suffered more or less severely from previous attacks of acute diseases, although they had recovered from them to some extent. One had suffered from occasional headache, with seizures of an apoplectic-like nature after fifty-five years of age. A similar case is related by Dr. Fuller (*Diseases of the Chest,* p. 602). Another had been thirty years in India, exposed to malaria, and had suffered from frequent attacks of intermittent fever. A boy, aged ten, had fever at the age of four years, afterwards chorea, and subsequently scarlatina and rheumatism five months before death. Mental anxiety and domestic distress, inducing great irritability and nervousness, were the antecedents of two other fatal cases; and five other cases are described as having to all appearance enjoyed excellent health.

Dr. Begbie, senior, in a most interesting communication to the Medico-Chirurgical Society of Edinburgh (January 15, 1851), relates some passages in the lives and deaths of the Rev. Dr. Chalmers, and of Dr. Abercrombie, which bear on the previous general health of these two distinguished men. Of the Rev. Dr. Chalmers he relates that, "to a mind of the highest order, and of wondrous energy, he united a hale and vigorous, a manly and robust frame. He spared no exertion, either mental or physical, in carrying out the great object of his life. He was hardly ever incapacitated by infirmity or loss of health in prosecuting his enterprise; and from early manhood to green old age, even up to his latest hour, he toiled and spent his energies and strength."

These things Dr. Begbie mentions "to show that the fatal disease which lurked within, which was progressive in its nature, and probably of long standing, could neither have produced serious uneasiness nor proclaimed its presence by any unequivocal signs." But it is related that, thirteen years before his death, Dr. Chalmers had a sudden seizure on the street, of what proved to be a serious and alarming illness. He lost the power of the arm and leg of the right side, and experienced diminished sensation on that side. His face was pale, the skin cool, the pulse soft and frequent. After a few weeks of rest and quiet he completely recovered, and returned as vigorously as ever to his professional duties; but with accumulating years there came a disposition to obesity; and with the silver grey on the massive forehead came also the pallid and somewhat sickly look of fading health. He was sometimes sick at stomach from some trivial ailment arising from indigestion; but he was never faint, nor ever swooned away.

Of Dr. Abercrombie it is related that he enjoyed, during a long series of years, uninterrupted health; but three years before his death he was suddenly seized with loss of power and impaired sensation over the left side, but without the loss of consciousness or any affection of speech. He experienced great anxiety, and complained of præcordial uneasiness and slight headache. He sighed frequently, and had a cold skin and pallid countenance. The pulse was frequent and small at first; but after a while it subsided in frequency and rose in strength. Dr. Abercrombie's own impression was that his illness was paralysis, connected with cerebral disease. It never occurred to himself or his medical advisers to connect the symptoms with deranged circulation arising from a damaged heart; and although he complained of præcordial uneasiness, which never amounted to actual pain, and of something more than uneasiness in the left arm and shoulder, and at the base of the scapula, yet the circulation was regular, though feeble—a feebleness which might have been accounted for by the active depleting measures he himself employed, and the scanty fare to which he subjected himself. Although he recovered from this attack he continued pallid; and just before his death he was observed to be breathless on ascending a stair.

In the cases detailed in the *Transactions of the Pathological Society*, *cardiac symptoms* occurred sometimes suddenly after exposure to cold, with pain in the chest, shortness of breath, and palpitation. In some of the cases associated with *angina pectoris* the pain was sometimes excessive, shooting down the left side and arm, especially after any excitement. In other cases there were *cardiac symptoms,* consisting of a dull pain in the region of the heart or ensiform cartilage before death, associated with dyspnœa. In some *angina pectoris* prevailed for many years. The cardiac affection in several instances betrayed itself by cough, dyspnœa, and general debility, a sense of oppression at the chest, and desire to draw a deep breath, the breathing being sometimes embarrassed and aggravated by exertion. In seventeen out of twenty-nine cases death was sudden and unexpected, and cardiac disease had never been observed, and only in one case suspected to be present, by the medical attendant.

In most instances the attacks of giddiness and apparent coma are due to syncope, causing a deficiency in the supply of blood to the brain, from feebleness of the heart's action. Unlike what happens in cases of apoplexy connected with hypertrophy of the left ventricle and excitement of the arterial circulation, the patient when attacked is pale, has a feeble pulse, and presents more or less lividity of the lips—symptoms which may serve as indications of the true nature of the disorder (FULLER).

The condition of the pulse having been observed after the cessation of an acute attack of disease, in twelve cases out of twenty-nine, which afterwards terminated fatally, it was noted as "irregular and unequal;"

as "feeble, rapid, and irregular;" as "feeble and intermitting, or occasionally so every eight or ten beats;" as "large, jerking, and compressible, one hundred and six per minute—afterwards a hundred, but small, jerking, and regular to the last;" as "small and feeble, but no irregularity or intermission." In one of the cases the pulse was observed for two years and a half previous to death to be irregular and uncertain in its action—a condition which disappeared during an attack of bronchitis, but which reappeared as the patient recovered from the bronchitic attack—varying from *seventy* to *eighty* in a minute. In other cases, where its condition had been long noted, it is stated to have been weak, irregular, and intermitting, numbering at first *eighty*, becoming weaker, often intermitting, and more slow (*sixty* to *seventy*) shortly before death, or small and feeble. The slowest pulse recorded is *fifty-five*. In one case of death under the influence of chloroform, the pulse at first was *ninety-four*, regular, and of average firmness: subsequently, under its influence, it was accelerated, and suddenly began to get smaller, weaker, and imperceptible. In extreme cases the pulse may fall as low as twenty-six or thirty in a minute, owing to the failure of certain systoles of the heart to communicate a pulsation to the radial artery, the heart itself beating at the rate of fifty-six or sixty in a minute (Fuller, C. J. B. Williams, Gairdner).

The sounds of the heart have been observed to be modified. In one case "a loud bellows murmur" was heard all over the chest; death followed after complete destruction of the mitral valve by rupture. In another case there was extensive dulness over the heart, and a loud bellows murmur with the first sound; and in this case the edges of the mitral valve were fringed with bead-like vegetations. In a third case a *diastolic bruit* existed in the region of the aortic valves, the heart's action ultimately becoming tumultuous, and the sounds obscured, attended with a rolling action four day's before death. In this case a band of lymph was found extending across the aortic orifice, with irregular vegetation over the sigmoid valves, while at the opening of the coronary artery a false aneurism opened into the muscular substance of the left ventricle. In a fourth case the systole was attended with a loud and prolonged *bruit*, loudest over the mitral valve, followed by a distinct natural second sound, to which succeeded a remarkably prolonged interval of rest, and the rhythm was frequently irregular. Associated with these sounds was some thickening of the aortic valves, and especially a peculiar degeneration into fibrinous matter of the muscular substance towards the base of the heart. In a fifth case the area of the *præcordial* dulness was *less* than natural, and there was a systolic endocardial grating murmur, with feeble impulse, and the mitral valve thickened.

In four only out of the twenty-nine cases were the cardiac sounds so modified that (in the absence of valvular disease) the cause of the modification was ascribed to the morbid condition of the muscular tissue of the heart. In one of these cases the impulse and sounds of the heart were observed to have been feeble six weeks before death. In a second case the impulse was noted to have been feeble and of limited extent, the second sound being very indistinct, but no murmur. In a third case the heart's action was feeble and irregular, the sounds being weak; and in a fourth case the sounds were muffled, but no *bruit* existed.

Other symptoms associated with this remarkable degenerescence and formidable disease were,—in some cases severe vomitings and faintings, or a peculiar sinking and sense of faintness, with profuse perspirations; paleness and lividity, with urgent dyspnœa for some weeks before the fatal result; sudden aggravation of cardiac symptoms, of cough, or of general debility, for some days before death. In one case there was evidence of scurvy or of purpura; and thirteen days before death this patient became unsteady in gait, had impaired vision, with a vacant countenance, dilated and sluggish pupils, headache, and pains round the orbits, with spectral illusions, partial blindness, and hæmorrhage from the nose and bowels (Bristowe, *Path. Society's Trans.*, vol. v., p. 93). In another case the general symptoms consisted of slight dyspnœa, with evidence of acute rheumatism; and four days before death dyspnœa became urgent. In a fourth case, for four years previous to death, sudden and severe attacks of shortness of breath were observed on exertion. These attacks were relieved by *ether* and *ammonia*, but they were always followed by faintness and exhaustion, with coldness and lividity of the face and extremities (Quain, *Path. Society's Trans.*, vol. iii., p. 82). In the fifth case, what is termed "biliousness" was troublesome, with a sense of heaviness and oppression referred to the sternum. Two months before death there was general *malaise*, numbness of the fingers of the left hand, with tingling or uneasy sensations over the surface generally. A sixth case suffered from attacks of headache at intervals of a few months, associated with flashes of light before the eyes, and darkness of half the objects seen. Such attacks lasted about a week, attended with loss of speech, impairment of memory, and feebleness. In three cases, attacks of *angina pectoris* are recorded. In one of these, six weeks before death, the attacks became so violent that the patient lived in constant dread of pain, which was of a most excruciating kind, accompanied by a feeling of suffocation. Three days before death he attempted suicide by cutting his throat in one of these paroxysms; but the wound was very superficial, and healed. A convulsive fit continued for ten minutes two hours before death.

The modes of death may be described as follows:—

In *eleven* out of twenty-nine cases the death was absolutely *sudden* or *instantaneous: two* were *comparatively sudden in bed,* the patients having been previously up at the night chair; *three* patients were found lying dead; and in *three* a death-struggle existed of from *three* to *five* minutes' duration; in *one case* four paroxysms of rigor, nausea, and "spasm of the stomach," with small and contracted pulse, occurred at intervals within twenty-four hours, and at last suddenly proved fatal; another patient died in a paroxysm of *angina pectoris;* another under the influence of chloroform.

Death by syncope is another usual mode of death in such cases. Thus died Dr. Chalmers, the eminent Scotch divine. It is a mode of death apt to occur in cases of convalescence from injury; and the cases of Dr. Pereira, who died while convalescing from rupture of the *tendo Achilles,* and of Sir Cresswell Cresswell, who died while recovering from an injured patella, might suggest the necessity of a cardiac examination in similar cases, and the maintenance of a sufficiently stimulating diet, and wine, if previously taken, with a careful avoidance of such circumstances as may tend to syncope, in handling the injured parts of men otherwise healthy, but advanced in years.

Morbid Anatomy and Nature of the Change observed in the Minute Tissue of the Heart.—A considerable variety of description under this head has been given, which may be classified as follows:—

(1.) *Cases in which there is a very moderate amount of degeneration of tissue, or alteration in the bulk of the heart,* but in which the functional disturbance is serious in the extreme, and the case rapidly fatal in its issue (Williams, l. c., vol. ii., p. 186).

(2.) *Cases in which, amongst apparently healthy tissue, a portion of the muscular substance has undergone degeneration*—the evidence of degeneration consisting of "the disappearance of the cross-marking of the muscular fibrillæ," and "the fibres being filled with oleo-albuminous or fatty granules." The coronary artery in such cases is found ossified or obstructed, going to the seat of degeneration (Quain, l. c., vol. ii., p. 188; and vol. iii., pp. 262, 270, 273). In the case of the Rev. Dr. Chalmers, Dr. J. H. Bennett, Professor of Physiology, of Edinburgh University, made the *post-mortem* examination, and reported that the substance of the heart throughout consisted of fatty granules. The muscular fasciculi could scarcely be seen, although here and there traces of the longitudinal fibrillæ could be observed. No transverse striæ were anywhere visible. The heart was flabby and unusually soft. The coronary artery was loaded with calcareous deposit, much contracted, and in one place obliterated, presenting considerable resistance to the knife.

In these cases the substance of the heart is discoloured where the degeneration is sufficiently marked. The degenerate parts are of a pale yellowish colour, and the texture tears readily. These changes may be seen in large masses, or pervading long tracts of tissue, or particular parts only, such as the papillary muscles or *columnæ carneæ.* This is the form most often associated with other evidences of the general marasmus of old age, which may be a ripe old age or a premature one. It is especially associated with the *arcus senilis* and fatty degeneration of arteries.

(3.) *Cases in which the death could not be ascribed to fatty degeneration of the heart alone, but where other lesions existed*—such, for instance, as fatty degeneration of the cerebral arteries, resulting in death by apoplexy; or in cases in which apoplectic seizures occur—a lesion we owe so much to Sir James Paget for elucidating—or in which, according to Dr. Quain, obstruction to the flow of blood from the head leads to congestion of the brain and hæmorrhages. The source of the obstruction Dr. Quain believes to be due to the want of power of the right ventricle (on account of degeneration) to maintain the circulation through the lungs; the blood, therefore, accumulating in the brain, tends to death by apoplexy, for the most part meningeal (*Path. Society's Trans.*, vol. iii., p. 190).

(4.) *Cases in which, in addition to the degeneration of the heart's fibres, they are also encroached upon and rendered powerless by the growth of fat intruding upon them, and covering them up.*

Atrophy, wasting, and disappearance of the proper muscular tissue are the immediate results of this encroachment, terminating in a thinning of the muscular parietes of the organ. Along with this especially atrophic result, some of the fibres of the heart which remain undergo the fatty degeneration within the sarcolemma; but this homogeneous sheath seems to remain intact, while the nuclei within or upon it disappear, or break up into streaks of oil dots. Thus, true "sarcous elements" ultimately come to be replaced by minute opaque molecules (the nature of which has not been in all cases determined), or actually by small drops of oil.

(5.) *There are cases of this degeneration which result in the "cardial apoplexy" described by Cruveilhier, where hæmorrhagic spots, or extravasations of various sizes, occur in the substance of the muscular tissue; the surrounding tissue is found in the state of fatty degeneration, and the coronary arteries leading to the degenerate tissue and hæmorrhagic spot are ossified or obstructed* (Quain, *Path. Soc.*, vol i., p. 192; vol. ii., p. 190). A condition somewhat of this kind seems to have occurred in the case of Dr. Abercrombie. The late Mr. John Goodsir, Professor of Anatomy in Edinburgh University, who made the *post-mortem* examination, reported that irregular ecchymosed spots were situated near the rupture which proved fatal. These spots consisted of effused blood; but their connection with ruptured vessels could not be distinctly made out. The serous membrane over them was quite entire. Both coronary arteries were much dilated as they passed from the aorta; and throughout their course they contained in their walls much atheromatous matter. The heart was slightly enlarged and dilated, rather loaded with fat, and remarkably soft, as if from interrupted nutrition. Mr. Goodsir examined the tissue near the rupture with the microscope, and found that the muscular fibres had undergone the fatty degeneration. The granules were arranged in transverse rows, and some of the fibres were nearly empty. To the naked eye the muscular tissue of such hearts presents a pale, mottled, or dirty yellow appearance. It has none of the flesh-red hue of health. The heart is soft and flabby to the touch; and its texture at the degenerate parts so friable, and sometimes brittle, that it yields to the slightest pressure, and may be torn without difficulty.

The elasticity of the muscular substance is so completely lost that, when cut across, the walls at once collapse.

Diagnosis.—The diagnosis of a degenerate heart is by no means easy; and a diagnostic value having been attached to the existence of fatty atrophy of the cornea—the "*arcus senilis*"—in connection with this disease, it is necessary to notice the circumstance only to mention that, as a sign of fatty heart, it is by no means to be depended upon in every case.

Treatment.—Iron in its various forms, quinine, and mineral acids are the medicinal agents suggested by the nature of this disease. Freedom from anxiety, thorough repose of mind, entire avoidance of fatigue, gentle and regular exercise in the open air, careful attention to the state of the skin, a generous and stimulating diet at regular intervals, in moderate and equable amount at each meal, are the main hygienic indications calculated to impart tone to the system, improve the condition of the blood, and so induce a more healthy nutrition of the heart (FULLER). A salt-water sponging bath should be used daily. Most druggists supply the saline materials for such baths. In cases where digestion is sufficient, cod-liver oil, cream, and milk may be given with great advantage (TANNER). The bowels should be so regulated as to render straining at stool unnecessary.

Living as we do in a tumult of incessant excitement, hurry, and competition, "the struggle for existence" is maintained till the heart fails almost unperceived. The physician can but indicate the fact; and in the life that we now live, the disease, if it is not more frequent, is certainly obtruding itself more on public notice than hitherto.

FIBROID DEGENERATION OF THE HEART.

LATIN EQ., *Degeneratio fibrosa;* FRENCH EQ., *Dégénérescene fibroïde;* GERMAN EQ, *Fibröse Entertung;* ITALIAN EQ., *Degenerazione fibroide.*

Definition.—*A change of a very gradual kind, in the muscular substance of the heart, which results in its replacement by fibre-like elements, with scarcely any exudation or new material capable of growth, but sometimes with an extensive interstitial deposit, thickening and indurating the textures.*

Pathology.—The nature of fibroid degeneration, as usually seen on membranes and on valves of the heart, has been described in vol. i., p. 98. In the heart it has been described in *cases in which there is an interstitial deposit,* exudation, or growth of material, thickening or indurating the substance of the heart, and in which the new material and the sarcal particles within the muscular sarcolemma alike undergo degeneration (Risdon Bennett, Bird, Ogle, *Path. Society's Trans.*, vol. iii., pp. 273, 276, 281). In such cases there was evidence of previous or of existing pericarditis, and sometimes of rheumatism; the heart was hypertropic, increased in weight, and the fibrillæ exhibited undoubted evidence of extreme brittleness (Barlow, *Path. Society's Trans.*, vol. iv., p. 71; Bristowe, Peacock, Cholmeley, l. c., vol. v., pp. 84 and 102; vol. vi., pp. 147 and 148). A section showed the muscular substance of the walls of the ventricle to be increased in thickness, encroached upon, and in part replaced by some adventitious product. Towards the base, the muscular substance gradually disappeared at the expense of its outer part, so that at the distance of three-quarters of an inch from the aortic valves, and from that point upwards, it was entirely wanting, and was replaced by a firm, dense, slightly translucent fibrous tissue, which extended some little way upwards on to the aorta, and downwards on the exterior of the muscle, gradually losing itself in the substance of the pericardium.

Several masses of deposit existed in the muscular substance, of an irregular form, opaque, yellowish, and somewhat firm. This deposit consisted of cell-elements, fatty particles, and degenerate muscular fibres. The cells were spherical, and varied in size from $\frac{1}{1000}$th to $\frac{1}{200}$th of an inch in diameter, having very thin walls, easily broken down, and containing a few granules. Some larger cells showed nuclei in their interior; and they were arranged in lines, so as to give the appearance of fibres. This deposit was considered of a doubtful nature as to its being malignant, tuberculous, concrete pus, or fibrinous deposit, such as is sometimes seen in the spleen. This latter view was adopted by Dr. R. Bennett, Mr. Simon, and Dr. Quain. The deposit in the heart resembled a similar lesion figured by the late Sir Robert Carswell in the fourth fasciculus of his "Morbid Anatomy," plate 3 (*Path. Society's Trans.*, vol. iii., p. 273). In some cases the deposit or altered part appears to be circumscribed by a boundary line, where blood-globules are abundantly present; and had the process of softening continued with surrounding vascular activity, "a circumscribed abscess must have been the result; if absorption had taken place, a fibrous degeneration would have been left" (Quain, l. c., p. 281). The simultaneous occurrence and progress of both these forms of morbid process has also been observed (Ogle, l. c., p. 282). In one case, where the heart weighed *twenty* ounces, the lesion appeared to consist of lymph amongst the muscular tissue, which lymph, becoming converted into more condensed fibroid material, compressed, and ultimately replaced, the muscular substance (Bristowe, l. c., vol. vi., p. 150).

Dr. Henry Kennedy, of Dublin, has observed that *enlarged heart* forms an important element in the natural history of the affection (Ranking, *Abstract,* vol. xxx., p. 93).

ANEURISM OF THE HEART.

Latin Eq., *Aneurysma;* French Eq., *Anévrisme;* German Eq., *Aneurysma;* Italian Eq., *Aneurisma.*

Definition.—*A protrusion of the textures of the heart covered in by the endocardium and the visceral pericardium, caused by a degenerate portion of the wall of the heart yielding to the pressure of the blood.*

Pathology.—This lesion is usually a result of *myocarditis,* where a portion of the heart's texture is broken down by the effects of inflammation into a finely granular detritus. Although myocarditis is seldom seen in this form, yet its results are apparent after absorption of the fluid material in the shape of irregular patches of cicatricial membraneous substance, which, yielding to pressure of the blood, forms what is known as a true (or *chronic*) *aneurism of the heart.*

The following summary of the theories that have been held regarding the original formation of such aneurisms is given by Dr. N. Pelvet and by Mr. Henry Arnott in the *Transactions of the Pathological Society of London,* p. 151, vol. xix. They are regarded—

(1.) As a result of incomplete rupture (Breschet, Corvisart, Lobstein).

(2.) As a result of ulceration of the endocardium (Kreysig, Laennec, Bouillaud).

(3.) As a result of alteration of the lining membrane, the nature of which is not stated (Reynaud, Ollivier).

(4.) As a result of inflammatory softening (DANCE, CHASSINAT, and HARTMANN).

(5.) As a result of inflammatory changes (similar to those described in the text), by which the muscular substance is replaced by fibrous material (CRUVEILHIER, ROKITANSKY, CRAIGIE, PEACOCK, TORGET, MERCIER, THURNAM, NIEMEYER, and most modern pathologists).

Such aneurismal sacs may attain the size of a hazel-nut, walnut, or a hen's egg, or may even become as large as the heart itself. The entire heart in such cases is generally dilated.

More than eighteen examples of such forms of disease are to be found described in the pages of the *Transactions of the Pathological Society of London.* One remarkable instance is related by Mr. Henry Arnott (l. c., vol. xix., p. 149), which is a unique specimen of a cardiac aneurism, "leading out from the upper part of the left ventricle, behind and below the aortic opening, forming a large pouch, extending backwards, upwards, and to the left, with a patent orifice two inches in diameter. Its walls, two to three lines thick, had undergone calcification to a considerable extent, studding the fibroid material of which they were otherwise composed. Of eighteen cases recorded in the Society's *Transactions*, it is stated that the aneurismal pouch in fifteen sprang from the left ventricle, and in six of those from the upper part of that cavity. It is also recorded that one of the first cases related in the *Transactions* is an aneurism of the *left auricle*—a condition so rare that Rokitansky speaks of only one undoubted instance on record, besides one in the Pathological Museum at Vienna. A summary of seventy-four cases is also given by Thurnam, in fifty-eight of which the left ventricle was the seat of the aneurism, and nine of these were of a very large size. One of them was so large, and opened from the apex of the left ventricle, that it equalled the size of a second heart, and was filled with laminated coagula (*Med. Chir. Trans.*, vol. xxi., p. 227, 1858). In seventeen only of these cases was Dr. Thurnam able to obtain information as to the occupation of the sufferers; but of these seventeen cases he writes:—"It is a striking fact, that out of this number, eight, or about one-half, should have been soldiers—a circumstance that would lead one to suspect that exposure, to which this class of persons is subjected, and the forced exercise they undergo, may have something to do with the production of this lesion." Several preparations are to be seen in the Army Medical Department Museum at Netley.

Another unique case is related by Mr. Thomas P. Pick in the same volume, p. 156. Just below the semilunar valves an aneurismal pouch, the size of a pigeon's egg, existed, communicating with the left ventricle by an opening sufficiently large to admit the little finger. Another aneurism existed in the septum between the ventricles, and communicated with the aorta by a small opening at the bottom of one of the sinuses of valsalva, behind one of the semilunar valves.

Symptoms are obscure, beyond general evidence of diseased heart and the physical signs of extended dulness; but the sphygmograph may show evidence of prolonged arterial expansion, as in the case recorded by Mr. Henry Arnott, where the symptoms resembled those of aortic dilatation.

ACUTE ANEURISM OF THE HEART.

LATIN EQ., *Aneurysma acutum;* FRENCH EQ., *Anévrisme aigu;* GERMAN EQ., *Acutes aneurysma;* ITALIAN EQ., *Aneurisma acuto.*

Definition.—*Swellings caused by blood effused into the substance of the heart, owing to inflammatory softening and rupture of the endocardium and muscular tissue.*

Pathology.—This lesion is to be distinguished from the one previously described. It arises from laceration of the endocardium from endocarditis; the blood then forcing its way through the rupture, continues to tear asunder and to separate the cardiac muscular texture, and so producing a rounded circumscribed sac, seated upon the walls of the heart, as an appendage. Thus an acute aneurism of the heart is formed and bounded at its entrance by torn and ragged endocardium, its wall consisting of the forcibly separated fibres of the muscular substance of the organ (NIEMEYER).

Symptoms.—These are so obscure that instances are on record in which such serious aneurismal conditions exist in which the patient has been able to follow his usual employment " with little symptoms of so grave a state of matters, and, indeed, with so little discomfort, that he expressed himself as having enjoyed excellent health." A case of this kind is recorded by Dr. John Murray, in the *Pathological Society's Transactions,* vol. xx., p. 131. The patient—a man—had gone to bed the previous evening in his usual health, and after drinking a glass of beer in the morning he felt queer, and set off for the hospital, but fainted on the way. A double basic and a systolic apex murmur was heard over the cardiac region. He died in an hour.

RUPTURE OF THE HEART.

LATIN EQ., *Diruptio;* FRENCH EQ., *Rupture;* GERMAN EQ., *Ruptur*—SYN., *Zerreissung;* ITALIAN EQ., *Rottura.*

Definition.—*Bursting or rupture of some part of the walls of the heart, including its visceral pericardium.*

Pathology.—*Spontaneous Rupture and Ramollissement* of the walls of the heart have been occasionally met with during diseased conditions, especially *fatty degeneration, myocarditis, cardiac abscess,* and *true* or *acute cardiac aneurism.* A healthy heart never bursts, in spite of the greatest strain; but, associated with any of those diseased conditions, the heart may rupture during some unusual exertion, death usually ensuing in such cases with symptoms of internal hæmorrhage.

In these degenerated hearts the organ is flaccid, so that if an incision is made into the ventricles the walls collapse. Its substance tears with great facility. The degeneration which leads to rupture is almost always accompanied by some change in its colour, which is sometimes deeper than natural, and at others, according to Laennec, of a yellowish tint, like that of an autumnal leaf—an appearance which does not necessarily occupy the whole thickness of the muscular substance, but often merely the central layers. This degenerescence is sometimes general, but often partial, affecting only the walls of one ventricle of the interventricular partition, or the walls of one auricle. It is from this cause perhaps, rather than from any other, that patients sometimes die from rupture of the heart.

Examples of rupture of the right side of the heart are more rare than those of the left; or, according to Bouillaud, there are six ruptures of the left side to four of the right side. Rupture of the auricles is perhaps as frequent as that of the ventricles; or, out of the ten cases mentioned, four were ruptures of the right auricle and two of the left auricle. The extent of the rupture, when it takes place in the ventricle, is various. In one case the ventricle was ruptured from its apex to its base, along the sulcus which separates the two ventricles; in another the rupture was from ten to twelve lines; in a third the base of the ventricles was severed from the aorta, and one of the aortic valves split transversely. It is remarkable, however, that the rupture has seldom been found at the apex, where the walls of the heart have least force and consistency. The number of the ruptures is as various as their seat; thus, out of forty-eight cases collected by Ollivier, eight were multiple. Again, in two cases related by Rostan, there were two ruptures in each case towards the apex of the left ventricle. Morgagni gives one case, and Portal another, in which there were three ruptures in the left ventricle; and Andral met with a third, in which there were five ruptures; but, of these, three were superficial, only two opening into the cavity of the left ventricle.

Crovisart is the first who has given examples of another kind of rupture of the heart, and it is that of the *carneæ columnæ*, or tendons of the valves. It is probable that rupture of these parts is owing more frequently to ramollissement, or to induration, than to any other cause. Laennec, however, mentions a case in which it appeared to result from ulceration. In three cases related by Corvisart the rupture followed some violent exertion; and Bertin saw a case in which one of these tendons was ruptured in consequence of a violent fit of coughing. Rupture of the *cordæ tendiniæ* sometimes occurs in connection with mitral valve disease.

Evidence of such a lesion is seen after death in the ruptured filaments curling up and forming little elevations of the retracted ends, which generally become covered with fibrinous coagula and hardened lymph. When several rupture, the edge of the valve-lappet recedes, forming a broad raised margin (*Navy Medical Report* for 1868, where excellent drawings are given of such a lesion).

Symptoms.—The first symptom in all these cases has been a sudden sense of suffocation; and the patient has in general suddenly died, although in some instances he has survived a few days.

In rare instances a brief period of violent pain under the sternum has been experienced, shooting towards the left shoulder and along the arm. It is rare that rupture is survived beyond a few hours, and then only when the extravasation of blood is a mere filtration through the degenerate cardiac tissue, the rent gradually increasing. Symptoms then are less active, and physical signs may mark the progress of the flow into the pericardium. Usually, however, death is sudden, the pericardium being filled with blood flowing from an outwardly irregular but smooth-edged rent of variable length, opening into the deeper flesh of the heart, which seems torn, mangled, and imbedded in blood-clot (NIEMEYER).

Of the cases of absolutely sudden or instantaneous death from fatty degeneration of the heart, referred to under that topic and analyzed by the author, *nine* died from rupture or laceration of some part of the texture of the heart. Such was the mode of death of Dr. Abercrombie: a rent half an inch in length existed in the posterior aspect of the left ventricle, from which the pericardium was suddenly filled with blood. Such cases die of broken hearts—literally, not figuratively. Their cords are either torn asunder, or the fibres and minute vessels of the substance of the heart are lacerated. In one instance there was sudden rupture of the mitral valve. In five, the substance of the left ventricle was ruptured, two at the upper and posterior part, three across or along the anterior wall, and more or less close to the septum. In one there was rupture of the septum itself, penetrating the right ventricle.

PARASITIC DISEASE OF THE HEART.

LATIN EQ., *Morbus parasiticus;* FRENCH EQ., *Maladie parasitaire;* GERMAN EQ., *Parasitische krankheit;* ITALIAN EQ., *Malattia parasitica.*

Definition.—*Lesions caused by the localization of parasites in an immature or non-sexual condition.*

Pathology.—The nature of the parasites associated with such forms of disease has been already fully described in vol. i., pp. 116, 168, and 170. It remains only to be noticed here that cysticerci have been found in the human heart when enormous numbers of them existed at the same time in other muscles of the body. The echinococcus has also been met with (NIEMEYER).

Hydatids or Echinococcus cysts have been found in the walls of the heart, beneath the inner membrane. Dupuytren found hydatids in the thickness of the right auricle, forming a tumor projecting into the cavity as large as the heart itself. Morgagni found in an old man—who had in no degree suffered from palpitation, syncope, or irregularity of pulse, but had died of acute disease—a cyst the size of a cherry in the walls of the left ventricle. Such parasitic cysts abound in sheep and oxen.

MALFORMATIONS OF THE HEART.

LATIN EQ., *Deformitates ingenitæ;* FRENCH EQ., *Vices de conformation;* GERMAN EQ., *Missbildungen;* ITALIAN EQ., *Vizi di conformazione.*

Definition.—*Deviations from the normal development of the heart occurring in the earlier periods of gestation, and before the termination of fœtal life.*

Pathology.—After what has been already written regarding malformations in vol. i., p. 225, the more practically important and common malformations of the heart may be here simply enumerated. They may be arranged under the following heads:—

1. *Misplacements of the heart* (*Ectopia Cordis*) occurring congenitally, as in cases of transposition of the viscera. For an account of all that is known on this subject, the reader is referred to a paper by Professor Allen Thomson in the *Glasgow Medical Journal* for July, 1853; and to the *Lancet* of August 8, 1863, where the condition is described by Professor W. C. Maclean as having been recognized during life. For an excellent report on cases of *ectopia cordis*, see *Pathological Society's Transactions*, vol. vi., p. 98.

2. *Congenital deficiency or absence of the pericardium*, in which the heart is, as it were, naked, and lying in one cavity with the lungs (*Pathological Society's Transactions*, vol. iii., p. 60; vol. vi., p. 109).

3. *Arrest of development of the heart at an early period of fœtal life:*—as in hearts with one auricle and one ventricle; with imperfect separation of the ventricles; cases of contraction or absence of the pulmonary artery, the aorta arising from the right ventricle, and the *septum* of the ventricles imperfect. In cases of imperfect *septa*, the aorta sometimes arises from the two ventricles. Sometimes the ventricular *septum* is wholly absent.

4. *Premature closure of fœtal passages—the foramen ovale and ductus arteriosus—malformations causing changes which ought not to ensue till after birth.* The consequences of such premature results are chiefly cyanosis, combined with imperfect dilatation of the branches of the pulmonary artery.

5. *Irregularity of the valves, and origins of vessels, which may not in the first instance interfere with the functions of the heart, but which are apt to lay the foundations of disease in after-life.* Examples are to be seen in cases where two aortic valves occur; where there is fusion or union of two of the valves; where there is transposition of the aorta and pulmonary artery, both auricles opening into the left ventricle (l. c., vol. vi., p. 117). Excess of pulmonary valves (l. c., vol. iii., p. 301; vol. iv., p. 102). (See vol. i. of this work, p. 225.)

The **treatment** of malformations which are associated with cyanosis is mainly preventive of dyspnœa and palpitation, by the avoidance of fatigue and mental excitement, the maintenance of temperature, and especially by a nourishing diet and warm clothing.

CYANOSIS.

LATIN EQ., *Cyanosis;* FRENCH EQ., *Cyanose;* GERMAN EQ., *Cyanose*—Syn., *Blausucht;* ITALIAN EQ., *Cianosi.*

Definition.—*A peculiar blue condition of the skin symptomatic of various malformations or derangements of the heart and great vessels, so that a small portion only of the blood is subjected to aëration in the lungs.*

Pathology.— The blood-corpuscles absorbing oxygen in the lungs convey it through the circulation to every part of the body, and thereby render the metamorphosis of tissue possible. If this absorption of oxygen in the lungs by the blood-corpuscles is for any reason stopped or diminished, *arterial* blood retains the properties of *venous* blood, and the condition termed *cyanosis* arises.

In its *minor degree* it is associated with various forms of cardiac and pulmonary derangement, having the effect of obstructing the flow of blood in the veins of the lungs and of the system generally. The chief of these is constriction or partial obstruction of the pulmonary artery, combined with systemic venous engorgement. In such an acute form it may be associated with *croup, diseases of the heart,* and *cholera.*

In cases of cyanosis the skin is usually thin, the capillaries abnormally large; hence, when obstruction to the pulmonary and systemic venous circulation causes imperfectly aërated blood to flow throughout the system—and still more so in cases when, in consequence of some congenital malformation, a small portion only of the blood is subjected to the aërating influence of respiration—a dark, dusky, more or less livid hue is imparted to the skin.

In its more severe form the condition is usually associated with such a malformation, disease, or injury of the heart or great vessels, as permits venous and arterial blood to mix, and after mixture to be so distributed to the systemic capillaries. A patulous condition of the *ductus arteriosus,* an open *foramen ovale,* a deficiency of part of the *septum* of the ventricles, a heart formed of one ventricle and one auricle only—the aorta and pulmonary artery rising from a common trunk—are amongst the usual conditions which lead to cyanosis.

It is usually, therefore, a congenital affection, and the physical signs vary with the precise condition of the heart and arteries to which the the cyanotic discoloration is due. The action of the heart is usually more forcible than in health, and hypertrophy and dilatation of the right ventricle are almost always present. Deficiency of animal heat is also a constant phenomenon.

The causes of cyanosis are arranged by Vogel into two groups:—(1.) In the one group the supply of oxygen to the blood, and consequently the oxydation of the blood-corpuscles, is prevented by derangements of the respiration or circulation (*i. e.*, by causes which are external to the blood); (2.) In the other group the blood-corpuscles lose the property of absorbing oxygen. This occurs in some cases of *pyæmia* and the last stage of *pulmonary tuberculosis.* There seems to be a diminished capacity of the blood-corpuscles to redden themselves.

Treatment must depend upon the nature of the lesion or disease which gives rise to the cyanosis.

ANGINA PECTORIS.

LATIN EQ., *Angina pectoris;* FRENCH EQ., *Angine de poitrine;* GERMAN EQ., *Angina pectoris;* ITALIAN EQ., *Angina pectoris.*

Definition.—*Pain or spasm of a weakened heart* (CHEVERS), *referred to* *the lower part of the sternum, or to the præcordial region, extending through the chest to the left scapula, and up the sternum to the root of the neck. The pain is characterized by its suddenness, its severity, and by a sense of constriction or of burning. It compels the patient, if walking, instantly to stop,—almost prevents inspiration. The pain is felt likewise in the left shoulder, whence it sometimes reaches to the elbow, rarely to the hand, often with a sensation of numbness in the parts. A tendency to syncope exists, associated with intense anxiety, and a sensation of approaching dissolution.*

Pathology.— This disease had attracted little attention till Dr. Heberden, in 1772, drew the attention of the profession to it by two papers published in the second and third volumes of the *Transactions of the London College of Physicians.* He connected it with disease of the heart; and it has ever since been treated of in conjunction with diseases of this organ. It has subsequently been studied by Drs. Black, Parry, and Jenner, and by many Continental physicians; and Dr. Parry's work —*An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*—will well repay perusal, though published so long ago as 1799.

The immediate cause of the paroxysm, as shown by the most weighty testimony, appears to be a sudden impediment to the *coronary* circulation of the heart, and particularly to the return of the blood by the coronary veins. These results are in general due to a temporarily over-distended state of the chambers of the heart, and an inability in them to empty themselves, whether owing to weakness of the muscular parietes of the left ventricle or to other causes. If the cavity of the left ventricle is considerably dilated, or its walls attenuated or softened, or otherwise degenerate, the contractile power of its muscular tissue will be impaired in proportion. If the circulation under these circumstances happen to be suddenly hurried, or the heart's action suddenly disturbed, the cavity of the left ventricle may become so much distended as to render it incapable of contracting efficiently upon its contents, which would be quickly followed by distension of the auricle on that side, and, if relief were not soon experienced, by distension of the right side of the heart (Bellingham).

. The organic lesions of the heart most apt to be attended by *angina* are conditions of the aortic valves which permit of free regurgitation, with a rigid dilated state of the ascending portion of the arch of the aorta, which permits the blood from the large vessels to regurgitate into it, combined with either of the following conditions of the left ventricle:—(1.) Dilatation of the cavity; (2.) Attenuation of the parietes; (3.) Softening or degeneration of the muscular tissue (Bellingham).

Any one of those morbid conditions may be present, or two or more of them may be combined, and yet *angina* may not necessarily occur, so long as the circulation continues tranquil, and so long as the left ventricle is able to get rid of the blood which enters its cavity, and does not get over-distended. But if the heart's action becomes disturbed by some sudden mental emotion or other cause; or even without this occurring, if the stomach is loaded with indigestible food, and it and the intestines are distended with flatus, by which the cavity of the chest is encroached upon, and the heart's movements are impeded, a paroxysm of *angina* is the general result. John Hunter, who suffered greatly from this disease, used to affirm that his life was in the hands of any person or circumstance which acted powerfully on his mind; and, in fact, he ultimately died in St. George's Hospital, from strong but suppressed feelings on a point in which he was interested. Ascending a staircase or other acclivity, or indeed any active exertion, is a powerful exciting cause. In persons who have had previous attacks, the paroxysm is liable to supervene during sleep, as the result of a frightful dream disturbing the heart's action, or of considerable distension of the stomach by flatus impeding the movements of the heart.

The late Sir John Forbes, in an able article on this subject in the *Cyclopædia of Practical Medicine,* has shown that *plethora* becomes a very common complication of *angina pectoris.* The very existence of *angina* tends to produce *plethora,* if it did not previously exist; a sedentary life and abandonment of all active bodily exertions are almost inevitable consequences of the disease.

Angina pectoris ought therefore to be regarded rather as a *symptom of organic disease of the heart* than as a distinct form of disease. What *dyspnœa* is to the lungs, *angina* appears to be to the heart; so that Bellingham has termed it the "dyspnœa of the heart."

It has been defined by Romberg as hyperæsthesia of the cardiac plexus; and by Bamberger as *hypercinesis* (excessive motion), with *hyperæsthesia,* the cardiac plexus being assumed to be the source of the pain as a matter of theory only.

Its paroxysmal character, with intervals of immunity, associates its pathology with disorder of the nervous system of the heart, to which the organic changes give a predisposition (NIEMEYER).

Symptoms.—The paroxysms of this disease generally supervene suddenly, and are characterized by a constrictive anxious pain, fixed most commonly on the left lower half of the sternum, and rarely extending above the fourth rib. Occasionally, however, it extends over the whole anterior portions of the chest, along the neck to the lower jaw, into the back and shoulder, down the arm to the elbow, and even to the hand and fingers. In this course it affects externally the superficial cervical plexus and its ramifications, as well as the anterior thoracic nerves, the cubital nerve, and its divisions. The pain is also sometimes sub-sternal, and then follows the course of the nervous plexus placed between the folds of the mediastinum, and also the branches of the eighth pair which go to the large arteries and surround the bronchial tubes. The pulse is sometimes rapid, sometimes hardly to be felt; and the breathing is sometimes accelerated, at other times it is imperceptible. Mr. Hunter, when labouring under the paroxysm, could scarcely feel his pulse, and thought he should die unless he exerted his voluntary muscles to aid respiration. Many have died so—asphyxiated.

Darwin has seen the action of the diaphragm, and consequently the phrenic nerves, affected; while Laennec mentions that the lumbar and sacral nerves also partake of the pain.

Besides the parts which have been mentioned, the gastric system is much affected, the patient perhaps being in an instant distended with flatus, and only relieved by repeated eructations. The urine is sometimes suppressed during the paroxysm. In all cases where the patient is not broken down by disease, the mind is clear, but the face and extremities are cold and pale. At length the paroxysm subsides gradually, when much flatus is discharged, accompanied by a copious and almost involuntary flow of pale limpid urine and the patient for the time recovers.

The duration of the paroxysm depends on the persistence of the impediment to the coronary circulation. Sometimes the pain only lasts a few minutes, while at other times it will continue for two or three hours, a whole day, or even longer. The interval of respite from pain is likewise very uncertain—from a few hours to a few days, or a few months. Each repetition, however, increases the tendency of the paroxysm to return, and increases its violence; and at length, perhaps, an aggravated attack occurs which puts a period to the patient's existence. So seems to have died the late John Leech, as John Hunter did.

Prognosis.—The danger is in proportion to the nature and degree of the organic lesion on which the disease depends. Sir John Forbes found that of sixty-four recorded cases of angina forty-nine died, almost all of them suddenly.

Treatment.—The indications for the treatment of *angina pectoris* are to be found in a study of the lesions on which the paroxysms are found mainly to depend. Medicine can do little more than mitigate the severity of an attack; and this is generally best done by diffusable stimulants, such as *brandy*, *ether*, *chloroform*, *ammonia*, *chlorodyne.* Alcohol in small doses often repeated, *sesqui-carbonate of ammonia* in doses of *three* to *five* grains, the *muriate of ammonia* in doses of from *ten* to *twenty* grains, have each purely powerful stimulant effects. Hot bottles and sinapisms should be applied to the feet. The bowels may require to be rapidly and efficiently acted upon.

Dr. Fuller has proposed the use of digitalis in *angina pectoris.* Dr. Fothergill has used it in true angina, not mere *cardiac asthma*, where diffusible stimulants were of little use, but in which the addition of thirty *minims* of *tincture* of digitalis repeated at the end of half an hour gave great relief. Repeated attacks in the same person gave an opportunity of testing the use of the digitalis, and according as it was given or withheld, so did the benefit result or not.

It has also been beneficial when the angina was associated with fatty heart.

In that form of *angina pectoris* where there is evident distension of the right ventricle, accompanied by palpitation, dyspnœa, and lividity of the face (*cardiac asthma*), the frequent use of digitalis seems to exercise a stimulating influence on the sympathetic cardiac ganglia and on the muscular fibres (FOTHERGILL).

PALPITATION AND IRREGULARITY OF THE ACTION OF THE HEART.

LATIN EQ., *Palpitatio et tumultus cordis;* FRENCH EQ., *Palpitations;* GERMAN EQ., *Herzklopfen und Unregelmässigkeit in der Herzthätigkeit;* ITALIAN EQ., *Palpitazione ed irregolarità del cuore.*

Definition.—*Palpitation and disturbances of the action of the heart unconnected with organic mischief.*

Pathology.—The interest which attaches to this subject consists in the difficulty which sometimes exists in the recognition of *functional* as distinguished from *organic* disease; and the fact that great uneasiness and distress, both mental and bodily, result from the occurrence of functional disturbance, while its persistence is apt to induce organic disease. Death has resulted from simple functional disturbance (GRAVES).

The irregularities are of the following kind:—The heart may beat abnormally slow, may intermit, may have a rolling action, or its pulsations may be so frequent, and its action so irregular, as to be termed R*alpitation.* These states are believed to be caused by an irregular innervation of the heart, by which it is rendered morbidly sensible or insensible to its natural stimulus, the blood. The excessively slow pulse is often caused by some pressure made high up in the cervical portion of the spinal cord, or by congestion or pressure on the brain. The other states are perhaps inexplicable, and may be considered as ultimate facts. The irregular and rolling action of the heart is in general accompanied with hypertrophy, or other disease of that organ.

Fits of palpitation, however, may occur in the most healthy subjects, and in the most healthy hearts.

Palpitation is an abnormal innervation of the heart, by which its actions are rendered often highly irregular, and its pulsations remarkably increased in frequency.

The excitability of the hearts of young people is readily accumulated and as readily exhausted. Everybody is aware how powerfully every passion and every affection acts on the heart and changes its healthy beat; as also how every error in diet, or any over exertion, may produce the same effect. Every moral, as well as almost every physical cause, may consequently be the remote agent in the production of palpitation, while every pathological state of the heart may be accompanied by it.

As a primary disease, palpitation seldom occurs before puberty, but after that period it is common, and often to a most distressing degree in both sexes. The female, however, suffers more than the male, and especially during amenorrhœa, or at the period of menstruation, and in more advanced life when menstruation ceases.

That palpitation is merely a disease of the function of the heart is evident from the number of young persons who suffer from it, and who afterwards attain a hale old age. Laennec says it is generally believed that habitual palpitation of the heart at length terminates in hypertrophy or dilatation of that organ; but he adds, "I have seen nothing to establish this fact." Palpitation, however, is a common symptom of disease of the heart. Regarding palpitation as over-action of the heart, is apt to engender the belief that it indicates excess of power; on the contrary, the palpitating heart is more nearly allied to asthenia. It is evidently a laborious effort on the part of the heart, and indicates that it is over taxed. It is generally intimately connected with more or less of ventricular engorgement; especially as a result of the obstruction to circulation from diminution in the calibre of the arteries, as in Bright's disease, jaundice, and whenever poisoned blood is coursing through the vessels. The palpitation thus induced is evidence of over-taxation. The symptoms of increased action are subjective and perceptible only to the patient—the result of extreme excitement of the sensory nerves of the organ.

The forms of functional disturbance which simulate organic disease of the heart are palpitation, fluttering, or a "rolling" action of the heart, sometimes associated with a valvular murmur and irregularity, feebleness, or altered rhythm of the heart's action. The causes of such phenomena are common to adolescence and middle adult life, and are most frequently traceable to excessive mental exertion and sedentary occupation; great anxiety and strong mental emotion; nervous exhaustion from various causes—such as uterine irritation, excessive venery, masturbation; the influence of poisons on the heart's action—such as that of tobacco and alcohol; *gout*, *rheumatism*; derangements of the stomach and liver, characterized by the existence of flatulence and acidity (FULLER).

Exaggeration of the functional acts is characteristic, and the general symptoms indicate great distress, a sense of fulness and of deep oppression in the *præcordial* region, pain, breathlessness, and tendency to faintness. Frequent giddiness, with pain, heat of head, singing in the ears, flushing of the face, coldness of the extremities, are associated with forms of dyspepsia characterized by excessive flatulence, acid eructations, restlessness at night, depression of spirits, and mental anxiety as to the nature and probable issue of the disease;—all contribute to induce and maintain the functional disturbance of the heart (FULLER).

The characteristics of palpitation, as due to functional and organic causes, have been already indicated (see pp. 547), and repeated examinations of the patient are necessary to arrive at a just conclusion. Perversions of rhythm have sometimes been attributed to functional disturbance merely; but, when altered, cardiac rhythm is not merely of temporary duration, but is attended with giddiness, faintness, or actual syncope, it is more likely, in the absence of positive knowledge as to the state of the heart's tissue, that such altered rhythm is due to organic or textural degeneration. Dr. Fuller has so found it in every case he has examined.

Functional disorder of the heart (under the name of "*irritable heart*") is very common among soldiers, and is occasionally also met with in young persons in civil life. It was very common among the soldiers during the late American war, and was considered due to *cardiac muscular exhaustion* (HARTSHORNE). It was familiarly known as the "trotting heart;" and was characterized by great frequency of action, constantly recurring attacks of palpitation, and pain in the præcordial region.

Such palpitation happens chiefly during exercise, but often occurs when at rest, as in the night, hindering sleep.

Dizziness and headache are sometimes constant, or only during the fits of palpitation.

Pain may be dull and constant, or shooting and paroxysmal; its site generally over the apex, where the skin is sometimes also sensitive.

The very rapid action of the heart is accompanied by an abrupt jerky impulse of irregular rhythm, with a short sharp first sound, and a distinct second sound. Sometimes the first sound can hardly be heard, as in the asthenia of continued fever. There are no cardiac nor neck murmurs; and the area of percussion dulness is not increased. The pulse is greatly influenced by position, being less by twenty beats when the patient is lying down, and is usually compressible. The breathing is generally distressed on exertion, but otherwise there is no constant increased frequency of respiration, and the general health is often good.

The disorder is an obstinate one, and wholly unfits a soldier for active duty.

The antecedents have generally been—(1.) *Forced marches;* (2.) *Fever;* (3.) *Camp diarrhœa* (Dr. Clymer, "Da Costa's Medical Diagnosis;" Hartshorne, in *Amer. Journal of Medical Science*, July, 1864).

Symptoms.—The attack of palpitation may be sudden, or it may be preceded by acidity, flatulence, or other affection of the stomach. It has many degrees. In young persons of a delicate constitution it often occurs in a slight degree nightly; so that the patient, on going to bed, passes many hours sleeplessly, not only feeling his heart beat, but also hearing it. His subsequent sleep is unrefreshing, and he awakes in the morning more tired and jaded than when he went to bed.

In some cases, as in young women labouring under leucorrhœa, the palpitation is constant, the pulse beating for many weeks at 150 to 180 strokes in a minute. In other cases it is paroxysmal. When the paroxysm is formed, the pulse may still preserve a regular rhythm, only greatly increased in frequency, while its force may be increased or diminished. In the severest cases, however, the pulse is so rapid that it has a mere vibratory motion, and cannot be counted, while its rhythm is extremely irregular. The force of the heart's action also is at one time excessive, and at another it is not to be felt. In general the contraction of the ventricles is so rapid that it is impossible to hear the sound of the auricles; and again, so singularly irregular is the action of the heart that the auricles may contract at the same time as the ventricles, or perhaps contract three or four times for the ventricles' once; and, indeed, the heart appears to act with every possible degree of irregularity. Thus it is characterized by an accelerated and sometimes unrhythmical beating of the heart, accompanied usually by a feeling of dread and by dyspnœa. In general the other branches of the eighth pair are affected besides the cardiac branch; for the patient often becomes distressingly distended with flatus, and that almost on the instant, while his deep sighing shows the pulmonary as well as the gastric branch to be involved. The patient having lain in this state, pale, anxious, and restless for a greater or less length of time, the fit at length terminates, and the pulse perhaps is restored to its natural frequency and healthy rhythm as instantaneously as it had lost them. The patient now passes a considerable quantity of pale limpid urine, and, though feeble from exhaustion, is once more able to sit up and so far to exert himself.

The duration of the paroxysm varies; sometimes it lasts a few minutes, sometimes a few hours, while Laennec speaks of a paroxysm connected with organic disease which lasted a week. The interval between the paroxysm is also uncertain. In young persons it may occur every twenty-four hours, two or three times a week or every month, or a still longer period may elapse.

Diagnosis.—The fact of palpitation cannot be mistaken; but the nature of the irregularity is not always easy of recognition. Chlorosis, hysteria, diseases of the womb, leucorrhœa, excessive venery, must all be recognized as capable of accounting for the condition.

Prognosis.—Palpitation is seldom dangerous, unless conjoined with organic disease of the heart; and when merely an idiopathic disease in youth, it frequently subsides as the patient advances in life.

Treatment.—During the paroxysm the patient should lie flat on his back, bare his neck and chest, and allow the air to blow freely over him. The best remedies are, *camphor mixture* and *æther* ʒi., with some slight opiate, as the *syrup of poppies*, ʒj., or else *tinct. hyoscyami*, ♏xx.; and this should be repeated every quarter, or every half hour, or hour, according to the severity of the attack, till the heart's action is soothed. Cold brandy and water, as it is always at hand, is an excellent substitute for, or adjuvant to, that medicine. Again, if the attack occurs shortly after a meal, some purgative medicine should be given to clear the stomach and bowels.

Leeches to the *os uteri* in females, and to the fundament in males, sometimes relieves palpitation—proceeding in the one case from uterine hysteria, and from varicose veins and hypochondriasis in the male.

Digitalis is useful in some cases of irregularity of the heart's action; such, for example, as in—

(*a.*) Palpitation and acute depression of heart's action from *shock* (*Paralysis of the sympathetic* of Romberg), marked by a small feeble pulse, coldness of skin, cold breath, diminished temperature, and evidences of the organic system of nerves being acutely depressed. In such a case the heart's walls contract ineffectively. The heart does not respond to the stimulus of its contents, and there is failure of its action. In this condition Dr. Fothergill bears testimony that, combined with other stimulants, its administration in a case of *shock*, following parturition, was followed by the most satisfactory results. Dr. Wilks bears similar testimony (*Med. Times and Gazette*, Jan. 16, 1864). In this case the patient seemed to be in *articulo mortis*—the face livid, no pulse at the wrist, and a mere fluttering heard over the region of the heart. Brandy and ether had been given with no result; and dissolution being imminent, it was determined to administer *digitalis*, of which half-drachm doses of the *tincture* were given every hour. After four doses re-action took place, and after seven doses complete recovery occurred. The full veins and condition of the heart indicated distension and paralysis of the heart (Fothergill, *Brit. Med. Journal*, July 29, 1871).

(*b.*) The irregular action of cardiac asthenia.

(*c.*) Of palpitation in dilated heart.

The paroxysm past, the patient, though much exhausted, speedily recovers his usual health, which is generally feeble. It is useful, however, to continue the medicines which have been mentioned, but at longer intervals, for some time.

It is important in these cases, however, to counsel the patient strictly as to diet, for without such auxiliary assistance medicine is often of little service. On questioning these patients, we constantly find that the palpitation returns after tea or after breakfast, or whenever hot tea or hot coffee has been drank; and in these cases it is extremely desirable to wean the patient from all hot slops, and to induce him to drink cold water at his breakfast, and indeed at every meal; his wine also should be limited to two or three glasses of sound sherry, and should be drank diluted with water. There are few tonics so beneficial as the natural tonic of cold water; and persons once accustomed to it feel a return to a modern breakfast as a punishment rather than a gratification.

Section XII.—Diseases of the Blood-Vessels.

(*a.*) *Diseases of the Arteries.*

ARTERITIS.

Latin Eq., *Arteritis;* French Eq., *Artérite;* German Eq., *Entzündung der arterien*—Syn., *Arteritis;* Italian Eq., *Arteritide.*

Definition.—*Inflammation of the textures of an artery.*

Pathology.—Inflammation of arteries is a disease by no means rare; and the results of inflammation are common, grave, and serious, in proportion to the region where arterial inflammation occurs, especially in connection with the changes known by the name of *atheroma,* which are "in continuity with *arteritis,*" and which graduate from a condition in which no inflammatory results can be found into one in which inflammation is unmistakably present (Moxon). Mere redness of an artery, without any alteration of the inner membrane, may be simply cadaveric, owing to imbibition of blood-pigments.

The *post-mortem* appearances of true arteritis are,—great vascularity, and especially of the *vasa vasorum,* a thickened and pulpy state of the inner and lining membrane, which, having lost its glistening aspect, has a villous, rugous, or granular aspect.

Chronic endarteritis commences with relaxation and infiltration of the texture. In one form there is gelatinous thickening of the inner coat, so that a moist, pale, reddish layer seems to lie upon the inner aspect of the artery—sometimes in spots, sometimes spread over a surface. This layer consists of a liquid resembling mucus, in which fine elastic fibres and round or spindle-shaped cells lie imbedded, the whole covered by epithelium (Niemeyer).

There may also be semi-cartilaginous induration, in opaque, bluish, boiled white-of-egg-like plates, lying on the inner surface of the artery. Under the microscope numerous fusiform and reticulate cells are seen in broad fasciculi of connective tissue. The process is thus seen to be an active one, with proliferation of cells, due to the action of some irritant strain or distension.

Lymph may be seen on the surface of the vessel, and slight excoriations or superficial ulcerations may be observed here and there. The elasticity of the artery no longer exists, so that rupture takes place very readily. When inflammation begins in the middle tunic, the tissue beneath the internal coat is of a speckled redness. The spots soon become white or yellow, and elevated above the inner surface, resembling small pustules, which may even liquify, so that actual abscesses have been established in the wall of an artery (NIEMEYER). "The most important place for *arteritis* to occur is the aorta, especially the arch, where the induration and loss of elasticity is attended by impairment of function, leading, sooner or later, to death; and this is called '*aortitis*'" (WILKS).

Inflammation of the several tissues of the aorta bear the same relation to the process as described in *pericarditis*, *myocarditis*, and *endocarditis*. It is rare for the external (*tunica adventitia*) coats—the connective tissue—to be inflamed, except when inflammation or ulceration of the adjoining lymphatic glands, the trachea, or other neighbouring organs extend into the aorta. In the chronic form it is often involved in *pericarditis*, and in some cases of *endocarditis* its inner coats may be extensively infiltrated by diffused inflammatory products (NIEMEYER). The middle structures are most prone to undergo fatty degeneration, or simple atrophy.

Chronic inflammation of the internal coat of the arteries, forming the incipient stage of ossification and atheroma, is one of the most frequent of diseases (VIRCHOW, NIEMEYER).

Symptoms.—Local pulsation of large vessels like the aorta is extremely energetic; and as we know that in cases of inflammation of the carotid there is pain when the vessel is pressed upon, so pain in an inflamed aorta may be aggravated by motion. A murmur is appreciable in aortitis—a loud, rough, systolic bruit—due to the passage of blood over the rugous and unpolished state of the inner membrane (GENDRIN, BAMBERGER, PARKES). In a case related by Dr. Parkes it was heard over the third dorsal vertebra, down into the lumbar region (*Med. Times*, Feb. 23, 1850).

So long as aneurism does not occur, nor rupture or stoppage of one of the smaller vessels by the formation or arrest of a clot of fibrin, there may be few or no symptoms to indicate chronic inflammation. But if the results of chronic arteritis are extensive, especially in the form of *atheroma*, the demands upon the heart become increased, and hypertrophy may arise. If the chronic inflammation spread from the arterial walls to the valves, insufficiency and stenosis may be the result. If insufficiency of the aortic valves occur, the hypertrophy, if existing, may not be maintained, from failure or imperfect supply of blood through the coronary arteries, and so degeneration of the substance of the heart may result. Then follow all the usual symptoms of retarded circulation, with overloading of the venous system—cyanosis, dropsy, and suppression of urine. Evidence of disease in the peripheral arteries warrants the belief that *aortitis* may also be present in a more advanced stage of development.

As the peripheral vessels become more dilated, and their walls more rigid, the pulse generally feels hard and full; and the course of the elongated arteries, as seen in the temporal and radial regions in thin persons, is remarkably sinuous, and their curvature increases with each beat of the pulse, which is visible. The artery can be felt as a hard, irregular cord.

Causes.—Rheumatism, gout, and syphilis seem to have a decided influence in the development of aortitis and arteritis generally.

Treatment.—Leeches should be applied freely over the course of the vessel; and large doses of opium, or of *ether* with *chloroform,* may be given to relieve pain and dyspnœa. *Iodide of potassium* or *colchicum* may also be indicated.

FATTY AND CALCAREOUS DEGENERATION—*Syn.*, ATHEROMA, OSSIFICATION.

LATIN EQ., *Degeneratio, adiposa et calcarea*—Idem valent, *Atheroma, Conversio in calcem;* FRENCH EQ., *Dégénérescence, graisseuse et calcaire*—Syn., *Atheroma, Ossification;* GERMAN EQ., *Fettige und kalkförmige Entartung*—Syn., *Atheroma, Verknöcherung;* ITALIAN EQ., *Degenerazione, grassosa e calcarea*—Sin., *Ateroma, Ossificazione.*

Definition.—*Results of parenchymatous inflammation of the inner arterial tunics.*

Pathology.—The able summary by Dr. Moxon, in the sixteenth volume of *Guy's Hospital Reports* for 1870 and 1871, "On the Nature of *Atheroma* in the Arteries," clearly shows that the true pathology of this lesion is a result of *parenchymatous inflammation,* as originally described by Virchow, and demonstrated by Billroth, Niemeyer, and others. The nature of this process has been already fully described under the topic of "*Parenchymatous* Inflammation" in the first volume, p. 37; and also under the topic of "Endocarditis," vol. ii., p. 567.

Chronic inflammation of the internal coat of the arteries is one of the most frequent of diseases (VIRCHOW, NIEMEYER). The relaxation and infiltration of the internal coat already described, where there is gelatinous and semi-cartilaginous thickening of the inner arterial tunic, shows that development of atheroma consists in changes of the nature of parenchymatous inflammation. A microscopic examination of an atheromatous patch shows that it consists of soft yellow matter, and gives proof of a disorganized and degenerate state of the walls of the blood-vessel, by the conspicuous yellow faded-looking patches, accompanied in graver states by a stoney layer, or a soft matter, made up of the common products of tissue-degeneration—namely, granules of lime and fat, shreds of tissue and cholesterine plates (MOXON).

But these results are not obtained without an active process of morbid change preceding them, and with profuse generation of cell-elements and corresponding nutritive disturbance, owing their origin to certain irritants acting on the tunics, and especially to undue strains or distension, or to special irritants, such as alcohol, syphilis, gout, rheumatism; and in the form of *endarteritis deformans* it is an extremely common disease of advanced age. I believe that a large proportion of cases of inflammation of the larger vessels, ending in *atheroma,* are of syphilitic origin; and I believe there are similar inflammations of the mucous membrane of the large intestine, ending in forms of dysentery, which are also syphilitic. Such spots of chronic inflammation are always found at the points most exposed to strains or distensions, such as the ascending portion and arch of the aorta, and the places of origin of the vessels which pass off laterally. As the recent accounts given of atheroma by Billroth, Niemeyer, Wilks, Moxon, and others, are based on the excellent descriptions of the process given by Virchow, and which cannot be surpassed, it is given here as follows:—

"At no period in the course of this century," writes Virchow, "has a complete understanding ever been come to as to what was to be understood by the expression '*atheromatous change*' in a vessel. Some have taken the term in a wider, others in a narrower sense, but still it has perhaps been taken in too wide a sense by all. When, namely, the anatomists of the last century applied the name of atheroma to a definite change in the coats of arteries, they of course had in their minds a condition similar to that of the skin, to which, ever since the days of ancient Greece, the name of *atheroma*, *gritfollicle* (*Grützbalg*) —sebaceous or epidermic cyst—had been assigned. It is self-evident, therefore, that the idea of atheroma pre-supposes a closed sack. Nobody ever called anything in the skin an atheroma that lay open and uncovered. It was therefore a curious misapprehension when people recently began to call changes in the vessels atheromata which were not seated below the surface and shut off from the surrounding parts, but belonged to the surface. Thus, it has come to pass that, instead of an enclosed deposit being in accordance with the original meaning of the term called atheromatous, a change has frequently been so termed which commences quite at the surface of the internal arterial coat. When the matter began to be examined more minutely, and fatty particles were found at very different points in the walls of the vessel, both when atheroma was and was not present—when at last the conviction was obtained that the process of fatty degeneration was always the same, and was identical with the atheromatous change, it became the custom to unite all the forms of the fatty degeneration of arteries under the designation '*atheroma*.' Gradually, people even came to speak of an atheromatous change in vessels that only possessed a single coat, for in them, too, we meet with fatty processes.

"At all times there have, moreover, been observers who regarded the ossification of vessels as a change belonging to the same category as atheroma. Haller and Crell believed that the ossification proceeded from the atheromatous matter, and that this was a juice which, like that exuding under the periosteum of bone, was capable of generating plates of bone out of itself. Afterwards it was recognized that atheromasia and ossification were two parallel processes, which, however, might be referred to a common origin. Now it would, I think, have been logical, if in the next place an understanding had been come to as to what this origin was from which the atheromatous change and the ossification proceeded. But, instead of this, the track of fatty degeneration was pursued, and thus the atheromatous process was extended to a number of vessels in which, on account of the thinness and the simple structure of their walls, the formation of any depot, which could really be compared to an atheromatous cyst of the skin, was altogether impossible.

"The state of the matter here also is more or less very simply this, that two processes must be distinguished in the vessels, which are very analogous in their ultimate results.

"First, the *simple fatty metamorphosis,* which sets in without any discoverable preliminary stage, and in which the existing histological elements pass directly into a state of fatty degeneration and are destroyed, so that a larger or smaller proportion of the constituents of the walls of the vessel perishes.

"And, a second series of changes, in which we can distinguish *a stage of irritation* preceding the fatty metamorphosis, comparable to the stage of swelling, cloudiness, and enlargement which we see in other inflamed parts.

"I have therefore felt no hesitation in siding with the old view in this matter, and in admitting an inflammation of the inner arterial coat to be the starting-point of the so-called atheromatous degeneration; and I have, moreover, endeavoured to show that this kind of inflammatory affection of the arterial coat is, in point of fact, exactly the same as what is universally termed endocarditis, when it occurs in the parietes of the heart. There is no other difference between the two processes than that the one more frequently runs an acute, the other a chronic course.

"By the establishment of this distinction between the different processes which occur in the arteries, the difference of the course they pursue is at once accounted for.

"If we examine atheromasia a little more minutely—for example, in the aorta, where the process is the most common—the first thing we see present itself at the spot where the irritation has taken place is a swelling of larger or smaller size, and not unfrequently so large as to form a really hump-like projection (BUCKEL) above the level of the internal surface. These projections are distinguished from the neighbouring parts by their translucent, cornea-like appearance. In their deeper parts they look more opaque. When the change has lasted for a certain time, the first further metamorphosis do not show themselves at the surface, but just where the internal comes into contact with the middle coat, as has been very well described by the old writers. How often have they distinctly contended that the internal coat could be stripped off over the affected spot! Hence arose the description of Haller, that the pultaceous atheromatous mass lay in a closed cavity, as it were a little cystic tumor between the internal and middle coat. The only mistake was that the tumor was regarded as a distinct body, separable from the coats of the vessels. It is rather the internal coat itself which, without any well-defined limits, passes into a state of degeneration within the prominent spot. The farther this degeneration advances, the more distinctly does an enclosed collection arise out of the destruction of the deepest layers of the internal coat; and at last it may be that the swelling fluctuates, and that upon cutting into it the pultaceous matter is evacuated, like the pus when an abscess is cut into. Now if the mass be examined which is present at the close of this process, numerous plates of cholesterine are seen, which display themselves even to the naked eye as glistening lamellæ—large rhombic tablets, which lie together in large numbers, side by side, or covering one another, and altogether produce a glittering reflection. In addition to these plates, we find, under the microscope, black-looking granule-globules, in which the individual fat-granules are at first very minute. These globules are often present in very large quantity; some of them are seen breaking up and falling to pieces, particles of them swimming about as in milk. Besides these there are amorphous fragments of tissue, of larger or smaller size, which still cohere, and are rather due to the softening of the rest of the substance of the tissue which has not undergone fatty degeneration; and in them heaps of granules are here and there imbedded. *It is these three constitutents together, the cholesterine, the granule-cells and fat granules, and finally the large lumps of half-softened substance, which give the atheromatous matter its pultaceous character,* and really produce a certain degree of resemblance to the contents of a pultaceous—sebaceous, epidermic—cyst (*Grützbeutel*) of the skin.

"With regard to the cholesterine, it is by no means a specific product appertaining to this kind of fatty transformation alone. On the contrary, we see, in every case where fatty products remain stagnant for a considerable time within a closed cavity in which but little interchange of matter can go on, that the fat sets free cholesterine. All the masses of fat which we meet with in the body contain a certain quantity of cholesterine in combination. As to whether the cholesterine which is set free had already previously existed, or whether a real new formation of it takes place in the parts, not a word can as yet be said, inasmuch as no chemical fact has, it is well known, been made out which throws any light upon the manner in which the formation of cholesterine is effected, or upon the substances, out of which cholesterine may be formed. This much, however, we must hold fast, that cholesterine is a product set free at a late period from stagnating, and particularly from fatty matters.

"If we trace the development of the atheromatous condition a little farther back, we come—anteriorly to the period when the pultaceous matter is found in the seat of the atheroma—to a stage, where nothing more is found than fatty degeneration in its ordinary form of granule-cells, and we distinctly convince ourselves that the process in this stage absolutely differs in no respect from that which, in the case of the heart and kidney, we have just declared to constitute the stage of fatty metamorphosis. At this period, immediately before the formation of the depot, the state of matters, as seen with a high power, is about as follows: On making a section we see the fatty cells which are interspersed through the tissue becoming larger towards the middle, and lying more closely together, but generally bearing the form of cells. As we proceed from within outwards, they become smaller and less numerous. All these cells are filled with small fatty granules which strongly reflect the light. Hereby is produced what looks to the eye in a section like a whitish spot. Between these fatty corpuscles runs a meshed basis-substance, the really fibrous stroma of the internal coat, which we plainly see continued towards the exterior into the normal internal coat.

"This fact, that we are able to acquire the direct conviction that the fibrous layer which lies over the depot is continued into the fibrous layer of the neighbouring normal portions of the internal coat, is one of especial value in the interpretation of these processes. In this manner the view which was for a considerable time defended by Rokitansky (that the affection consists in a deposit *upon* the internal coat) is also refuted. In a vertical section it is distinctly seen that the most external layers run in a curve over the whole swelling, and return into the internal coat; and the old writers were quite right when they said—speaking of a stage in which the formation of the atheromatous depot had already made considerable progress—that the internal coat over the whole of the depot could be stripped off in a piece. On the other hand, however, we can convince ourselves that the inferior layers of the internal coat run directly into the depot, and that their continuity has been broken by their degeneration; so that we have not to deal either with an interjacent deposit (between the internal and middle coat), as the old writers supposed, but the whole of what we have before us is degenerated internal coat.

"In some, particularly violent cases, the softening manifests itself even in the arteries, not as the consequence of a really fatty process, but as a direct product of inflammation. Whilst at the circumference a fatty softening takes place, in the centre of the seat of change a yellowish cloudy appearance is seen to arise, whereupon the substance almost immediately softens and disintegrates, and a mass of coarse crumbling fragments is found which fills the centre of the atheromatous depot.

"In the last place, it is a question where the seat of the fatty degeneration really is. Here, too, again (as in the cornea) it may be imagined that the fat is deposited in spaces intervening between the lamellæ; and even now there are still a small number of histologists who will not admit that connective tissue contains only cells, and no empty spaces. But if a section through one of these (atheromatous) patches be examined from below upwards, it is seen that the same structure which presents itself in the fatty parts shows itself also in the merely horny or half-cartilaginous layers. Bands of fibres, in the intersections of which small lenticular cavities appear, are found there, as they are also in the normal condition of the internal coat; but in the cavities and in the bands of fibres lie cellular elements. The enlargement which the part undergoes in consequence of the process, and which we call *sclerosis*, depends upon this—the cellular elements of the coat increase in size, and a multiplication of their nuclei takes place, so that spaces are not unfrequently found in which whole heaps of nuclei are lying. This is the mode in which the process sets in. In many cases division occurs in the cells, and a great number of young cells are met with. These afterwards become the seat of the fatty degeneration, and then really perish.

"Thus we have here an active process, which really produces new tissues, but then hurries on to destruction in consequence of its own development. But one who knows that the fatty degeneration is here only a termination, and that the process is really a formative one, inasmuch as it begins with a proliferation, he can readily imagine the possibility of another termination, namely, *ossification.* For here we have really to do with an ossification, and not merely, as has recently been maintained, with a mere calcification; the plates which pervade the inner wall of the vessel are real plates of bone. Since they form out of the same sclerotic substance from which, in other cases, the fatty mass arises, and since a real tissue can only arise out of a pre-existing one, it follows of course that, when the process terminates in fatty metamorphosis, we cannot assume this to consist in a simple dissemination of fatty particles which has taken place in whatever interspaces we like to fix upon.

"The essential difference which exists in a large vessel—as, for example, the aorta—between this process (*atheroma*) and simple fatty degeneration is therefore this, that in the latter a very slight swelling arises on the surface of the internal coat, a swelling which at once disappears if the superficial layers be removed by a horizontal section, and beneath which there still remains a portion of the coat unaltered. In the other case, on the contrary (that of atheroma), we have, in the extreme stage, a depot which lies deep beneath the comparatively normal surface, covered in by a thin film of internal coat, which separates the greasy paste from the current of the blood—called an *atheromatous pustule*—which afterwards bursts, discharges its contents, and forms the *atheromatous ulcer*. This commences as a small hole in the internal coat, through which the thick, viscous contents of the atheromatous depot are squeezed out on to the surface in the form of a plug; gradually more and more of these contents are evacuated and carried away by the stream of blood, until at last there remains a larger or smaller ulcer, which may extend as far as the middle coat, and indeed not unfrequently involves it. We have, therefore, always to deal with serious disease of the vessel, leading to just as destructive results as we see in the course of other violent inflammatory processes.

"In the valves of the heart, also, we find simple fatty degeneration taking place both at the surface and deep beneath it. The process generally pursues its course so latently that no disturbance is perceptible during life, nor are we able, in the present state of our knowledge, to name any very obvious anatomical change as being the subsequent result of it. On the other hand, what we call endocarditis—what can be demonstrated to arise in the course of rheumatism, and may indubitably appear as a sort of equivalent to the rheumatism of the peripheral parts—begins with a swelling of the diseased spot *itself*. There is, namely, no exudation, but the cellular elements take up a greater quantity of material, and the spot becomes uneven and rugged. Then we see, when the process runs its course somewhat slowly, either that an excrescence, a condyloma arises, or that the swelling assumes a more mammillated form, and afterwards becomes the seat of a calcification which may produce real bone. If the process runs a more acute course, the result is either fatty degeneration or softening. The latter gives rise to the ulcerative forms, in which the valves crumble to pieces, drop off, and embolical deposits are produced in remote parts" (Chance's Translation of Virchow's *Cellular Pathology*, p. 363).

Although, therefore, *atheroma* has generally been regarded as evidence of degeneration, and the term "*atheromatous*" as descriptive of "the defaced interior of arteries, as commonly seen in people of middle age or older," yet it seems clear that *atheroma* ought really to carry with it a meaning of active change, and be held as evidence of a previously active proliferation of cell-elements, the final ending of an *arteritis.*

OCCLUSION OF ARTERIES.

LATIN EQ., *Arteriæ occlusæ;* FRENCH EQ., *Occlusion;* GERMAN EQ., *Versachliessung;* ITALIAN EQ., *Chiusura.*

Definition.—*Lesions (local) which lead to a stoppage of the flow of blood through an artery at the seat of lesion.*

Pathology.—Exclusive of abnormal narrowness and contractions, often congenital, *occlusion of arteries* arises from the following lesions, as described by Rokitansky:—

(1.) *Contractions and obliterations,* as parts become disused in the course of development.

(2.) In consequence of disease in the coats of the vessel, especially by obliteration of the mouth of a vessel, occasioned by the excessive growth of the lining membrane within the trunk—a condition followed by atrophy of the vessel itself. The process may be seen on the inner surface of the aorta itself as laminæ of a delicate membrane, the growth of the inner coat itself. In smaller vessels the affected part contracts, until the passage is finally closed by the new growth blocking it up. A clot or plug forms, limited by the movements of the colateral circulation; and at last the artery shrivels and becomes atrophied throughout the extent of this coagulation and obstruction, becoming dilated above it.

Another form of occlusion, in consequence of excessive deposition, occurs when fibrinous vegetations form at the rough inner surface of the vessel, which may be *cretified, ossified,* or otherwise rough. Such may be seen in the smaller branches of the femoral arteries, leading to cases of senile gangrene.

(3.) Occlusion of arteries arising from different varieties of coagulation of blood:—

(*a.*) *Occlusion from arteritis.*

(*b.*) *Occlusion* from coagulation of blood, depending on an internal cause, such as Velpean's case of closure of the aorta from the third lumbar vertebra downwards, with a part of the iliac artery, owing to coagulation of a cancerous character in a case of malignant disease.

(4.) Contraction and obliteration from persistent pressure, as may be exerted by different tumors. The obliteration, however, is very rarely complete, unless complete coalescence of the opposed lining membrane takes place. The remainder of the occlusion is then completed by a plug reaching to the nearest branch, beyond which the vessel is finally obliterated, as in the case of tying of an artery.

The College of Physicians have recognized only two forms of occlusion—

(*a.*) From compression.

(*b.*) From impaction of coagula.

Under this latter two lesions are particularly referred to, namely:—

1. *Thrombosis* (local coagulation), the formation of a clot *in situ.*
2. *Embolism* (coagula conveyed from a distance).

A distinct knowledge of the nature of each of these lesions is necessary in order to appreciate the nature of the phenomena which are associated with each. There seems to be great ambiguity and uncertainty in the use of the words "*thrombosis*" and "*embolism.*"

The phenomena of embolism from the left side of the heart are apt to be expressed by gangrene of distant parts, as of the limbs, softening of the brain, or loss of vision. Gangrene, or decomposition of puriform matter, occurs most actively in the lungs, because here the process is brought most intimately in contact with the outer air; and here, therefore, the influence of impure air becomes so much more injurious; but cases of arterial embolism developed in this way (*i. e.*, primarily in the lungs) are not as yet known to be numerous.

The sources of embolism, or of coagula conveyed from a distance, imply some knowledge of the circumstances under which the blood spontaneously coagulates, *in situ*, or permits the separation of its fibrin to take place in the living vessels. Such are known as *ante mortem* clots or *thrombi;* and such process of clot formation, "*thrombosis.*"

In a pathological point of view, the following events in their order may be regarded as the most important:—

(1.) Local disturbance to circulation.

(2.) Stoppage or retardation, quiescence or slowness, of the blood-stream—*failure of heart's action;* compression or constriction of the blood-vessels.

(3.) Contact of rough surfaces, causing cohesion of red or white blood-cells into a mass, as a result of diseased artery.

The sources of local disturbance to the circulation are—

(1.) Irritation of the outer coats of the blood-vessels.

(2.) Changes which take place in them.

(3.) The formation of a clot and increase of coagulating fibrine.

(4.) Changes taking place in it, which furnish ample materials for further mischief; or

(5.) Organization of the clot takes place when it is converted into connective tissue, and generally remains harmless.

Arterial emboli, or coagula conveyed from a distance, are observed as a consequence of—

(1.) Gangrenous phlebitis of the pulmonary veins or of pulmonary tissue (*rare*).

(2.) Organic affections of the aortic or mitral valves on the left side of the heart, fibrinous concretions, and warty excrescences (*common*).

(3.) Atheromatous disease of the inner membrane of the artery. A first obstruction is generally followed by others—multiplicity and complexity of lesions being one of the characters of the disease. The most frequent sites of arterial emboli are—

(1.) Arteries in the fissure of Sylvius.

(2.) Internal carotids.

(3.) Arteries of the lower and upper extremities.

(4.) Splenic and renal arteries.

(5.) External carotid and mesenteric arteries.

The symptoms, in sequence, characteristic of the lesion may be noted as follows:—

(1.) Valvular disease.

(2.) After exertion, there occurs—

(3.) Palpitation, with the cessation of which—

(4.) The pulse disappears in the affected artery below the site of the plug—*e. g.*, in the *radial*—if the plug is arrested in the *brachial*, with pain in the hand of that side. These phenomena are all due to the sudden separation of fibrin from the warty growths on the valves. The particles are suddenly separated, and, being carried along with the stream of blood, become fixed in the artery. Recovery may ensue for a time; but

(5.) Repeated subsequent attacks occur, each associated with palpitation and irregular action of the heart; and, with the cessation of the cardiac symptoms, sudden and simultaneous obstruction of peripheric arteries.

(6.) At last gangrene ensues in some parts, and death results (MALMSTEN, of Sweden). In the brain such infarction leads to yellow softening. In the spleen and kidney the infarction consists of a conically-shaped mass of material exactly limited, of a colour varying with the size of the lesion, and generally more dense than the surrounding parenchyma (*Med. Times and Gazette*, vol. xvi., p. 273). The obstruction is generally at the point of narrowing of arterial branches immediately after bifurcation (*splenic pencilli*).

(7.) Strong pulsation of vessel on cardiac side of occlusion (TUFNELL).

The origin of the clot in arteries, as to where it is formed, is not generally satisfactorily determined. The coagula, which exist without simultaneous lesions of the arterial wall or adjoining capillary circulation, are never formed on the spot, but have been separated from some distant point in the circulation, and have been carried by the current of the blood as far as they could go. Such are genuine emboli. They are always found in places where a large arterial trunk, by giving off branches, suddenly acquires a more constricted calibre. Virchow, Paget, Malmsten, and Kirkes have shown, independently of each other, that—(1.) Fibrinous concretions may separate from the valves of the heart during life; (2.) That they may cause obstructions of particular peripheric vessels; (3.) That by admixture with blood they may have a poisonous decomposing influence similar to typhus or pyæmic poisons. In the lungs, for example, apoplectic foci become developed. Infarctions occur, and their results, in broad masses of fibrin, remain. On the arterial side of the circulation metastatic fibrinous wedges occur in the capillary vessels; petechial spots occur on the skin and on mucous membrane, pericardium, and peritoneum. Similar fibrinous deposits accumulate in the spleen. (See also, under *Phlebitis*, "Disease of the Veins."

ANEURISM OF THE AORTA.

LATIN EQ., *Aneurysma;* FRENCH EQ., *Anévrisme;* GERMAN EQ., *Aneurysma* —Syn., *Schlagadergeschwulst;* ITALIAN EQ., *Aneurisma.*

Definition.—*A spontaneous circumscribed partial dilatation of some portion of the aorta, consequent on lesion or degeneration of some of its walls.*

Pathology.—Aneurisms of the aorta most frequently arise from the ascending portions of the arch, and seem to spring most frequently from those parts of the vessel against which the current of the blood is most forcibly directed. The effects produced vary with the seat and the size of the tumor; and aneurism is generally one of the most distressing and puzzling of thoracic diseases.

Uniform dilatations of the entire tube are not regarded as aneurisms. The *chronic endocarditis,* resulting in *atheroma,* as described in the previous pages, is the local lesion which most frequently gives rise to aneurism, so that the morbid process which leads to aneurism is the same as that which leads to atheroma. Next in frequency is the simple fatty degeneration, without any preliminary thickening or cell-growth, in the tunics. From the outset there is opaque, white, or yellowish-white spots, slightly prominent above the surface, consisting of deposits of fat molecules in the tissue of the arterial coats. The third most common local lesion which leads to aneurism is simple thinning of the aortic wall, not uncommon among elderly people (NIEMEYER).

In consequence of some one or other of these changes, the aorta loses its elasticity, so that one part at last gradually yields, and becomes dilated by the pressure of the blood. Then, generally, on the occurrence of some sudden strain, the circular fibres of the arterial coat give way, leaving nothing but the outer and inner coat, the dilatation of which then goes on more rapidly. This event is sometimes recognized and remembered by patients as on the occasion of a sudden and violent muscular effort, lifting a heavy weight, and the like.

Dr. Moxon, with reference to atheroma as a lesion preceding aneurism, considers that the inflammatory changes in the blood-vessels tend more to produce aneurisms than do the atheromatous patches; so that the inflammatory changes alternately tend to the one or other of these results—namely, to aneurism oftener than to atheroma.

Aneurisms are now classified as being either (*a.*) *circumscribed;* or (*b.*) *diffuse.*

(*a.*) Circumscribed aneurisms are dilatations of a short portion of artery, which is sometimes widened in all directions, the tumor involving the entire diameter. More frequently, however, one side only is dilated, and the aneurism assumes the appearance of a tumor situated on the side of the vessel. Such circumscribed aneurismal tumors may have, again, secondary sac-like pouches, or dilatations of their walls (*saccular aneurisms*). At the outset, the tumor generally corresponds to the character of a *true aneurism of Scarpa,* consisting of all the arterial coats, but when the sac has attained some magnitude, the inner tunic only extends for a short distance into it. When still more fully developed, the middle tunic too dwindles away, and totally disappears, with traces here and there of the inner coat in a state of degeneration (NIEMEYER).

(*b.*) A diffuse aneurism involves a considerable extent of the entire calibre of the vessel, and if the dilatation ends abruptly, it is a *cylindrical* aneurism, and if gradually, it is a *fusiform aneurism.* Such are usually met with in the ascending and transverse portions of the arch of the aorta. On the walls of such arteries circumscribed pouches often form (NIEMEYER).

The College of Physicians require that the following forms be distinguished—(*a.*) *Fusiform;* (*b.*) *Saccular;* (*c.*) *diffuse,*—*i. e.,* where the sac is formed by the surrounding tissues.

The size of aortic aneurism varies from small walnuts, as within the pericardium, where they rarely attain any very great magnitude before giving way to growths of very large size. Outside the pericardium they may attain a very large size, pushing aside parts, and projecting in large tumors from the usual levels of the chest, destroying the sternum, ribs, and vertebræ by their constant and increasing pressure. The heart is usually hypertrophied.

From the ascending aorta aneurisms usually project towards the right half of the sternum, and become visible in the region of the upper ribs and costal cartilages of the right side. In the majority of cases they break into the right pleural sac, or burst externally. They are most frequent on the ascending portion of the aorta, before the origin of the innominate artery, and more common on the convex than the concave side.

Symptoms.—Aneurisms opening upon mucous surfaces, especially upon the air-passages, are generally attended with small and irregularly-repeated hæmorrhages. The persistence of these trifling amounts of blood in the expectoration justifies suspicion of aneurism, in the absence of any other circumstances to account for it.

Tumors, such as are caused by aneurism, often give rise to such symptoms as are suggestive of laryngeal disease; therefore, in all cases, the larynx ought to be examined on the one hand by the larnygoscope, and on the other a physical examination of the chest should be made for the signs of an aneurism or tumor. The frightful and agonizing dyspnœa is generally due, in cases of aneurism, to implication of the recurrent laryngeal nerve, and not to ulceration or disease of the larynx.

Generally, it may be stated that the symptoms are the result—(1.) Of crowding together and compression of the organs within the thorax, caused by the growth of the aneurism; and (2.) Of obstruction to the circulation. Aneurism of the arch, pressing on the trachea, generally causes the most intense dyspnœa, accompanied by a whistling sound on breathing and coughing.

The dyspnœa becomes spasmodic, asthmatic, and laryngeal, in paroxysms, if the pneumogastric or recurrent laryngeal nerves be stretched or irritated.

In some cases the dyspnœa may have a spasmodic asthmatic character, without any tendency to laryngeal spasm, associated with the persistence of small hæmorrhagic expectorations. In such cases the aneurismal tumor presses upon the bronchi towards the roots of the lungs, evidence of valvular disease and of pneumonia being absent.

In another class of cases, paroxysmal sufferings, in the form of *angina pectoris,* have their origin in the interference by an aneurism with the thoracic nerves, or with the venous circulation in the heart itself.

The variable character of the hæmorrhage, and the remarkable intermissions of such hæmorrhages from aneurismal sacs, are symptoms of thoracic aneurism which are now only beginning to be appreciated in their proper light, especially since the case of Mr. Liston drew the attention of the profession to them. Aneurismal hæmoptysis may occur in one of the three following forms:—(1.) A frothy bronchitic sputum streaked with blood; (2.) A rusty sputum, very like pneumonia, but usually more abundant, more frothy, and less viscid; (3.) A deeply dyed purple or brownish-purple sputum, like the so-called "prune juice" expectoration, characteristic of the third stage of pneumonia, and of certain forms of pulmonary hæmorrhagic condensation from valvular disease of the heart; (4.) Any of the preceding forms of hæmorrhage alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. The bronchitic varieties of sputum, either stained or streaked in different proportions with blood, occur chiefly in tumors pressing on the trachea and larger bronchi, and not producing consolidation of any part of the lung; while the "prune juice" sputum occurs when the lung is directly involved in the tumor, (W. T. Gairdner's *Clinical Medicine,* p. 454, *et seq.*)

Permanent contraction of the pupil on the affected side is in some cases another sign of aneurism, which Drs. Reid, Gairdner, Ogle, and others have clearly demonstrated.

The co-relation of the symptoms, as possibly indicating thoracic aneurism, is the main point for attention; and in addition to those noticed, raucous voice and aphonia are found sometimes associated with the lesion. Undue pulsation, dyspnœa at intervals, oppression at the præcordial region, with difficulty of swallowing solid food, are, in combination, significant of aneurism.

But the main combination of symptoms are those of compression of the parts within the chest and retarded circulation combined,—

(1.) *Compression of parts,* as of the right auricle, vena cava, or innominate vein, may lead to superficial venous congestion and dropsy of the upper half of the body, with headache, dizziness, and buzzing in the ears, and even fits of unconsciousness. If the intercostal nerves and brachial plexus are subjected to compression, violent pains, generally paroxysmal, are experienced in the right side of the chest, the arm-pit, and right arm. Compression of the *arteria innominata* of the left subclavian may render the radial pulse extremely small or quite imperceptible; and an appreciable inequality between the pulsations at the wrist of either side may also be a result of distortion of the arterial mouths, or of their stoppage by clots (NIEMEYER).

(2.) *Retarded circulation,* added to the phenomena already mentioned, is of great importance. The strongest evidence of this kind consists in the *pause, often so distinctly perceptible, which occurs between the beat of the heart and the wave of the arterial pulse,* AT A POINT BELOW THE ANEURISM. This phenomenon is most striking when the aneurism is situated between the points of origin of the great vessels of the arch. The pulse is then felt *later* at one wrist than the other.

The sphygmograph is calculated to yield important aid to diagnosis (SANDERSON, ANSTIE, and FOSTER), by showing in the pulse-trace the modifications in the movement of the blood produced by the diseased condition. These modifications are intimately dependent on the seat of the tumor, its size relatively to the vessel with which it is connected, and the elasticity of its walls (FOSTER).

The more important modifications of the pulse may be considered:—*first*, As to aneurism so situated on an artery that the pulse can be observed on the vessel below the tumor; and, *secondly*, Aneurisms affecting the aorta.

The beat of an artery, when carefully felt below an aneurismal tumor which implicates it, is found to present unusual characters: it is weakened, and generally retarded. The sphygmograph shows that modifications of the pulse occur both in its form and force. In the pulse-trace collected from an artery below an aneurism, the movement of the blood in the vessel approaches rather to that which is normally seen in the smaller arterial branches. The vertical line of ascent disappears; and often this line approaches in length that of descent. Thus we have a more feeble pulsation given to the finger; and, as the summit is slow to occur, there is a sensation of apparent retardation felt in addition. The following figures (42 and 43) represent the modifying influence of an elastic aneurismal sac on the pulse-trace of the left radial artery (DR. B. W. FOSTER). There was an aneurism of the left subclavian artery within the thorax. The right pulse shows the trace of another lesion under which the man laboured—namely, insufficiency of the aortic valves. So characteristic of the conditions in this case were the above traces, that they alone enabled Dr. Foster to arrive at the diagnosis to which ordinary means had led those watching the case. At the time when the above traces were taken, the tumor was evidently large and very elastic—hence the great modifications in the pulse-form produced.

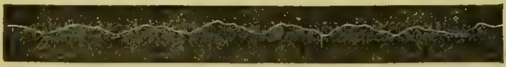

Fig. 42 *

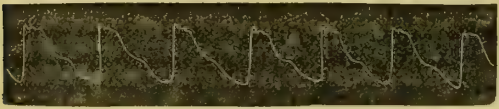

Fig. 43.†

In *aneurisms of the aorta*, very much less striking indications are afforded by the pulse-trace. Nevertheless, Dr. Foster's experience leads him to believe that the position of a thoracic aneurism may be indicated by the pulse changes—*e. g.*, when a small aneurism or even a considerable dilatation of the ascending aorta exists, the pulse of the right radial is generally reduced in size when compared with the left (Dr. Foster's *MS. Notes*).

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* Left radial. † Right radial.

The indications pointed out by Marey are,—(1.) Modifications in the force of the pulse; (2.) Modifications in the intensity of the dicrotism; and (3.) The existence of a constant difference in the pulse-form of the two radial arteries.

(1.) The force of the pulse, according to Marey, is seldom much diminished—a character of small value in diagnosis. The causes of this want of change in force reside probably (*a.*) In the small size of the tumor relatively to the volume of the aorta; (*b.*) In the fact that the sac is sometimes not placed in the direct route of the blood, but communicates with the vessels, and thus has a much less transforming effect upon the blood-movement than a tumor which must be traversed by the current; and (*c.*) In the thickness and slight elasticity of the wall of the sac often found in aneurisms in this situation. The force of the pulse, too, in these cases is altered very often, not in one artery alone, but in the vessels of both sides of the body; and, on the other hand, it must be remembered that the tumor, by compressing the orifice of one of the branches of the aorta, may cause a peculiarity in the radial of one side.

(2.) The modification in the dicrotism may exist in one or both radial pulses, and is occasionally a sign of much value.

In aneurisms of the descending thoracic aorta the dicrotism proper is often much increased in both pulses, but especially in the right, while the left radial is smaller than the right. This latter fact may be explained by the relation of the innominate and left subclavian to the blood current, the former of which receives the full force of the blood discharged from the ventricle; but the current has to deflect itself slightly to reach the left subclavian, and is partly drawn into the descending thoracic aorta, where the aneurismal sac gapes to receive it. The dicrotism is increased in these cases by the contraction of the aneurismal sac (DR. B. FOSTER).

(3.) The presence of a constant dissimilarity in the pulse-traces of the radial arteries is the most valuable sign in the diagnosis of aortic aneurisms. In many cases the finger can perceive a want of parallelisms in the beats of the radials, but often this is too slight to be detected by the finger. It sometimes shows itself in the trace by a slight difference in the dicrotism only; at others, the difference in form is more evident. When the tumor is so situated that it can be handled, we can gain valuable evidence as to its nature by observing the changes in the tension of the arteries, produced by its alternate compression and relaxation (DR. B. FOSTER).

Diagnosis.—Tumors (*cancers*) are the most common lesions with which aneurism may be confounded. For the points of differences the student must consider the subject of cancer, p. 903, vol. i., and contrast the lesions of the two conditions, *cancer* and *aneurism.*

Causes.—The causes of thoracic aneurism are exceedingly obscure; but there is good reason for believing that the morbid conditions associated with *alcoholism, gout, rheumatism, syphilis,* are the circumstances under which aneurisms are most apt to be developed, the elasticity of the vessel being impaired by structural changes, the result of a chronic *endarteritis.* With regard to the influence of syphilis, I may here observe that I dissected, during four years (at Fort Pitt and at Netley

Hospitals for invalids), twenty-six bodies of soldiers, in each of which a distinct history of syphilis was present, associated with unmistakable syphilitic lesions; and of these twenty-six cases, *seventeen* had the coats of the thoracic aorta impaired by characteristic changes—changes which are uncommon at an early period of life, and which I have every reason to believe were due to syphilis—a *syphilitic arteritis.* The changes are obvious, from cicatricial-like loss of substance of the inner coats, small local dilatations of the artery, and in several cases aneurismal expansions, one as large as an orange, which proved fatal. A characteristic case of aneurism of the thoracic aorta resulting from syphilis is also recorded by Assistant-Surgeon Alfred Lewer, in the *Medical Report of the Army Medical Department* for 1862, p. 512.

Syphilis may not be so prominent a cause of aneurism of the aorta as occasionally occurring and striking cases might lead us to believe. In common with gout and rheumatism, it certainly tends to impair the nutritious and functional properties of the blood-vessels, especially affecting their outer coats and innermost connective tissue, so that any mechanical straining of the vessel finds out its weakest part, and readily sets up an *endarteritis*, resulting in atheroma, or in aneurism, or in both. There is very strong evidence in the following records, collected by Staff-Surgeon Dr. Peter Davidson, as to the influence of *atheroma* in the production of *aneurism.* In 114 *post-mortem* examinations of soldiers dying at Netley, he found twenty-two cases of *atheroma* of the aorta. Of these, seventeen had a syphilitic history, one was doubtful, and four had had no syphilis, but had heart and lung diseases. Of the whole 114 cases, seventy-eight had no syphilitic history, and four had cases of *atheroma*, or 5·1 *per cent.*; twenty-eight had a marked syphilitic history, and seventeen had *atheroma*, or no less than 60·7 *per cent.* (*Army Med. Dep. Report*, vol. v., p. 481.)

Men suffer from aortic aneurisms much more frequently than women; and the majority of aneurisms are found in persons who are called upon to make violent muscular efforts (NIEMEYER), especially under circumstances of restraint from clothing or carrying weights.

It is now undoubted that there is an excessive production of heart and blood-vessel disease in the army compared with civil life, and that the army excess is especially in aneurism—which Dr. Lawson finds to be eleven times more frequent among soldiers than in civil life (Parkes, *Hygiene*, p. 538).

One condition—that of exertion, often rapid and long-continued—is constantly acting in the case of soldiers; and certain arms of the service always show a greater loss of strength from diseases of the heart and vessels than others. The following is the order in which the arms of the service appear to me to suffer most from these diseases, beginning with those who suffer the greatest:—*Military Train, Royal Artillery, Cavalry, Royal Engineers, Depot Brigade, R.A.; Coast Brigade, R.A.* The exertions connected with the duties of all of these arms of the service are undertaken with a bad arrangement of dress and equipments. The cavalry and artillery men are very tightly clothed, and the men are overweighted, notwithstanding that the horse carries some of the burden (PARKES). The pernicious influence of exertion carried on under unfavourable conditions is clearly shown in the observations made by Dr. Parkes, or carried out by others at his suggestion. "In the third Report of the Knapsack Committee are some experiments made by the surgeon of the Royal Marines at Gosport. Twelve men, with an average pulse of 82·6, after running 500 yards without knapsacks, had an average pulse of 114 (highest, 124; lowest, 104). After running 500 yards with the old knapsack, the average pulse was 148 (highest, 164; lowest, 132). All these experiments were made with comparatively light weights; but when the man is in full service order, the effect is much greater still, and the rapidity of the pulse continues for a long time after the work is done. With the knapsack alone the effect on the pulse is considerable. Thus, four strong soldiers carried the old regulation knapsack, service kit, great coat, and canteen, but no pouch, and no waist-belt (except in one man). The pulse (standing) before marching was on an average 88; after thirty-five minutes it had risen on an average to 105; after doubling 500 yards it had risen to 139; and in one of the men it was 164, irregular and unequal. After the double they were all unfit for further exertion. In a fifth man, who was not strong, the thirty-five minutes' marching raised the pulse to 120 from 94; after doubling 250 yards he stopped; the pulse then could absolutely not be felt. In another series the average pulse of four men, with the knapsack only, was 98 standing; after one hour's march, 112; after doubling 500 yards, 141" (Parkes's *Prac. Hygiene*, p. 540).

Assistant-Surgon Arthur B. R. Meyers, of the Coldstream Guards, in an excellent essay (which obtained the Alexander Prize in 1870), "On Diseases of the Heart among Soldiers," assigns the production of the majority of aneurisms of the aorta in the army to mechanical obstruction to the circulation, when the soldier is undergoing exertion, caused by the general constriction of his neck and chest by faulty clothing and accoutrements. Their most frequent situation, as shown by Dr. Sibson, is in that portion of the aorta which is subjected to the greatest strain; and the statistics of the army corroborate the observation of Dr. Sibson as follows:—*Ascending aorta*, 37; *arch*, 38; *descending aorta*, 12; *thoracic aorta*, 7; *abdominal aorta*, 15 in 109 cases; or, ascending aorta and arch, 75; other thoracic aneurisms, 19 (MEYERS).

The naval service suffers less than the army, probably because, however violent their great exertions may be when leaning bodily over the yards in reefing, hauling at ropes, and lifting heavy weights, there is no mechanical obstruction to their circulation by tight clothing (H. Leech, *Path. Transac.*, 1865).

Hereditary transmission of aneurism has been noticed by Dr. Fuller.

Prognosis.—Aortic aneurisms very rarely recover. But life may be prolonged if the disease is recognized early and judiciously managed.

Treatment.—Local bleeding is useful when there is pain and tenderness over the aneurismal sac; and general blood-letting may be useful if the circulation is excited, and the patient be of full habit, but not on the principle advocated by, or ascribed to, Valsalva.

Of all remedies, *digitalis*, *aconite*, and *veratrin* are the most useful in tranquillizing the action of the heart. They tend to regulate the circulation, without deranging the action of the stomach. The deposition of fibrin from the blood is more prone to take place when the circulation is "slowed;" indeed, it is the principle of treatment in the cure of aneurisms by pressure. The current of blood is not stopped, but is simply rendered more slow, so as to have an amount of stagnation of blood in the sac, favouring the separation of fibrin and its coagulation. A diminution of from *ten* to *fifteen* pulsations of the heart in the minute will thus greatly tend to the filling of the sac with coagula (FULLER).

A most interesting paper was published in the *Medical Report of the Army Medical Department* for 1862, p. 472, from the pen of Joliffe Tufnell, Esq., advocating the treatment of aneurisms of the thoracic and abdominal aorta on the principle here enunciated, of "slowing" the circulation. His treatment consists of restricted diet and perfect rest, in the horizontal position, for periods varying from eight to thirteen weeks, combined with the employment of such remedies as may be necessary for special ends. The horizontal posture must be *strictly and absolutely maintained*, in a light and cheerful airy room, into which the sun shines, and from which the patient may be able to have as cheerful a view as possible out of the window.

The diet must be confined to *three* meals, served at regular intervals, and restricted to the following in kind and in amount:—

Breakfast—Two ounces of white bread and butter, with two ounces of milk or cocoa.

Dinner—Three ounces of broiled or boiled meat, with three ounces of potatoes or bread, and four ounces of water or light red wine.

Supper—Two ounces of bread and butter, and two ounces of milk or tea.

These diets should make, in the aggregate, *ten ounces of solid and eight ounces of fluid in the twenty-four hours, and no more.* The object of the special diet is to maintain life on as little food as possible, without inducing restlessness, as in some irritable constitutions; but if such restlessness should occur, a little more food may now and then be allowed. *Anodynes, aperients, narcotics, sedatives,* and *tonics* are also useful aids in the management of the case. Of anodynes, Mr. Tufnell regards *lactucarium* as the most valuable, given in the form of a pill, either by itself or combined with *humulin* and *hyoscyamus.* Mr. Tufnell's practical suggestions in detail will well repay a careful study, and merit publication in a more accessible form than in a "*blue-book.*" The patient must avoid everything which tends to increase the action of the heart. Moderate living, without the plethora of excess, but with sufficient nutrition to maintain the circulation at a *uniform* flow, is the point to aim at. Fatal results may speedily follow any marked change of diet and regimen (COPLAND).

Pain and depression will generally be subdued by the hypodermic injection of *morphia*, commencing with one-fourth of a grain. Cough is to be relieved by sedatives and expectorants. Dyspnœa may require *tracheotomy.* Dropsy may be lessened by *mercury, digitalis, squills, juniper, decoction of broom-tops.* The heart's action may be regulated by *aconite* and *digitalis.*

During the past ten years (since 1861), the following three special modes of treating aneurisms have been proposed, besides Mr. Tufnell's dietic method:—

(1.) By the introduction of a quantity of fine iron wire into the aneurismal cavity, with the intention of supplying an extensive surface over which fibrine may coagulate. Dr. Murchison and Mr. Charles H. Moore practised this method in a case of sacular aneurism of the ascending aorta projecting through the anterior wall of the left side of the chest. Twenty-six yards of wire were passed through a small canula inserted into the tumor when it was evident that the patient could not live many days. With some modifications, it was shown that the experiment might be justifiable in some cases of sacculated aneurisms only (*Med. Chir. Trans.*, vol. xlvii., p. 129, London, 1864).

(2.) By the method of RAPID *pressure treatment*, as practised by Dr. William Murray, of Newcastle-upon-Tyne, and Lecturer on Physiology in the University of Durham. On the 19th of April, 1864, Mark Wilson, twenty-six years of age, was kept under the influence of chloroform for five hours, during which time pressure was made over the abdominal aorta immediately above the tumor, and maintained by a properly constructed tourniquet. It was not, however, till the fifth hour that pulsation in the tumor had almost ceased on removing the pressure, and by the evening it had become quite pulseless. Three months afterwards the man was at work as an engine-fitter, the tumor being then scarcely perceptible. The aorta, the iliacs, and femoral arteries were quite pulseless; and so complete seemed to be the collateral circulation established, that no inconvenience had arisen. Up to 1867 the history of this man showed the cure to be complete. (See "Rapid Cure of Aneurism by Pressure," by William Murray, M.D., published by J. and A. Churchill.) The cure by this method takes place rather by the coagulation of blood in the sac from sudden cessation of the current, than to any deposit of fibrin. The patient requires to be completely under the influence of chloroform, so that he may suffer the application of a powerful pressing instrument on sensitive parts, and so as to restrain all muscular movement. All movement of the blood in the sac must be completely arrested, and maintained in a motionless state, as in the application of a ligature to the vessel for aneurism. The duration of the treatment and maintenance of pressure can only be measured by the result as to the time when pulsation ceases in the tumor. The first condition of success is, that *complete arrest* of circulation in the tumor must be *steadily* maintained. *Irregular* pressure for ten hours has been known to fail. Consolidation has occurred in twenty minutes in a case of Dr. Heath's, of Sunderland, when complete and steady pressure was maintained, having failed in a first trial with irregular pressure.

(3.) Dr. Roberts, of Manchester, and Dr. G. W. Balfour, of Edinburgh, have each collected cases, some of them under their own care, in which the treatment of aneurism of the aorta by *iodide* of *potassium* was persistently carried out with remarkably beneficial results. In twelve of Dr. Balfour's cases there was undoubted diminution in the size of the sac, while in a few there was so complete a subsidence of the tumor, and improvement in all the symptoms, as to amount to an apparently perfect cure. In all twelve of Dr. Roberts's cases, save one, striking relief of suffering followed the use of the drug; in eight, undoubted diminution in the size of the sac took place; and in a few, complete subsidence of the swelling.

The dose varied from *five, seven, ten, fifteen,* and *twenty* to *thirty grains,* three times a day; and the relief of pain does not follow till an efficient dose has been taken. It is one of the earliest symptoms of amendment. It is perhaps better to begin with twenty or thirty grain doses at once, and intermit or suspend them for a day or two, if circumstances indicate the necessity of such a step. The object is to saturate the system with the drug as speedily as possible; which is believed to owe its curative agency to the power which it possesses of increasing the coagulability of the blood. Dr. Chuckerbutty's and Dr. Wilkinson's cases tend to show this. A few weeks may be sufficient to bring about the curative result; but Dr. Balfour's experience is, that any considerable amendment can only be procured by keeping the patient for many months, perhaps twelve or more, persistently saturated with the drug. (Balfour, *Edin. Medical Journal,* July, 1868, p. 33; Chuckerbutty, in *British Med. Journal,* 19th and 26th July, 1862; Roberts, *British Med. Journal,* January, 1863.)

The strict enforcement of the recumbent position is an adjuvant of paramount necessity in the the treatment of all aneurisms.

Tracheotomy may prolong life in some cases where *stridor* exists, if the laryngeal symptoms are the source of immediate danger (W. T. GAIRDNER).

RUPTURE OF ARTERY.

LATIN EQ., *Diruptio arteriæ;* FRENCH EQ., *Rupture;* GERMAN EQ., *Zerreissung*—Syn., *Ruptur der arterie;* ITALIAN EQ., *Rottura dell' arteria.*

Definition.—*Solution of continuity of one or more, or of all the arterial coats.*

Pathology.—Rupture of arteries occurs either (1.) From disease of the coats of the artery itself, especially from such lesions as hve been already described; or (2.) From disease external to the vessel.

Rupture generally also implies force or strain; and spontaneous rupture under such exertion or straining, as accelerates the force of the blood-current, only takes place in cases where the coats of the vessel are the seat of some of the lesions or degenerations already noticed. Thus the aorta, the femoral, and other vessels sometimes burst, and the rupture may only implicate the inner and middle coat, allowing the blood to flow into, and to separate by its pressure the outer from the inner tunics. A fusiform tumor filled with blood is thus formed; *and a dissecting aneurism* is the result from this *partial rupture* of the vessel, from which recovery is impossible (ROKITANSKY.)

In the course of a few hours or days, blood escapes by destruction of the outer coats, and becomes diffused into neighbouring parts. If from the aorta, the blood may escape into the pericardium, mediastinum, or the pleura, and so cause sudden death. If in an artery of the limb, considerable destruction of muscular and other textures may necessitate amputation of the limb, if the patient does not die from collapse.

Violent pain is experienced by the patient becoming pale, cold, and pulseless, death usually taking place from profound syncope.

INFLAMMATION, ANEURISM, DILATATION, AND MORBID GROWTHS OF THE PULMONARY ARTERY.

Pathology.—Any reference to diseases of the pulmonary artery, as distinct from diseases of *arteries* and *veins*, has not been made by the College of Physicians; yet, inasmuch as this artery is an anomalous vessel, whose coats are constructed like those of the arteries in general, while they are pliable and extensible, like the texture of veins; and, the lesions are important, especially in connection with the pathology of pulmonary consumption and hæmoptysis, diseases of the pulmonary artery are worthy of notice here.

It is a short wide vessel for about two inches of its course, when it divides into its right and left branches, which accompany the bronchial tubes into the substance of the lungs, and, ramifying *without anastomoses*, at length terminate upon the walls of the air-cells and on those of the bronchia in a fine and dense capillary network, from which the radicles of the pulmonary veins arise. It conveys dark coloured venous blood from the right side of the heart to the lungs.

Deposits of blood or lymph in the pulmonary artery and its branches occur as a result of inflammation of veins and venous embolism, and will be considered under diseases of the veins.

Acute inflammation is rare; but *atheroma,* as a result of chronic inflammation, is not uncommon in cases of deficiency of the mitral valve, with consecutive hypertrophy of the right ventricle; the atheromatous products leading to hæmorrhagic infarction in the lungs (DITTRICH).

Acute inflammation of the pulmonary artery has been described by Dr. Norman Chevers as occurring under the following circumstances:—

(1.) As a consequence of inflammation of the veins.

(2.) In cases of Bright's disease, and especially in persons habitually intemperate.

(3.) In rheumatic patients, as a result of exposure to cold.

(4.) In connection with certain forms of pneumonia.

Aneurisms of the pulmonary artery are described as exceedingly rare, and never attain any considerable size. Skoda reports a case where the aneurism was as large as a goose's egg, associated with cyanosis and dropsy (NIEMEYER).

Aneurisms in branches of the pulmonary artery occupying phthisical cavities in the lungs are by no means uncommon; and it seems probable, though comparatively few cases have been recorded, that aneurismal dilatations of branches of the pulmonary artery within vomicæ may be more common than is generally supposed, and would be discovered if carefully sought for, particularly in cases of fatal hæmoptysis (CHRISTOPHER HEATH). In the *Lancet* for 1841, an aneurism of this kind is described by Mr. Fearn, of Derby, in a man forty-one years of age, proving fatal after several attacks of hæmoptysis. Similar cases are also recorded by Dr. Cotton, in *Med. Times and Gazette* for Jan. 13, and Oct. 20, 1866, and *British Med. Journal*, Oct. 24, 1868; by Dr. Quain, in *Path. Soc. Transactions*, vols. xvii. and xviii.; and Dr. Moxon, in vol. xviii., p. 55, the third case he has met with, and reported in the *post-*

mortem Records of Guy's Hospital. In all his cases there was a history of hæmoptysis, and one of the patients died suddenly by the rupture of the aneurism.

Dr. Vald. Rasmussen, of Copenhagen, also reports, in his excellent paper on "Hæmoptysis," translated by Dr. Daniel Moore, of Dublin (*Edin. Med. Journal*, 1868, vol. i., p. 389), that hæmorrhages from a cavity are due to ruptures of small sac-like aneurisms, developed on branches of the pulmonary artery, running in the walls of cavities. The size of these aneurisms (in four cases) varied from that of a walnut to that of a pea, and under, formed by the dilatation of a vessel in contact with the inner wall of the cavity, the part of the vascular wall touching the cavity at the point of contact being dilated, while the remainder lies imbedded in the condensed vascular wall. The form is generally sacculated, with a tolerably uniform transition, between the walls of the aneurism and those of the vessel, without any proper neck. The surface of the aneurism is smooth, and in its cavity most frequently only freshly coagulated blood is found, and only in one instance fine decolorized adherent coagula. The walls are of various thickness, often two or three times thicker than the vascular wall from which they proceed; and when the vessels are small from which they grow, the aneurisms form only a slight dome-like dilatation. When the size is large, the walls are thin and particularly attenuated up to the point where rupture has taken place. Degeneration of the walls of the aneurism has also been met with, as separate, or confluent, and yellow sharply defined points. The rupture takes place always at the most prominent point of the sac; and there is usually found an irregular fissure-like rent, rarely exceeding in width two or three millimètres; most frequently it is only large enough to allow the knob of an ordinary probe to pass. In the opening, loose dark coagula adhere, more rarely firm and somewhat decolorized. The edges are attenuated and yellow, showing distinct fatty degeneration. The number of the aneurisms vary. In general one only is met with. In one case two were found close to each other. In another case there were as many as four—two and two, close to each other on the same branch. The vessels on which these aneurisms had formed were on an average from one to three millimètres in width.

Rasmussen is of opinion that the development of these aneurisms on the pulmonary artery stands in a definite causal relation to the formation of the cavity in the lung—want of support being one of the chief elements favouring the growth of the aneurism, combined with increased intra-vascular pressure, which during a fit of coughing is very considerable, and still more increased by condensed pulmonary tissue and obliteration of branches beyond the aneurism.

An important observation made by Deichler would indicate one form of pulmonary phthisis as originating in disease of the pulmonary artery. He found when a section was made through the centre of one of the tubercle-masses that it inclosed a little stem, knuckle, or portion of a minute pulmonary artery, about one-twentieth of a line in size. This knuckle or loop of artery was surrounded by tubercle. The walls soften by the new formation of cells; and the more the cells grow, the larger the tubercle-mass becomes, till it is finally lost in the larger knots. It is not improbable that the tubercle-knots are formed owing to a diseased state of the membranes of the stems of the blood-vessels. Evidence of such disease may be found in cases of *ectasis* or aneurismal distension of the walls of the pulmonary capillaries, as seen in the air-cells. Buhl and Zenker have described such cases (Virchow's *Archiv.*, 1862, p. 183).

Bearing upon this point, and upon aneurism of the pulmonary artery, I lately (1861) dissected a soldier who died suddenly by hæmorrhage from the lungs. On opening into one of the tubercle-cavities, it was found filled with coagulated blood; and projecting from a spot on the wall of this pulmonary cavity was a round tumor about the size of a walnut. This tumor had ruptured, and the rupture held a coagulum of blood. The tumor was found to be an aneurism of the pulmonary artery; and several other tumors of a similar nature, but of variable and much smaller sizes, existed in other cavities in the same lung, projecting from the pulmonary artery. They were proved to be continuous with this vessel—(1.) By the injection of spirits into them, and their distension thereby through the pulmonary artery as it left the heart; and (2.) By microscopic examination, which showed a delicate epithelial lining to these tumors, continuous and similar to that in the artery. The preparation is preserved in the museum of the Army Medical School at Netley.

Diffused dilatation of the pulmonary artery occurs with great frequency in cases which cause hypertrophy and dilatation of the right heart. An unusually large pulmonary artery which had four valves is described by Dr. Theophilus Thomson, in *Med.-Chir. Transactions,* vol. xxv., p. 247, London, 1842; and other morbid conditions, such as *ulceration* from pressure of aortic aneurism, may also be seen.

(b.) *Diseases of the Veins.*

PHLEBITIS.

Latin Eq., *Phlebitis;* French Eq., *Phlebite;* German Eq., *Phlebitis*—Syn., *Venenentzündung;* Italian Eq., *Flebitide.*

Definition.—*Inflammation of the texture of a vein, leading to changes in its texture and to local coagulation of blood within the inflamed part of the vessel (thrombosis), and often also to the subsequent phenomena of venous, pulmonary, and hepatic embolism.*

Pathology.—The varieties of *phlebitis* are described as (a) *Adhesive;* (b) *Suppurative.* The first set of phenomena to be considered are those which accompany the early stage of inflammation of the coats of a vein, because they mostly concern the physician, in connection with the important and dangerous phenomena of *embolism.* For other aspects of inflammations of the veins in the varieties of phlebitis, the student is referred to his text-books on surgery.

So long as pus-corpuscles, as such, were looked upon as the really noxious material which poisoned the blood in cases of malignant phlebitis, it was supposed that the tissue of a vein being inflamed, pus would be secreted from its inner wall, just as from a serous membrane. John Hunter has the credit of having suggested this; but he did so merely *as a query;* and subsequent writers adopted the suggestion without further examination and without evidence, (see Arnott "On the Effects of Inflammation on Veins," in vol. xv. of *Med.-Chir. Trans.*) Veins are exceedingly slow to inflame. When they do, the inflammation-changes begin in the connective-tissue, towards the outer parts of the vessel, and this even when irritant bodies are introduced into the cavity of the vein itself, (see Lee's "Essay," in vol. xxv. of *Med.-Chir. Trans.*) Mr. Lee introduced cotton-wool into a portion of vein emptied of blood. The lining membrane remained unchanged; and the lesions commenced outside the vessel. There the pus formed, and thence the inflammation spread by the connective tissue, and simply by continuity.

The generation of secondary multiple abscesses is attended by quite a different process. It was first shown by Cruveilhier that in the (so-called) inflammation of veins a clot of fibrin forms, and is always present, independent of any lesion in the vascular wall; and so he passed to the extreme belief that all inflammation essentially consisted in coagulation of blood in capillary vessels. The first great fact has now been quite substantiated—namely, that long before any visible effects of inflammation occur in the lining membrane of a vein, a clot of fibrin is formed at the part, and in this clot fluid comes to be produced, in all external appearances resembling pus. It must also be remembered that blood, when arrested in the vessels or extravasated out of them, coagulates at the ordinary temperature of the body—98° Fahr. (*Phil. Trans.*, vol. lxxxvi.)—a most valuable event in the cure of aneurisms.

From this observation as a starting-point, Virchow has developed his very beautiful explanations of the various phenomena connected with phlebitis. Inflammation of veins as the cause of secondary inflammatory phenomena and of multiple abscesses is to be rejected; but "coagulation of the blood in the living vessels"—the formation of a clot or "thrombus" (*thrombosis*)—are the phenomena which attend the formation of multiple abscesses, as in the phlebitis of pyæmia. The impulsion or projection onwards of a coagulum-clot, or thrombus, or substance detached from the walls or valves of the vascular system, and its subsequent arrest in the course of the circulation, is described under the name of *embolism.*

The coagula may travel in particles or larger masses from the veins to the heart, or from the heart to the arterial peripheric vessels (see under "Occlusion of Arteries," p. 639, *ante*).

Thus, on the one hand, *deposition* of morbid substances in various parts of the body distant from the heart is accounted for; and, on the other hand, results are explained on simple mechanical principles which hitherto have been obscure. Formerly, many of the cases thus capable of explanation would have been recorded as cases of "sudden death," or as "sudden retrocession of gout or rheumatism," or of "gout in the stomach," or "palsy of the heart."

All the cases, fully recorded, which illustrate the phenomena of pyæmia from phlebitis that I have ever examined have shown that the affection essentially begins by a real coagulation of the blood at *some* *definite fixed point;* and this is the most obscure part, and the most difficult to discover, in the history of all the cases. But where this beginning is traceable, the history is exceedingly significant, as pointing to some sources of *local irritation,* which, by simple disturbance of the flow of blood, determines in some way its coagulation in the living vessels.

The beautiful experiments of Professor Lister throw much light upon this subject (" Croonian Lectures," *Lancet,* Aug. 8, 1863). Any occurrence which sufficiently interferes with, lowers or disturbs the vitality of the structures enclosing blood, affects its fluidity, and tends to permit its spontaneous coagulation *in situ.* Some of the cases mentioned by M. Ribes, in 1825, occurring so far back as 1799, also illustrate this point by morbid anatomy. In one case chilblains was the starting-point. Clots formed in the veins, and proceeded upwards even to the superior *vena cava,* into the right auricle and ventricle. In one of the most striking cases of this description recently recorded, " a venous clot of fibrin twenty inches long was found in the right auricle and ventricle (Druitt, *Med. Times and Gazette,* July 19, 1862). It showed such marks on its surface as clearly demonstrated its formation in a vein; and other circumstances pointed to the chief vein of the thigh as the site of the primary formation of this *clot* or *thrombus.* Œdema of the limb prevailed, and the disappearance of the œdema was associated with those sudden cardiac and other symptoms which indicated the passage upwards of this *coagulum* or *thrombus* to the heart, where it was found coiled up in the right auricle and ventricle. In cases of fracture of bone, of amputations, of enlarged glands, of ulcer on the foot (imperfectly healed by scabbing), of open wounds, gunshot passing near vessels, and ulcers, we have great and many sources of irritation, leading to inflammation of a vein at a spot, and the accompanying *thrombosis.* The contiguity of these must be to more or less large veins, or to smaller vessels in or about these sources of irritation and disturbance.

But *pyœmia* is not always the result of phlebitis. " The immediate cause of pyæmia in any given case is, that some diseased part (which need not be an external wound) so affects the blood circulating through it, that this blood afterwards excites destructive suppuration in parts to which the circulation carries it—namely, commonly first in the lungs, or (in certain cases) liver and lungs, and later generally about the body" (Simon, *Public Health Report* for 1863, p. 60). In a case of which Mr. Simon was cognizant, and which is described by Mr. Bowman, fatal pyæmia in a young gentleman was apparently produced by an ulcerated state of the mitral valve of the heart. In another case Mr. Simon records fatal pyæmia produced by the penetration of pus from a small mesenteric abscess into the thoracic duct; and in the thirteenth volume of the *Pathological Society's Transactions* Dr. Bristow records several instructive cases, in which pyæmia complicated, *ab initio,* cases of *idiopathic necrosis unattended by external wound.* In no case, therefore, of swelled legs, enlarged glands, subacute inflammation, or hardening over the course of lymphatics or superficial veins, should the possibility of the mortal accident of *embolism* to the right side of the heart be overlooked; or the possible supervention of pyæmia.

The next set of phenomena characteristic of phlebitis with the formation of multiple abscesses, are those connected with the softening, disintegration, and breaking up of the thrombi or clots. Virchow was the first to demonstrate the results that ensue. He showed (1847) the embolic characters of certain products previously thought to be inflammatory in their origin (*e. g.*, white fibrine-like masses in the spleen, &c.); and he arrived at the following conclusions:—(1.) The occurrence of fibrinous plugs or clots in the pulmonary artery long before death is always secondary, where obstruction is independent of pneumonia, or other changes in the parenchyma. They are apt to arise in any part of the venous vascular system anterior to the lungs in the course of the circulation—*e. g.*, in the veins of the limbs, as in the case described by Druitt, already noticed; in the pelvic veins, as after the operation of ligature for internal piles, whence the clots are carried by the current of the blood to the right side of the heart, and thence into the pulmonary artery, and so to the lungs. Experiments on animals also supported his views. He introduced the pith of the elder tree, as well as animal substances, into the veins, and so produced violent pneumonias, commencing with inflammatory hyperæmia. These localized pneumonias extending, led to rapid deposition in the air-cells of fibrinous exudations, which became purulent, or the portion of lung gangrenous. As these changes advanced in the lung, pleurisy very soon was developed at the periphery—at first producing fibrinous coagulable exudation slowly over the affected portion of the lung; but rapidly, as it progressed towards the other side of the chest, inducing watery hæmorrhagic exudation into both serous cavities. The pleura then became gangrenous, and finally gave way to pneumothorax. Such severe lesions may be completed in so short a time as five days. Previously, however, to these important demonstrations of Virchow, there had appeared the observations of Sir James Paget on the obstructions of the branches of the *pulmonary artery*, and the sudden mode of death to which they gave rise (*Med.-Chir. Trans.* for 1844). Cruveilhier, Baron, and Dubini had also recorded similar cases; but the importance of Virchow's observations consisted in demonstrating the transportation of clots or plugs of fibrin or of blood from different parts of the venous vessels to the heart. Such plugs are known also to be sometimes arrested in the liver, giving rise to so-called metastatic abscess; and while those plugs which find their way to the lungs have their origin in any part of the venous periphery, those which find their way to the liver originate either in the *portal venous system* or in those veins round the *rectum*, *prostate*, *vagina*, or *uterus*, which (communicating alike with the *systemic veins* and with the *inferior mesenteric veins* of the *portal system*) may induce multiple centres of inflammation, and abscesses in the liver as well as in the lung (*Med. Times*, January, 1862). Simultaneously with Virchow's, or a little after his observations, we have the observations of the late Dr. Kirkes, on "The Detachment of Fibrinous Deposits from the Walls of the Heart" (*Med.-Chir. Trans.*, vol. xxxv., for 1852). Two most instructive cases of this kind have been recently related by Dr. Goodfellow, in which extensive arterial obstruction, gangrene of the lower limbs, and death, followed the separation of cardiac vegetations, (*Med.-Chir. Trans.*, vol. xlv.)

Thus it was gradually proved, that as clots occur in the veins, so they also may occur in or find their way into the heart's cavities; and next in the history of this interesting subject the connection is established between *peripheral clots* and *cardiac clots,* and how far the softening of these clots or *thrombi* may give rise to the lesions indiscriminately described as *pyæmia* or *phlebitis* by the phenomena of *embolism.*

Such, pathologically, are some of the causes which may determine the formation of clots either in large or in small vessels. But, after Cruveilhier's observations, the next link in the chain of evidence regarding the nature of *metastatical dyscrasiæ* was established by an arduous worker (then in the Army Medical Department, and since a distinguished Professor in the Royal College of Surgeons of England)—namely, Mr. Gulliver, formerly surgeon of the Guards. He showed that the puriform mass in the interior of clots does not originate in the wall of the vessel or clot, but is produced by transformation of the central layers of the clot—a transformation which may be imitated, as he did, by a chemical process. Sir James M'Grigor communicated Mr Gulliver's observations to the *Medico-Chirurgical Transactions.* Mr. Gulliver examined the clots microscopically, and found that the fluid was not pus; and to show that his observation was intimately connected with the observation of Cruveilhier, I found (by mere accident) that a remark on this point, in Mr. Gulliver's handwriting, exists on one of Cruveilhier's drawings contained in the Library of the Army Medical Department now at Netley.

This is the point from which Virchow starts in his interesting account of this subject—namely, the character of the contents of these clots—a puriform, but not a purulent substance (as Gulliver first showed), composed of granules chiefly. The question, then, immediately suggests itself, "What becomes of them?"

Secondary disturbances—not so much by the softened mass as by the detachment of larger pieces, and just so large as to be arrested in vessels more or less remote from the seat of the original clot—are known to occur: and on the advance of clots and their debris from small into large vessels, bits break off and flow on into the stream of blood.

Clots in peripheral veins, however small, are thus the sources of great danger. As a rule, they lead to secondary and multiple deposits and abscesses in the lungs; and it is chiefly differences in the size of the capillary vessels which determine their ultimate locality, where they act as any foreign body would. The debris of clots, and large cell-elements from clots, in the mesenteric veins, and from ulcers of intestines, passing through the liver capillaries and proceeding to the lungs, where they are arrested, illustrate this. The lungs have the smallest capillaries of all. They average from $\frac{3}{10000}$ths to $\frac{50}{10000}$ths of a line (scarcely sufficient to let pass a white cell of blood or of pus, which on an average measures $\frac{44}{10000}$ths of a line), whereas the liver capillaries have a much larger range—namely, from $\frac{20}{10000}$ths to $\frac{100}{10000}$ths of a line.

The most common cases of *thrombosis* in veins are to be seen in bedridden dropsical persons, whose veins in connection with the buttocks, such as those of the thighs and genitals, contain *ante mortem* clots, probably acknowledging the source of the irritation giving rise to them as due to the pressure of the weight of the body on the radicles of these veins, and the gravitation of the blood against them (MOXON). Thus, the coagula found in the veins are the products of local stasis, often caused by roughness of their inner surface, by alteration of structure, and relaxation of elementary parts. When the coagulum adheres only to one wall, with the effect of narrowing the passage, such a thrombus is never of an embolic nature. It is a clot, *thrombus*, plug, or coagulum of fibrin formed or forming there as a result of local causes of irritation; and *venous embolism* occurs,—(1.) When coagula arrive in the right side of the heart. Here they are attended with symptoms of exhaustion, pulse small and intermitting, followed by collapse and powerlessness of muscle. The patient, although inhaling deeply, seems to suffer from apnœa; the veins become highly turgid; and sudden death occurs if large concretions separate and suddenly obstruct the pulmonary artery; as in the case recorded by Druitt, and noticed at p. 653. The symptoms were,—hurried and anxious breathing; pulse rapid and scarcely perceptible; features intensely pale, bluish, and distressed; the whole surface of the body cold, but drenched in perspiration; no pain, but great agitation and feebleness; the air entered the air-cells freely, but the beating of the heart was a confused and feeble "*wobble*"—its rhythm and force were gone. The intellect was clear.

The *Phenomena of Pulmonary Arterial Embolism* are of two kinds; and the diagnosis of embolism may be summed up as follows:—

(A.) *Parenchymatous.*—(*a.*) Collapse of lung; (*b.*) Peripheral pleuritis of lung; (*c.*) Hæmorrhagic effusion; (*d.*) Capillary bronchitis, with cough and expectoration if the embolism is capillary (see vol. i., p. 73).

(B.) *Functional.*—(*e.*) A craving for air (anxietas): although a deep breath may be drawn without pain, yet every movement of the body tends to increase the anxietas; (*f.*) Lowering of temperature; (*g.*) Systolic murmur; (*h.*) Increased impulse of the right side of the heart; (*i.*) Irregularity of rhythm; (*k.*) Undulation of veins in the neck; (*l.*) Cyanosis, vertigo, albuminuria, œdema of limbs. These phenomena are seldom all present, death being too sudden.

Phenomena of Pigmental Embolism.—A peculiar form of embolism is associated with accumulations of pigment in the blood. It is primarily developed in the spleen, whence the thrombi are conveyed to the *vena portæ*, gradually increasing in circumference. Malarious fevers establish such pigments in the blood (PLANER, of Vienna). In such cases sanguineous extravasations, in great numbers, are found in the brain, and abscesses in the liver; laceration of hyperæmic capillaries is also a result—*e. g.*, Malpighian glomeruli of the kidneys.

PHLEGMASIA DOLENS.

LATIN EQ., *Phlegmasia dolens;* FRENCH EQ., *Phlegmasia alba dolens;* GERMAN EQ., *Phlegmasia dolens;* ITALIAN EQ., *Flemmasia dolente.*

Definition.—*Obstruction of the veins and lymphatics, causing a painful, non-œdematous, but brawny swelling of one or of both lower extremities, and attended with great prostration.*

Pathology.—The disease has also been named "milk-leg" or "white-swelling." It is most common after parturition, especially when much blood has been lost in the process of childbirth; and it not unfrequently attends malignant uterine disease.

Any great drain from the system, such as these states imply, is apt to be followed by no less rapid absorption; and the co-existence of foulness of parts from malignant disease, or decomposition of textures in connection with the lesion of the uterus after childbirth, or with bits of retained placenta. A rapid absorption of poisonous or acrimonious fluid is apt to occur locally, leading to instantaneous coagulation of blood, or to inflammatory changes in the coats of the uterine veins. The external iliac and femoral veins are also most commonly affected. In some way, not yet fully known, a severe *irritation* is next set up amongst the nerves, muscles, and lymphatics of the upper part of the thigh and leg, the lining membrane of the veins, and the areolar tissue of the limb. The results are a tense elastic swelling, till the leg may be twice its usual size, with pain and complete loss of power of motion, painful lymphatics, and obstructed veins.

The most intelligible explanation of these cases, and the one which the facts disclosed *post-mortem* tend most to support, is, that inflammation of the iliac and femoral veins is the proximate cause of the disease, and that in puerperal women the inflammation commences in the uterine branches of the hypogastric veins. Dr. Robert Lee's experience is to this effect; and (in the *Path. Soc. Transactions*, vol. ix.) Dr. Graily Hewitt gives an account of a dissection of a case in which the obstruction was in the left common iliac, having been produced in consequence of some morbid condition of the veins leading from the uterus.

Symptoms of *phlegmasia dolens* may set in immediately after labour, or at any time during the next five or six weeks. They are expressed by fever, headache, thirst, nausea, and pain, especially in the lower abdominal and pelvic regions, accompanied with great prostration. A chill or a rigor may usher in the disease, with or without pain or swelling of joints, and within twenty-four or thirty-four hours the swelling of one or both lower extremities may be apparent, commencing about the foot or lower part of the leg, from which it extends upwards. The acute stage lasts about fourteen to twenty-one days; and after recovery, if life is spared, many deep veins remain obliterated, compensated for by varicosity and enlargement of superficial ones; so that the limbs are useless for many weeks or months, and too often never recover their wonted power and shape.

Prognosis.—The disease has usually a favourable termination; and as the general health improves, the swelling and tenderness diminish.

Great care must be taken before and during subsequent pregnancies that the general health is maintained as well as possible.

Treatment.—The general health in such cases is generally feeble, and prostration exists; hence, leeches and blood-letting, as recommended by Drs. Davie and Lee, are usually inadmissible. The treatment followed and recommended by the late Dr. Tanner is the most rational, namely, sedatives and alkaline fomentations, perfect rest, simple diet, and opiates to relieve pain. "The fluid in which the fomentation flannels are to be wrung out is made by adding one pound of *bicarbonate of soda* and one ounce of *extract of poppies* to one gallon of boiling water. The flannels ought to be changed every thirty minutes, and applied over the whole limb, and over the groin and lower part of the abdomen wherever there is tenderness. The heat and steam from these are to be retained by means of impermeable cloths." Wine, brandy, milk, raw eggs, animal food, ammonia, and bark are sure to be required; and after the acute symptoms have subsided, there is no remedy so beneficial as efficient bandaging, together with the preparations of iron, quinine and tonic vegetable bitters, like *calumba,* aided by change of residence (TANNER).

CHAPTER XVIII.

DISEASES OF DUCTLESS GLANDS.

SECTION I.—DISEASES OF THE THYROID GLAND.

GOITRE.

LATIN EQ., *Bronchocele;* FRENCH EQ., *Goître;* GERMAN EQ., *Kropf*—Syn., *Struma;* ITALIAN EQ., *Gozzo.*

Definition.—*A specific affection of the thyroid gland, induced by the persistent use of water which has percolated through magnesian limestone rocks or strata, and containing the soluble salts of lime in solution.*

Pathology and Morbid Anatomy.—The characters of the swelling of the thyroid gland, associated with this morbid state, appear to be different at different stages of its existence. At first the tumor is soft, but it gradually acquires a firm and even a cartilaginous consistence. In the soft condition the cell-elements of the gland seem to secrete a fluid of a thick, ropy, viscid, gelatinous appearance; but when the consistence increases, the hypertrophy of the cell-elements is generally more obvious than the fluid secretion, its blood-vessels seem increased in size and number, and ultimately cysts become developed, in which the glairy fluid abounds. In the more advanced cases, osseous, or at least calcareous deposits occur, and occasionally the whole organ is transformed into an osseous-like capsule, filled with matter of various kinds, which has been likened to jelly, suet, and honey. Sometimes the gland preserves its original lobulated form, its relative proportions being maintained; at other times there is no distinction of parts or lobules. The right lobe is more often enlarged than the left (ALIBERT, RICKWOOD, GREENHOW). Solid aggregations of calcareous particles have been found in the goitrous gland (CEELY). The tumor, originally of a simple nature, is liable to enlargement of its arteries, to expansion of its cells, or the formation of cysts on its surface and in its interior, or to inflammation and ulceration of its component tissues. Such ulceration is apt to assume a malignant scirrhus-like appearance.

The prevalence of the disease is limited to certain districts of peculiar geological formation; and wherever it prevails, popular opinion has always regarded the water used for drinking as being in some way connected with its cause. Alike in England and in Oude, where goitre prevails, the people are convinced that water in some way is the cause of goitre. It was at one time believed that *snow-water* from the summits of lofty mountains contained the poisonous material which established *goitre* among the inhabitants of the valleys. The people of almost all the valleys of Switzerland drink snow-water, or the water of the glaciers; but *goitre* abounds in some of the valleys only; and in Greenland and Lapland, where snow-water is commonly used, goitre is unknown. Again, the disease exists in countries in which snow is never seen, as in Sumatra; and in places where snow never lies sufficiently long to be used as a drink, as in Derbyshire. It prevails in places where *pump-water*, rather than *surface-water*, is used. Still, the evidence is very conclusive which points to something specific in the quality of the water as tending to the development of *goitre*. At Nottingham, in England, where this disease prevails, the common people refer it to the *hardness* of the water—*i. e.*, to its impregnation with calcareous salts, *sulphate* or *carbonate of lime;* and it has even been affirmed that the presence of magnesian limestone always implies the endemic co-existence of the disease (INGLIS). In Captain Franklin's expedition to the polar sea, *goitre* was found to be very prevalent at Edmonton, where the soil is calcareous, and contains numerous fragments of magnesian *limestone*. The disorder attacks those only who drink from the *water* of the river (the Saskatchawan). In its worst form it is confined almost entirely to the half-bred women and children who make use of the river-water. The men, who are often from home journeying through the valleys, drink the melted snow, and are less affected; and, should incipient symptoms of the disease come on in winter, when they live at home and use the water of the river, the annual summer voyage to the sea-coast generally effects a cure. The natives who use *snow-water* only, and drink from the rivulets which flow through the plains in summer, are exempt from the disease; but the residence of a single year at Edmonton, if the river-water of the Saskatchawan is used, is sufficient to render a whole family the subjects of goitre (RICHARDSON). The disease has been known to occur and to affect a family in a very short time, who, being free from the disease while using the *surface-water*, had a well dug, and obtained their water by tapping a limestone rock; and after drinking from this well for a short time, the disease appeared among them. There are some waters in a goitrous district in Switzerland, issuing from the hollows of certain rocks, and trickling along crevices in the mountains, the drinking of which will produce *goitre*, or augment goitrous swellings, in eight or ten days, while the inhabitants who avoid these waters are free from the disease (BALLY, WATSON). Dr. Coindet, of Geneva, states that the use of the hard pump-water in the lower streets of that town brings on goitre very speedily; and at Cluses, on the Arne, numerous cretins and goitrous persons are seen in the streets;

while lofty cliffs of mountain limestone tower over the town; and through the crevices of these cliffs copious streams of water flow. In Yorkshire, Derbyshire, Nottingham, Hants, and Sussex, in England, where the disease prevails, there is a ridge of magnesian limestone running from north to south through the centre of the district. All along that line goitre prevails to its greatest extent; and, diverging to either side, the disease is found to diminish (Inglis, *Treatise on English Bronchocele*). The disease has been found to prevail in one great section of the province of Kemaon, in India, south of the Himalayan mountains, and to be almost entirely absent from another section of that district. Both of them agree in their external aspect, altitude, and climatology, but differ so remarkably in their geological formation that an examination of the rocks of the district, into the very villages where the disease abounded, or did not abound, enabled one to predict whether the inhabitants were affected with goitre or not. No instance occurred, in a district extending over 1,000 square miles, in which goitre prevailed to any extent where the villages were not situated on or close to limestone rocks (M'Clelland). Dr. M'Clelland visited 126 villages scattered promiscuously over an area of upwards of 1,000 miles. The following are the results he obtained:—

1. Five of these villages were built upon hornblende and mica slate, or on siliceous sandstone, or on green sandstone. They contained 290 inhabitants, not one of whom was a *cretin*, or was affected with *goitre*.

2. Seventy-one of the villages in the same district were built upon clay-slate. These contained 3,957 inhabitants; and among them there were twenty-two persons with goitre, or *one* in two hundred of the population. There was not a single cretin.

3. Thirty-five of the villages, having a population of 1,160, were built upon Alpine limestone; and in them 390 persons, or more than *one-third* of the inhabitants, had goitre, while thirty-four of them were cretins, or about one person in every thirty-five.

Lastly, goitre is extremely frequent at Secrora, near Lucknow, and in all that district of Oude which stretches towards Nepaul and the Goruckpore district beyond the Gogra. In the jungles of the Teraie, at the base of the Nepaul hills, the disease is very frequent; and in Nepaul itself, among the inhabitants of the *Cis* and *Trans*-Himalayan regions, it is constantly met with (Bramley, Greenhow; *Indian Annals* for 1857 and 1859). All along the line of Teraie, on to Goruckpore, goitre is so prevalent that one in ten persons is afflicted with the horrible disfigurement. The kingdom of Oude is geologically made up of the diluvial detritus of the Himalayan chain, which abounds in limestone, and the soil of the district contains abundance of lime, which is taken up by the waters that percolate through it from the rivers and from the rains and floods. The lime thus taken up and held in solution with carbonic acid gas is frequently found deposited round nuclear fragments of flints or stones, and is then known by the name of "kunker;" so that wherever "kunker" abounds, there soluble salts of lime, silica, alumina, and sometimes magnesia and protoxide of iron will be found (Sleeman, O'Shaughnessy, Greenhow).

There is a remarkable circumstance connected with the prevalence of goitre in Oude, which is, that it affects animal as well as man. At Hissawpore, a village about twelve miles distant from Secrora, on the Surjoo river, dogs and other animals are affected with it (GREENHOW). Mr. Bramley noticed the same curious fact in Nepaul; for "on one occasion a goat brought forth a kid with a goitre as large as its head. Puppies of a month old, bred from English dogs, are very frequently affected by it, as are also lambs."

A curious outbreak of *acute goitre* occurred in 1860 in the garrison of Briançon (Hautes Alpes). The mean strength of the garrison during the year was forty-eight officers and 954 men; and from this force fifty-three cases of acute goitre (fifty-one soldiers and two *enfants de troupe*) were admitted in the year, and in the first three months of 1861. One case occurred in as short a time as *eight* days after arrival at Briançon, and one after *sixteen* months' stay; but the majority (thirty-nine) occurred after from *eight* to *eleven* months' sojourn. The rapidity of growth of the gland was remarkable. In some very predisposed subjects eight days sufficed to show a large increase; and the form of the tumor was most frequently bilateral. The sanguino-lymphatic temperaments and robust constitutions were those most attacked. There was no obvious hereditary tendency; but several of the men came from departments where goitre is more or less prevalent. The men who came from maritime places, and who were placed at Briançon under quite unusual conditions (4,285 feet above the sea-level), suffered most. M. Collin, who records the outbreak, does not say a word about the composition of the water; but the position of Briançon renders it highly probable that the water of the place is charged with lime and magnesian salts (Parkes, in *Army Med. Department Sanitary Report* for 1860, p. 385).

The disease is known to prevail at the base of lofty mountains in many parts of the globe. It is endemic at the foot of the Alps, where it is frequently associated with *cretinism*—a sort of idiocy, associated with atrophy of the brain and deformity of the body. It is also endemic at the foot of the Apennines. It is common in Derbyshire, where it is called the "Derbyshire Neck." It is met with in some flat situations in Norfolk; and in one village about five miles from Cambridge it is extremely common (WATSON). In South America goitre is met with both in the upper and in the lower course of the Magdalen river, and in the flat high country of Bogota, 6,000 feet above the level of the stream (HUMBOLDT). It is also common at the base of the South American Andes. In North America many cases occur in the vicinity of the Blue Ridge, in Virginia. It is prevalent in the mountainous regions of Pennsylvania, New York, New Hampshire, and Vermont (DUNGLISON). In India it prevails in Oude, and along the line of the Himalayan range. It seems to be more common in females than in males, and is rarely seen before the age of puberty; but in districts where the disease abounds, it is on record that children are sometimes born goitrous of goitrous parents (GODELLE, WATSON). The evidence of hereditary transmission, in the strict sense of the term, appears to be doubtful; but predisposition may exist in some, rather than in others, to the development of the disease.

It may generally, therefore, be concluded, from the cumulative nature of the evidence, that a poison exists in association with lime and magnesia in geological formation, whose action induces undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for blood-vessels (KÖLLIKER, VIRCHOW); and it is fair to connect the unusual quantity of lime taken into the system with such premature and abnormal ossifications. Wherever chemical examination of the water used by the inhabitants of the different places where goitre and cretinism prevail has been made (as it has been especially in India), it has always been found to contain a large quantity of carbonate of lime; whereas the water derived from the clay-slate rock, and which was drunk by the inhabitants who did not suffer from *goitre*, contained none. Such observations as those described, and especially those of M'Lelland and Greenhow, show that neither the atmosphere, the elevation above the sea-level, the physical aspect of the country, nor locality, have anything to do with the production of goitre; but they prove almost to demonstration that the affection is due to some specific action of the drinking-water which flows from rocks of a particular geological formation named magnesian limestone. The circumstances under which these affections were found by M'Lelland to exist in the low burning plains of Bengal formed a striking corroboration to his observations in the hills of Kemaon. Goitre and cretinism are very prevalent in different parts of the district of Goruckpore. The soil of the district is of two sorts. One, to which the natives give the name of "bhat," characterizes the lands bordering the river Gunduk and its branches. This soil is remarkable for the large proportion of calcareous matter which it contains. One specimen, on analysis, yielded upwards of 25 *per cent.* of carbonate of lime. Goitre and cretinism are very prevalent in the villages built upon this soil. In some of them 10 *per cent.* of the population are affected; and of the children in the villages where goitre prevails 10 *per cent.* are *cretins.* The dogs and cats of these villages are also often affected with the disease. On the other hand, the lands on the banks of the Gogra consist of a soil to which the natives give the name of "bangar." It is much less retentive of moisture than the "bhat" land, and requires irrigation for the production of winter crops. This "bangar" soil is very siliceous, and contains scarcely any lime. Goitre and cretinism are unknown in the villages built upon this soil (*Brit. and For. Med.-Chir. Review,* Jan., 1861).

The natives of Oude ascribe their goitres to drinking certain waters; and they adduce cases to prove that by partaking of the water of certain wells they get the disease, and by deserting those wells they sometimes become cured of it (GREENHOW). Thus almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of goitre. Remarkable instances are known wherein the exchange of well for rain-water, for drinking purposes, has been followed by the best effects, and even by the disappearance of goitrous tumors. Dr. Greenhow states that in Oude, where the water of wells believed to be injurious, in consequence of their excessive impregnation with lime, has been given up, and other water used instead for drinking, great benefit has been felt, and goitres have decreased in size, even though the subjects of them have continued living in the same village as before. He was assured also, by several of his patients in Oude, that certain wells were known by them to be deleterious, and that the natives of the villages avoided them accordingly, having learned to do so from experience. He tested the water of the wells most shunned by the natives, and found it to contain a great excess of lime; and he concludes, from his own investigations in connection with others, that the use of drinking-water containing lime is the main cause of goitre. How it acts on the system is as yet unknown.

Treatment.—The indications are—(1.) To remove from the district where the disease prevails; (2.) Improvement of the water used as drink; (3.) Elimination of the goitrous poison, or change of constitution. As with the poison of *mercury* and of *lead*, so with goitre, *iodide of potassium* has a wonderful effect in subduing the swelling, and probably in eliminating the poison which produces goitre, whatever that may be. *Iodine* has thus acquired the reputation of being almost a specific against goitre (Coindet, Straub, Gairdner, Manson, Lugol). In robust subjects it has been advised that the administration of *iodine* should be preceded by general blood-letting, on the principle that the abstraction of blood favours absorption; but this should not be done unless the *iodine* does not seem to be producing any effect; and whatever may be the preparation of *iodine* employed, it must be persevered in for a length of time, and the dose gradually increased (Dunglison). The best form of administration is that in which the *iodine* is mixed with and dissolved by *iodide of potassium.* It is then held in solution, and is sometimes known as *Lugol's solution.* It is made by dissolving *one part of iodine* (five grains) with *two parts of iodide of potassium* (ten grains), and adding water (to the extent of *one pint*). It contains *one grain* of iodine in four ounces of the solution. The treatment ought not to commence with a larger dose than *one drachm* three times a day. Small unirritating or alterative doses are the most efficient, and they may be gradually increased when necessary. The remedy may also be employed externally in the form of a liniment or ointment—the *unguentum iodum compositum,* of which a small portion may be rubbed upon the swelling night and morning. In some parts of India the application of an ointment of the *biniodide of mercury* was found very efficacious. The ointment is prepared by adding finely-powdered *biniodide of mercury* to melted lard or mutton suet. This ointment is then applied to the goitre about an hour after sunrise, and is rubbed in, by means of an ivory spatula, for about ten minutes—*the patient sitting with his goitre held well up to the rays of the sun as long as he can bear the exposure.* The ointment will probably produce a blistering effect, although no vesicles appear on the skin; and in the course of the day the ointment should be gently rubbed in again, and the patient sent home, with orders not to touch it with his hands, but to allow the ointments to be gradually absorbed. A second application is sometimes necessary in very bad cases. In 1855 no less than 500 or 600 persons were sometimes treated in a single day; and it is estimated that altogether about 60,000 patients have been so treated; so that the cases in the district are now far less numerous than formerly;

and the disorder is thus being extinguished (Mouat, in *Indian Annals* for April, 1857).

When medical treatment fails, surgeons have attempted to give relief to the symptoms by one of three operations; but so long as the disease does not interfere with any of the important functions of the body, nor produce serious discomfort, surgical interference is not warrantable. These operations are,—(1.) The introduction of setons through the tissue of the diseased gland—an operation which has been successful. A thin double wire is to be passed through the gland, and left there for a week (QUADRI, TANNER, JAMES). (2.) Tying the thyroid arteries which supply the goitre with blood, and so starving the tumor, has been attended with varied success (COATES, BRODIE, EARLE, WICKHAM). These means having failed, (3.) The gland has been extirpated—an operation which few surgeons would now think of undertaking.

Sir Thomas Watson justly observes, regarding these surgical interferences for the radical cure of goitre, that "there is not one of which the *average* results have been sufficiently prosperous to warrant its repetition, except in cases where life is put in jeopardy, or made miserable by the swelling; and where other methods, and particularly the treatment by *iodine*, have been tried and have failed" (*Principles and Practice of Physic*, vol. i., p. 795). He makes, however, an exception in favour of puncturing any cell or cyst containing fluid, which sometimes makes up a considerable portion of the tumor. Such cysts may be punctured without much risk, and with great relief to the patient.

EXOPHTHALMIC BRONCHOCELE.

LATIN EQ., *Bronchocele exophthalmica;* FRENCH EQ., *Goitre exophthalmique;* GERMAN EQ., *Klotzaugenkropf*—Syn., *Basdow'sche Krankheit;* ITALIAN EQ., *Broncocele esoftalmico.*

Definition.—"*Enlargement, with vascular turgescence of the thyroid gland, accompanied by protrusion of the eyeballs, anœmia, and palpitation.*"

Pathology.—A form of *goitre* totally different from the endemic form, and which has received the name of *exophthalmic goitre,* the *goitre* of *anœmia* or *spanœmia.* It is rare in children; more common in females than in males; and co-exists with wasting discharges, or supervenes upon them; such as leucorrhœa and menorrhagia in females, and hemorrhoids in males. It is sometimes associated with heart disease. The normal nutrition of the nerve-centres is obviously impaired. Sleep is disturbed and unrefreshing. Digestion is impaired. Pallor and anæmia therefore ensue, with excessively frequent palpitation of the heart and carotid arteries. A systolic *bruit* may be heard over the region of the heart, and sometimes also over the carotids; and during any excitement, a rushing or throbbing feeling is experienced in the head. The thyroid body now seems to act as a diverticulum. Simple hyperæmia prevails in the gland, which subsequently becomes hypertrophied and very large, partly from dilatation of its vessels, and partly from infiltration of the tissues with serum, or from simple hyperplasia. Sometimes cysts form with serous or colloid contents. Palsy of the vaso motor nerves connected with the carotids, thyroid gland, and heart, is the explanation given of this disease, which is far more common among women than among men.

The exophthalmic state is similarly induced; continued distension of the intraorbital vessels, with growth of fat, presses the eyeballs forwards; and the exophthalmic state may be reduced by pressure applied to the carotids. In fatal cases the eyes are observed to recede within the orbit after death (BEGBIE). Vision is rarely impaired. Dilatation of the cavities of the heart is the lesion which is induced in that organ, and dilatation of the veins is the last expression of the disease (Fletcher, in *Brit. Med. Jour.*, 23d May, 1863).

Symptoms.—Palpitation long continued, with frequent pulse (120 to 140), and excessive vascular excitement of the arteries of the upper extremities and carotids, especially with enlargement of the neck and prominence of eyeballs, are the earliest signs of this disease. A whistling noise is heard over the region of the thyroid, and a peculiar sensation may be felt by the hand laid over the gland: otherwise the general symptoms are very irregular; and the disease generally drags on for months or years.

The result may be unfavourable, by reason of dilatation of the heart, with diminished functional power. The patient becomes cyanotic and dropsical, with dyspnœa. Death takes place also, but more rarely, by cerebral symptoms. As a rule, however, the disease ends in recovery.

Treatment.—The use of iron and tonics generally are indicated; and considering what has been already said under diseases of the heart, the use of *digitalis* is indicated in connection with the cardiac symptoms.

The indications for treatment are, to allay the irritability of the stomach by the use of ice; to give bland, unstimulating, nutritious food in small quantities and at short intervals; to produce sound and refreshing sleep by morphia, or any such stimulating soporific; to administer digitalis; to steady the weak heart and control its excitement. Iron may improve the state of the blood; but the *hygienic* conditions in which the patient lives are mainly to be rectified, (FLETCHER, l. c.) Dr. J. Warburton Begbie recommends the use of *belladonna* in combination with iron (*Dublin Quarterly Journal of Medical Science*, Nov., 1865, by Dr. W. Moore).

SECTION II.—DISEASES OF THE SPLEEN.*

SPLENITIS.

LATIN EQ., *Splenitis;* FRENCH EQ., *Splénite;* GERMAN EQ., *Milzentzündung;* ITALIAN EQ., *Splenitide.*

Definition.—*Inflammation of the substance of the spleen.*

Pathology.—Primary inflammation is a disease which is extremely rare in this country. It is usually limited to certain districts, as Cambridgeshire, Essex, or other paludal places. It is common in the East Indies, especially in the low marshy districts of Bengal, and occurs in the paludal districts of other parts of the world. Now and then it is said to originate from a blow or other accidental violence, but which are much more apt to cause rupture than inflammation. Hæmorrhagic infarctions, occurring during the course of fevers and general contagious diseases, are the most frequent causes of consecutive inflammation and suppuration of the spleen (NIEMEYER). These infarctions are roundish or wedge-shaped collections of fibrine, varying in size from that of a pea to a hen's egg. At first they are brown-dark, or brownish-red, and quite hard. The lesion is sometimes seen in children under ten years of age.

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* As the spleen is a ductless gland, its diseases are treated of in this chapter rather than among diseases of the digestive system, following the diseases of the liver and pancreas, as in the classification of the College of Physicians.

The few cases of disease of the spleen occurring in this country will account for its pathology having been little studied. In diffuse inflammation, however, of this viscus, we find it enlarged, of a deep venous colour, and its tissue so softened as to be readily broken down, or even reduced to little more than the consistency of coagulated blood. Diffuse inflammation may terminate by resolution, or pus may form; and in this case one or more abscesses, often containing several ounces of pus, have been formed. The abscesses sometimes make their way to the surface. Dr. Baillie mentions that the spleen has been found in a state of gangrene.

Symptoms.—Acute inflammation of the spleen is seldom seen unless accompanied by ague; and the additional symptoms are probably tumefaction and some pain of the left side, followed, perhaps, by dropsy.

In chronic affections even abscesses will sometimes form without any marked local symptoms. Dr. Abercrombie gives the case of a gentleman who was dyspeptic, but took a great deal of nourishment, who was much reduced in strength and flesh, but whose pulse was seldom more than 96 to 100—whose nights were good, though he was occasionally slightly feverish, and who was able, till within a few days of his death, to drive out in his carriage. At length he died, after suffering for two or three days from diarrhœa, but without any suspicion of the spleen being affected. On examination, however, the spleen was found enlarged, and in its centre an abscess containing several ounces of pus.

The more common form of diseased spleen is hypertrophy; and in these cases it can almost always be detected by palpation or percussion, sometimes extending low down into the pelvic region, well over on the right side of the *linea alba,* and extending backwards almost to the spine. In these cases the patient complains of weight and uneasiness, rather than of soreness; his pulse is natural, but the countenance extremely sallow; his person greatly emaciated, his bowels irritable; and these symptoms are for the most part accompanied by œdema of the lower extremities, or by ascites. The most remarkable part of the history of these cases, however, is, that notwithstanding the sallow and emaciated state of the patient, he is often seized towards the close of the disease with hæmorrhage from the stomach and bowels, often so profuse that many pints have been passed or thrown up, greatly exhausting the patient, and rapidly hastening his dissolution.

The course of chronic splenitis is generally long, the patient usually surviving one or more years in the worst cases.

Diagnosis.—Enlarged spleen can only be confounded with encephaloid or other tumor of the abdomen.

Treatment.—Bleeding in *splenitis* has not been found to effect a cure, while mercury has been found for the most part, not only not to be useful, but even to be most pernicious. "I feel," says Mr. Twining, "more anxious fairly to show the baneful effects of mercury in the disease now under consideration, because the instructions usually laid down in the best systems of medicine do not inculcate the avoidance of mercury in any case of enlarged spleen, nor do they advert to the pernicious effects of that state of disease which I have termed vascular engorgement." In further proof of his position, Mr. Twining mentions thirteen cases in which the patient either died of mortification of the cheek, the nose, the upper lip, or after having lost all his teeth or a large portion of the jaw, in consequence of the use of mercury, and, if he survived the employment of this medicine, the spleen remained permanently enlarged. Dr. Voigt, physician to the Danish establishment at Serampore, writes that, although most authors recommend mercury, it is an indisputable fact that a very small quantity, even a few grains, generally occasion a profuse salivation and so violent an affection of the mouth that mortification sets in, the teeth drop out, the bones become carious, and death ensues. Consequently, mercury and bleeding ought to be little used; and in India, in their stead, a *spleen mixture*, not very dissimilar to that recommended by Celsus, is most in vogue. The best, according to Mr. Twining, consists of the following:—

℞. Pulv. Jalap; Pulv. Rhei; Pulv. Calumba; Pulv. Zingib; Potass. Supertart., ā ā ʒj.; Ferri Sulphat., ℈ss. Tinct. Sennæ, ʒss.; Aquæ Menthæ Pip., ℥ixss. Of this mixture an ounce or an ounce and a half is to be taken twice a day, or such quantity as may produce three or four stools in the twenty-four hours.

The spleen mixture is, in some instances, efficacious, but in a much larger number of instances it entirely fails; and under these circumstances the *iodide of potassium* and the *bromide of potash* have been recommended. The dose of the bromide of potash is gr. v. to x. *ter die*, combined with *camphor mixture*. The marked influence of the *biniodide of mercury*, in the form of an ointment rubbed into the skin over the surface of a *goitre*, in reducing its enlargement, has been already noticed (p. 663, *ante*). It has been similarly used in cases of enlarged spleen; and the invalid soldiers who suffer from enlarged spleens invariably apply for some to take away with them when they are discharged from Netley Hospital.

HYPERTROPHY OF THE SPLEEN.

Latin Eq., *Hypertrophia;* French Eq., *Hypertrophie;* German Eq., *Hypertrophie;* Italian Eq., *Ipertrofia.*

The spleen is more often hypertrophied, as a result of malaria or of *leucocythœmia.* In the *Medical Commentaries* a hypertrophied spleen is mentioned which weighed 11 lbs. Portal speaks of another that weighed 30 lbs.; and Lieutaud met with one, in a woman who had been ill seventeen years, that weighed 32 lbs.. It is singular that these large tumefied spleens sometimes subside very rapidly. Abercrombie mentions one that subsided in a week after the ague on which it depended had been arrested. The hypertrophied spleen is generally more or less indurated.

The spleen is occasionally atrophied, so that little more than a rudimentary spleen remains. It is also found indurated, and often greatly softened. Hydatids have been found in the spleen. In a few instances small portions of the spleen, about the size of a nut, are found indurated and nearly white. These appearances generally arise from embolism.

The following morbid condition is regarded by the College of Physicians as furnishing one form of splenic hypertrophy, namely:—(*a.*) Leucocythæmia; but, as will be seen, the disease here described as *leucocythæmia* has a much more extended pathology.

LEUCOCYTHÆMIA.

LATIN EQ., *Leucocythæmia;* FRENCH EQ., *Leucocythémie;* GERMAN EQ., *Leukæmie*—Syn., *Leucocythæmie;* ITALIAN EQ., *Leucocitemia.*

Definition.—*A disease,* sui generis, *in which the number of white corpuscles in the blood is greatly increased, with a simultaneous diminution of the red. This state is brought about by chronic exhausting diseases, exposure to cold and wet, or serious acute affections—such as typhus fever, pneumonia, puerperal fever, affections of the lymphatic glands or of the spleen, and is attended sometimes by cough or diarrhœa, epistaxis, hæmorrhagic effusions, furunculous or pustulous eruptions.*

Pathology.—In the present state of our knowledge regarding leucocythæmia, an account of the phenomena which attend upon its course and the conditions of its occurrence are all that can be given.

Having conveyed, in the previous editions of this work, an erroneous chronological account of the discovery of this remarkable disease, and of the steps through which the views regarding its pathology, as at present entertained, were successively reached, I am the more anxious now to do justice to those distinguished men whose conjoined investigations have enriched science with the knowledge of leucocythæmia which we now possess.

In the sixty-fourth volume of the *Edinburgh Medical and Surgical Journal* a case of disease of the spleen is described by Dr. Craigie, "in which death took place in consequence of purulent matter in the blood." The case occurred in 1841, and proved fatal on the 1st of April of that year. The late Dr. John Reid examined the case, and, "on examining the blood of the veins of the abdomen and sinuses of the brain by the microscope," found "that it contained globules of purulent matter and lymph." Dr. Craigie inferred "that, by some means or other, purulent matter and lymph had been mixed with the blood, and, circulating with it, had given rise to the peculiar febrile and inflammatory symptoms which occurred during life, and to death in the manner in which it had taken place." He inferred—that the spleen was the only organ from which the purulent matter and lymph could have proceeded, it having been for several weeks in a state of chronic inflammation—that in this form of disease of the spleen the pus-cells are secreted, and, being mixed with the blood, they cause much disorder in the sanguiferous system, and finally destroy the patient. Dr. Craigie thus clearly recognized a connection between the diseased state of the spleen and the changed condition of the blood; and that "it was in some respects new." On this account he made a correct description of it, expecting at some future period that the chief facts might be confirmed. He kept the case unpublished till 1845, and it was only published then in consequence of the occurrence, to another physician in the same hospital, of a case "in many if not in all respects similar, which led Dr. Craigie to anticipate similar results, and which went far, as he thought, to confirm his conclusions deduced from the first case, (*Edin. Med. and Surg. Journal* for 1845, p. 400, *et seq.*) The details of the second case referred to by Dr. Craigie were published by Dr. Bennett in the same volume of the *Edinburgh Medical and Surgical Journal* immediately after the account of Dr. Craigie's case. Dr. Bennett describes his case as one of "*Hypertrophy of the Spleen and Liver, in which death took place from suppuration of the Blood;*" and although the most evident lesion during life was enlargement of the spleen, Dr. Bennett agrees with Dr. Craigie "in thinking that the immediate cause of death was owing to the presence of purulent matter in the blood, notwithstanding the absence of any recent inflammation or collection of pus in the tissues," and that it produced the febrile symptoms. In these valuable papers Drs. Bennett and Craigie are at issue, however, about the source of the pus in the blood. Dr. Bennett considered his case particularly valuable, because he believed it demonstrated "the existence of *true pus*, formed universally within the vascular system, independent of any local purulent collection from which it could be derived" (l. c., p. 414). He believed the white corpuscles he saw in the blood "were true pus-globules," and he then was of opinion that they were formed in the *liquor sanguinis* within the vessels, independent of inflammation or of phlebitis, or what was then understood by *pyœmia;* that the transformation had taken place throughout the system, and that the whole mass of blood was affected: and the case appeared to him capable, in this respect, of furnishing an important fact which may serve to throw light on the doctrine of *Zymosis,* as applied in Pathology (l. c., p. 423).

In the same year (1845), about a month after the publication of Dr. Bennett's case, Professor Virchow, of Berlin, described and explained, in *Froriep's Journal,* of November, 1845, a disease of the blood as due to an increased development of white blood-cells. No inflammation of veins was observed; and the hypothesis of any spontaneous formation of pus in the blood (pyæmia) was contrary to the pathological doctrines of Virchow. To this form of disease he gave the name of "white" or "colourless blood." As to its cause, he distinctly points out, *as primary,* the condition of the spleen, entertaining the idea of an increased formation of colourless blood-cells, through the functional relations of that organ.

In 1845-46 several cases of leucocythæmia during life were recognized in this country. Dr. Fuller, of St. George's Hospital, in December, 1845, was the first to determine this condition during life; and it was also diagnosed by Drs. T. K. Chambers and Walshe, in London, and by Dr. Douglas, in Edinburgh, in 1846.

Virchow continued to oppose the view of Dr. Bennett as to this affection having its origin in the formation of pus in the blood, or that its formation is in any way of the nature of a *Zymosis;* maintaining, on the contrary, the correctness of his first opinion, that the essence of the disease consisted in an increase of the colourless blood-cells (*Medicinische Zeitung*, August and September, Berlin, 1846).

In 1847 Virchow collected and described cases from the older authors, and compared them with those observed in England and those seen by himself; and finding also a further basis in favour of his views in the theory of Hewson and Donné, regarding the function of the spleen in the propagation of the blood, he naturally became more confirmed in his opinion, that, from some altered relation of the spleen to the blood-cells, might be established those conditions under which "white blood" would be produced. In the *Archives of Pathological Anatomy* of the same year a case of great value is described by him as a new form of this "white-blood" disease. In this form the spleen presented no change whatever, whilst the lymphatic glands were enormously enlarged. He was now able to declare with more confidence that the "white-blood" disease proceeded from a primary affection of the spleen and lymphatic glands, as a direct consequence of which an increased development of colourless blood-cells takes place; and thus he considers that the disease, in its turn, throws light on the physiological functions of these glands as eliminating organs of the blood.

In 1851-52 Dr. Bennett gave a most interesting and systematic view of the whole subject, first in the *Monthly Journal* of 1851, and afterwards in a separate work, entitled *Leucocythæmia, or White-cell Blood*, in which the explanation of the pathology of this disease is no longer referred to the spontaneous development of pus in the blood.

Although Dr. Bennett originally set out upon what has since been believed to be an erroneous track in the interpretation of the phenomena of leucocythæmia, yet he was the first to show that these phenomena were new to science, while the more apparently correct generalization and elaboration of the subject seems, from the chronological evolution of the history of the disease, to be mainly due to Virchow. He was the first to recognize and regard the colourless cells in leuchæmia or leucocythæmia to be identical with the colourless globules of the blood—the view at present generally entertained. More recent observation, pointing to the formation of pus from the white corpuscles of the blood, may (if found correct) again change the views now generally entertained. It is greatly to be regretted, however, that so much personality and ill-feeling has been imported into the historical records of this interesting discovery; and if the reader desires to read the several sides of the lengthened controversy, let him consult—(1.) The original cases of Craigie and Bennett in the sixty-fourth volume of the *Edin. Med. and Surg. Journal;* (2.) A review of Virchow's *Handbook of Pathology*, in the June number of the *Edin. Monthly Med. Journal* for 1854, p. 546; (3.) An able letter by Professor Kölliker, in the October number of the same journal, p. 374; (4.) A reply by Dr. Bennett to the same, p. 377; (5.) Dr. Bennett's work on *Leucocythæmia;* (6.) Dr. Bennett's more recent Lecture (vi.) in the *Lancet* of April 4, 1863.

The morbid state expressed in the definition has been observed by many physicians and pathologists under various complex conditions; and hence a variety of opinions have been entertained regarding it in Germany, England, and France, by Vogel, Remak, Henle, Nasse, Weber, Rokitansky, Kölliker, Parkes, Jenner, Gulliver, Piorry, Bichat, Velpeau, and others, who have described cases since Bennett and Virchow first wrote on the subject. The increase of the colourless corpuscles of the blood, which is the prominent character of this disease, does not seem in any case to have existed or occurred by itself. Other morbid states, or some change-producing event in the constitution, such as childbirth (Robert Paterson), precede, co-exist with, or succeed the augmentation of the colourless corpuscles.

The most frequent complication consists in the enlargement of the spleen. This enlargement is so constant that its existence, if not otherwise accounted for, would at once indicate that leucocythæmia prevailed, and would suggest a microscopic examination of the blood. In nineteen cases, Vogel writes that the splenic enlargement was present in sixteen; and in three cases the weight of the spleen was estimated at more than seven pounds.

Constituents foreign to normal blood have been found by Scherer in a qualitative analysis of the blood in a case of leucocythæmia, where the spleen was enlarged. These consisted of lactic, acetic, and formic acids, gelatin, and a peculiar substance (hypoxanthin) to the amount of from ·4 to ·6 *per cent.* Scherer finds, also, that this same substance exists naturally in the pulp of the healthy spleen. This form of the disease has been named *splenæmia* by Virchow. In it the globules (white) predominate which are peculiar to the elements of the spleen.

The *liver* is also frequently enlarged in this disease, but not to so remarkable a degree as the spleen. Vogel writes that as often as thirteen times out of nineteen cases it was either enlarged or otherwise morbidly altered.

Affections of the *lymphatic glands* predominate in some cases, rather than enlargements of the *liver* or the *spleen.* According to Vogel, such have been observed eleven times out of nineteen cases. Virchow considers that some kind of lymphatic *diathesis* prevails,—that there is a progressive inclination of the lymphatic system to the formation of the lymphatic elements. In some instances observed by him there seems to have been a new formation of glandular tissue, or that the glandular tissue tended to grow beyond the pre-existing boundaries of the glands. He has observed this development of lymphatic gland-tissue to take place in the liver in a remarkable case which contained numerous small whitish granules, about the size of the natural lobules of the liver, and which exhibited under the microscope nuclear and cellular elements quite like those of the lymphatic glands. This infiltration of the liver followed the ramifications of the portal vein. In one of these cases he observed a similar alteration in the kidney.

This constitutes the *lymphatic* form of *leuchœmia,* or *lymphœmia,* first described by Virchow in 1847.

In such cases the elements of the lymphatic glands prevail in the blood, which is then characterized by innumerable round granulated nuclei, generally provided with nucleoli, of the size of the usual nuclei of the lymphatic glands. Here and there are also to be seen cells, consisting of such a nucleus, surrounded by a membrane closely attached to it.

There are three possible conditions given by Virchow under which these elementary cells in the blood may originate,—(1.) They may multiply in the blood by the subdivison of pre-existing cells; (2.) They may be primarily introduced into the blood through the lymph or chyle, which are conceived to convey the developed as well as the undeveloped globules derived from the lymphatic glands, the spleen, and its connecting tissue; (3.) That they are formed on and detached from the walls of the blood-vessels has not yet been proved.

Virchow regards the colourless blood-corpuscles as simple cells, without any specific character, whose transformation into red globules cannot take place; that they therefore form a relatively superfluous constituent of the blood—a kind of excess or waste. The transformation of lymph-globules into red globules takes place before passing into the general circulation; and it appears that if a certain cell, when passing into the blood, has gone beyond that stage of development, it is ever afterwards unfit to undergo its specific coloured metamorphoses. The idea that the cells are of a purulent nature has been now abandoned; and there is no evidence to support the doctrine that they are the result of *pyœmia.* Besides the spleen and the lymphatic glands, the other blood-glands, such as the *thyroid gland* and *supra-renal capsules,* are occasionally degenerated, as well as Peyer's glands and the mesenteric glands; and leucocythæmia may arise from disease in them as well as from the spleen or lymph-glands.

The statistics regarding the ages at which the disease has been observed are given by Vogel as follows:—One case was observed under ten years of age, two between ten and twenty years, three between twenty and thirty years, seven between thirty and forty years, four between forty and fifty years, three between fifty and sixty years, and three between sixty and seventy years.

Symptoms.—In the majority of cases there are obvious indications of general ill-health; and the most prominent symptom has been tumefaction of the abdomen, depending upon an enlarged spleen and liver. Ascites and anasarca of the lower half of the body are not unfrequently present; and a tendency to œdema may commonly be observed, the general surface of the body being usually pale. Transitory pains are frequently experienced in the abdomen.

Intestinal disorders are often also present, such as vomiting, constipation, or diarrhœa, and jaundice is not unfrequent; but diarrhœa is one of the most dangerous complications, and the most difficult to arrest or control.

A considerable amount of dyspnœa may prevail, which cannot be accounted for by elevation of the diaphragm merely. Hæmorrhage often occurs in the form of epistaxis, or takes place from the gums. A persistent increased secretion of uric acid has also been observed in the urine. The disease generally runs a chronic course, and a high degree of emaciation ordinarily accompanies it. Leucocythæmia is usually well established before it is noticed, and before any remarkable disturbances in the general health have occurred. It is not till towards the fatal termination that any fever sets in, which then assumes the hectic type.

The Diagnosis of the disease consists in demonstrating the extreme increase of the colourless blood-cells, which may be done as follows:—

1. By microscopic examination of the blood, for which a single drop is sufficient, most conveniently taken from a needle puncture in the finger, and examined under a power of at least 250 diameters. If the disease exists, the colourless corpuscles will be seen to form a sixth, a fourth, or even a half or more of the numbers of the whole corpuscles.

2. If a large quantity of blood is obtainable by venesection or by the cupping-glasses, and freed from fibrine by heating, and placed in a high narrow little glass, so that the corpuscles sink to the bottom, the upper part of the mass looks whitish coloured, like milk. The milky character does not vanish on agitation with ether, and is not produced by fat-globules suspended in the blood-serum, but by the prodigious number of the colourless blood-corpuscles.

3. The clot of leucocythæmic blood shows on its surface greyish-white granulations, which, being observed under the microscope, are seen to consist almost entirely of colourless corpuscles, distinguishing the condition in leucocythæmia from the ordinary buffy coat; and the separated serum being clear, and not turbid, distinguishes the condition from a fatty condition of the blood.

4. In the dead subject there are found in the heart and in the great veins large, soft, semi-fluid greyish-yellow coagula, which, on microscopic examination, are seen to consist almost entirely of colourless corpuscles (VOGEL).

Causes.—The causes which bring about leucocythæmia are entirely unknown; but it seems several times to have suggested itself to Virchow that acute inflammatory processes may lay the foundation of the morbid state; and in an interesting review of the writings of Virchow in *The Brit. and For. Med.-Chir. Review* for July, 1857, there is related a case of the lymphatic form of the disease, whose origin obviously dates from inflammatory swellings of the lymphatic glands after exposure to cold and wet. I saw a similar case in Guy's Hospital, in July, 1863. It occurred to a man after exposure to cold and wet on Epsom Downs at the time of the races there.

My friend, Dr. Robert Paterson, of Leith, from whose teachings in the Edinburgh Royal Infirmary I profited as a student, records some cases in connection with pregnancy, in the *Edinburgh Medical Journal* for June 1870, p. 1073. He there shows how this disease is able to linger long in the constitution in a masked or subacute form, and unmarked by any easily recognized symptom, until the occurrence of some change-producing event in the constitution, such as childbirth, after which the rapidity of its fatal course was to be measured, not by days but by hours. He records several cases. His observations are borne out by Vidal, who says that pregnancy in four cases out of ten is the commencement of leucocythæmia.

Prognosis.—Hitherto no case of cure is known. The duration of the disease is from thirteen to fourteen months—the minimum three months, the maximum four years.

Treatment.—The most varied remedies have been tried without checking the increased formation of colourless corpuscles; but it is suggested that if it is possible to discover the glandular or splenic affection early, before the alteration of the blood has made much progress, it is probable that the disease may be averted. Tonics, nutrients, and stimulants are indicated, to support the system. The use of the nitro-muriatic bath ought not to be neglected, and the indications given under anæmia and chlorosis may be followed.

PECULIAR ENLARGEMENT OF THE SPLEEN AND LYMPHATIC GLANDS—SYN., HODGKIN'S DISEASE.*

Definition.—*A disease characterized by a peculiar white deposit in the spleen, sometimes also in the liver, kidney, and lungs* (WILKS), *and by an enormous enlargement of the lymphatic glands throughout the body, accompanied during life by a remarkable anæmia and disposition to anasarca.*

Pathology.—Although this disease has not yet been recognized in the nomenclature of the College of Physicians, yet it seems to be an affection presenting as striking peculiarities as any in the Nosology, and therefore deserving of a distinct appellation and description (WILKS). It seems to have arrested the attention of Drs. Bright and Hodgkin many years ago; and they also recognized the frequent association of diseased lmyphatic glands with it. Dr. Bright, indeed, refers to the circumstance as having been originally pointed out by Dr. Hodgkin, in vol. xvii. of the *Medico-Chirurgical Transactions* (1832). Dr. Wilks considers that the subject has got into a false position, mainly in consequence of having been referred to in connection with lardaceous disease. I fear I have aided in this confusion; and therefore I hope to aid Dr. Wilks, by thus endeavouring to remove the subject from the false position in which it has been placed, by giving here a summary of the pathology of the affection as given by him (*Guy's Hospital Report*, vol. xi., p. 56).

The disease is not to be confounded with lardaceous disease or any other morbid process. The enlarged glands may be met with in various forms of disease, and sometimes with lardaceous disease; and thus the two affections may seem to have a relationship they may not really possess. The lesion also appears to have a likeness to tubercle on the one hand, and to cancer on the other. It is, however, a disease, *sui generis.*

The enlargement of the glands "appears to be a primitive affection of these bodies; and there is no reason to suppose it is due to *inflammation* or *scrofula,* nor, indeed, attributable to the formation of any adventitious structure. It appears, in nearly all the cases, to consist of a pretty uniform texture throughout, and thus to be the consequence of a general increase of every part of the gland, than of a new structure developed in it (HODGKIN).

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* A disease not recognized in the nomenclature of the College.

"In conjunction with this affection of the absorbent glands is the state of the spleen, which is strictly pervaded by defined bodies. We might suspect that the bodies in the spleen, like the enlarged glands, were due to an enlargement of a pre-existing structure—an idea which may derive some support from the fact that, although in the human spleen no glandular structure is distinguishable, in those of some inferior animals a multitude of minute bodies exist which appear to be of that nature. Malpighi, indeed, considered the acini to be glands (Wilks, *loc. cit.*, p. 59).

In the last few cases observed by Dr. Wilks, the new adventitious material has been found in the liver, kidney, and lungs, besides in the spleen.

The peculiarity of the affection consists in the whole glandular system being specially affected; and it must take its place in the ranks of malignant diseases, or amongst those affections which are characterized by new growths in the system. The peculiar symptoms are to be referred to the universal affection of the glands. The lymphatic glands appear to be affected for a considerable period, perhaps many years, before the system suffers, and that subsequently the spleen becomes specially involved, and afterwards the other organs. It is possible that propagation to the spleen takes place in the course of the lymphatics, thereby affecting the corpuscles of the spleen, which are intimately connected with the absorbent system. The deposit in Glisson's capsule of the liver may be transmitted through the same channel. In malignancy the disease takes a place between cancer and tubercle.

Morbid Anatomy.—In the lymphatic glands the microscope shows an abundance of cells, scarcely distinguishable from the normal secreting bodies, and with more or less fibre-tissue. In the liver the material is much tougher and fibro-nucleated; whilst in the lungs, spleen, and kidneys it is composed mostly of cells resembling tubercle.

Dr. Wilks has never met with well-marked leucocythæmia in Hodgkin's disease, except in one case where the spleen was hypertrophied; and only one case where lymphatic enlargement and lardaceous disease were associated.

Death occurs through the derangement of the structure and functions of the glands.

Whether the disease is constitutional from the commencement, or whether the system is infected from a local source, is a question yet unsolved. It may commence in one part, and from this, as a focus of contamination, be propagated through the body; or it may be constitutional from the beginning.

Symptoms.—General ill health, paleness and sallowness of complexion precede any other signs. Lymphatic glands are subsequently found enlarged, as in the neck, axillæ, and groins. Weakness is felt, which increases; and the patient can no longer walk nor run up a stair. He totters in the legs from feebleness. Sexual appetite is lost, and flesh is lost; so that emaciation with marked anæmia exists—a pale sclerotic, and a feeble pulse. The legs become œdematous.

Prognosis is generally unfavourable; and no special treatment can be indicated beyond that laid down in cases of anæmia and of leucocythæmia.

LARDACEOUS SPLEEN—Syn., AMYLOID DISEASE; WAXY SPLEEN.

LATIN EQ., *Lien lardaceous*—Idem valent, *Morbus amylodes*, *Lien cereus;* FRENCH EQ., *Dégénérescence lardacée*—Syn., *Maladie amyloïde;* GERMAN EQ. *Speckige entartung, der Milz*—Syn., *Amyloide entartung;* ITALIAN EQ., *Milza lardacea*—Sin., *Malattia amiloide.*

Definition.—*A disease in which the texture of the spleen, and especially the Malpighian sacculi, are filled with lardaceous material, so that it is much larger and heavier than in health.*

Pathology.—The chief part of the substance of the spleen seems to be made up of distinct translucent Malpighian sacculi closely crowded together (BENNETT).

The lardaceous disease of the spleen is frequently associated with a similar condition of other organs, especially the liver and the kidney. It is a condition characterized by great firmness, a peculiar waxy-like consistence, and with a distinctness and transparency of the Malpighian sacculi which are not usually very obvious (W. T. GAIRDNER). Of all the viscera and tissues of the body, the spleen is the one which is comparatively the most frequently affected by the lesion, although it was generally unknown up till 1853-4. Of the bodies examined in the Royal Infirmary of Edinburgh it was observed in 10 *per cent.* of all.

In the most extreme cases of the lesion the spleen is enlarged, and has a swollen aspect. Its weight and density are greatly increased. It feels to the touch like the consistence of wax and lead, and its section shows a dry and smooth surface. After exposure to the air, its pulp may become of a bright red colour; and then the Malpighian sacculi appear large and distinct, forming round, colourless, *transparent* granules about the size of a pin's head, slightly prominent, and so hard that they may be picked out by a knife. The pulp is greatly diminished in quantity, and seems in some to be entirely absent; so that the sacculi are crowded together, and the tissue in their vicinity appears as a continuous mass of a globular form. A small arterial twig can sometimes be seen passing into or through these Malpighian sacculi at their central part.

Microscopic sections are easily made, and their characters are even more striking than those presented to the naked eye. Under a low power (40 to 60 diameters) the Malpighian sacculi appear as large clear spaces, of a circular or oval form, surrounded by the dark-red pulp. Under the higher powers (above 250) the nature of the alteration is seen to consist mainly of an alteration in the normal corpuscles of these sacculi, which are converted into, replaced, or added to, by masses of a colourless, dense, highly translucent and homogeneous material, and a careful examination discloses the outlines of irregular cell-forms. But these masses, owing to their roughness, are not easily broken up into the particles which compose them (SANDERS). The translucent parts are very little acted on by re-agents (acids, alkalies, alcohol); but if alkalies, such as *liquor potassæ*, be first applied to a microscopic section, the iodine test will fail until the section is neutralized by acid. Its characters are marked and permanent, so that there is no difficulty at any time in recognizing or identifying the characters of lardaceous disease in the spleen.

Several varieties of the waxy spleen may be noticed. Two forms at least are distinctly indicated by Virchow, Sanders, and Wilks (although the latter observer says three):—

1. *The form in which the Malpighian Sacculi are the structures prominently affected.*—They are sometimes large, and sometimes very small. In the former state we recognize the sago-like granules described by Virchow. In the latter case they are best seen after exposure to the air for a little time, or after having been well washed in water and then put in alcohol or into a solution of chloride of zinc, which brings out the waxy appearance very distinctly. In the most extreme cases they never occupy more than about half the bulk of the organ, the intervening pulp-substance being healthy.

2. *A form in which the Pulp-substance and Trabeculæ are mainly affected, leaving the Sacculi intact.*—It presents the same waxy consistence, the smooth dry section, and other characters of the lesion; but the sacculi are obscured by the peculiar translucent substance which pervades the pulp, and which looks as if melted tallow had been poured into the trabecular spaces (Wilks).

Histologically, there is no real difference between these two forms, because the corpuscles of the sacculi and of the surrounding pulp-substance are now known to be identical (Busk and Huxley, in Wedl, p. 247).

The disease seems to commence—

(1.) In the arterial capillaries, where the little lateral bulgings are in connection with the Malpighian corpuscles.

(2.) These sacculi contain at first normal splenic cells; but at a later stage the contents of the sacculi become irregular and granular, and corpuscles of a gelatinous lymphy appearance become changed into the minute masses of the waxy substance.

(3.) Subsequently the trabeculæ and pulp-struma become affected (Busk and Huxley, as above).

Some cases of albuminoid disease related by Dr. Jenner are evidently of this second nature.

From this description, and from what has been written in Part I., vol. i., on the subject of *Lardaceous disease* (q. v.), the term "*degeneration,*" which has hitherto been generally used, is quite inapplicable. The spleen is added to in bulk and weight by a new material; its textural elements are not replaced, which is the nature of degeneration to effect. Neither has it been shown that the new material is "*amyloid;*" but it is rather of the nature of albumen. The phrase "*amyloid degeneration,*" therefore, is one which is incorrect in both its terms (Wilks), and one which ought no longer to be used.

It is only by an extended inquiry into the class of cases in which lardaceous disease is found that we can hope to learn anything satisfactory as to its pathology; and the reader is referred to the excellent reports upon ninety-six cases already given by Dr. Wilks, in vol. iv. (1856) and vol. xi. (1865) of *Guy's Hospital Reports;* and by Dr. Pavy, in vol. x. of the same valuable series. These cases prove indisputably that lardaceous disease of the spleen, or any other organ, is one implying a long-standing and deep-seated cachexia; and, in its most intense form, is seen after a protracted caries and necrosis of bone; having its origin in scrofula, or syphilis, or even external injury, where the injury leads to protracted bone disease. Hence the question is still undecided, whether the disease arises from a local source, such as the injured bone and the morbid processes going on in it, or whether it is a constitutional general disease. Wilks gives several instances, such as the following:—"A strong young man, in good health, falls and strikes his hip; a disease is thus set up which causes his death at the end of two years: his organs were then found to be lardaceous" (No. 89 of cases in *Guy's Hospital Reports*, vol. xi., p. 47). It is evident that the question is one of great practical importance to the surgeon.

Symptoms.—As regards the spleen, lardaceous disease is only known to exist by its discovery after death—no symptoms that can be recognized (unless enlargement and hardness) having ever been perceived as constantly associated with the lardaceous affection.

SECTION III.—DISEASE OF SUPRA-RENAL CAPSULES.

ADDISON'S DISEASE—Syn., BRONZED SKIN, MELASMA ADDISONII.

LATIN EQ., *Morbus Addisoni*—Idem valent, *Cutis œrea, Melasma Addisoni*; FRENCH EQ., *Maladïe d'Addison, Melasma Addisonii;* GERMAN EQ., *Addison'sche Krankheit*—Syn., *Malasma Addisonii;* ITALIAN EQ., *Malattia dell' Addison*—Sin., *Melasma dell' Addison.*

Definition.—*Disease of the supra-renal capsules, with discoloration of the skin; or, a morbid state which establishes itself with extreme insidiousness, whose characteristic features are anœmia, general languor and debility, and extreme prostration, expressed by loss of muscular power, weakness of pulse, remarkable feebleness of the heart's action, breathlessness upon slight exertion, dimness of sight, functional weakness and irritability of the stomach, and a peculiar uniform discoloration of the skin, which becomes of a brownish olive-green hue, like that of a mulatto, occurring in connection with a certain diseased condition of the supra-renal capsules. The progress of the disease is very slow, extending on an average over one year and a half, but may be prolonged over four or five. The tendency to death is by asthenia, the heart becoming utterly powerless, as if its natural stimulus—the blood—had ceased to act.*

Pathology.—The pathological significance of morbid states of the supra-renal capsules was brought prominently before the profession, both in this country and on the Continent, by the original observations of the late Dr. Addison, then the senior physician of Guy's Hospital. The cases recorded in the medical journals since Dr. Addison wrote, which connect uniform discoloration of the skin (a condition now known by the name of "bronzing") with various morbid states of the *supra-renal* bodies, are now so numerous that, as a clinical fact, the connection cannot be disputed; but the exact relationship and pathological significance of the morbid states thus connected are still open questions, especially as regards the pathology of the constitutional cachexia which exists. Morbid states of the *supra-renal* capsules are not always attended with bronzing of the skin. It appears, indeed, if the cases recorded are carefully analyzed, that symptoms and phenomena of a very important kind have been lost sight of in describing this constitutional disease, while an undue importance has been placed upon the bronzing of the skin. It is to the cachexia that Dr. Addison calls special attention; but his commentators have been carried away by the inquiry regarding the colour of the skin and its connection with the capsular disease. These writers have overlooked the more important portion of his observations, and have been induced to consider the *causes* and *nature* of the bronzing of the skin, which, being established, may be received as a most valuable symptom of a prevailing constitutional cachexia, in which the abnormal deposit of pigment is associated with anæmia and intense prostration, with the phenomena stated in the definition. There can be no doubt that the bronzing must appear of very secondary importance compared with the symptoms and pathology of that peculiar cachexia which attends the cases of *supra-renal* capsular disease, as described by Drs. Addison and Wilks. There seems to be no doubt that the cases Dr. Addison described belong to the class of diseases now under consideration; and that they are similar in many respects to forms of anæmia already noticed, and more especially to leucocythæmia. "In almost all the cases," writes Mr. Hutchinson, "there would seem to have been great deprivation of the coloured constituents of the blood, as manifested by the pallor of those parts of the skin not involved in the bronzing, the great flabbiness of the muscles, and the pearly state of the conjunctivæ." By a strange oversight, however, it would appear that the blood has been subjected to but few examinations. "In two only" of the cases, writes the same author, "was the blood examined, and in both of them it was found to be loaded with white corpuscles." In all the cases a most remarkable and fatal cachexia prevails, and the value of Dr. Addison's observations consists in showing that a peculiar bronzing of the skin, combined with *asthenia*—of which emaciation is not a necessary accompaniment—attends this cachexia, and indicates organic disease of the *supra-renal* capsules associated with this constitutional state. His observations are at the same time of the greatest value, as showing how well-directed pathological inquiry may advance the science of physiology; for the cases detailed throw some light on the influence, at least, of the *sapra-renal* capsules in the maintenance of health.

Although the change of colour of the skin would seem to be a most marked and constant symptom, still its does not appear, as has been stated by a reviewer of Dr. Addison's work, that this change "is one of the earliest symptoms of the disease." On the contrary, there are good grounds for believing, both from the history of the cases, and from physiological experiment and observation, that the change in the colour of the skin, which has been termed "*bronzing*," does not come on for a long time—that from its nature as a pigmentary change it is of slow production; that while, in almost all the well-marked cases in which it has occurred, the symptoms had existed from one to three years, in other cases where it did not exist it has been alleged that time was not afforded between the establishment of lesions in the supra-renal bodies and the fatal issue for the production of the pigmentary deposit. The change of colour seems to depend on the chronicity of the disease; so that, if the disease progresses rapidly, no discoloration is observed, the phenomena being simply those of asthenia. One very important point is thus remarkably deficient of illustration—namely, *the early symptoms of this cachexia* independent of *bronzing of the skin.* From what has been shown relative to the disease, it would appear that when *bronzing* of the skin has been established, a sign of disease has been discovered when it is too late to be of any service, for all the cases appear to have terminated fatally in which this state was unequivocally established.

In malarious, malignant, and cachectic diseases it is not unusual, but rather the rule, for the serum of the blood to assume a dark and dirty hue, and that ultimately the cutaneous surface comes closely to approach the colour of jaundice, differing from it only in being more lurid and dusky; and it is believed that the hue of the skin, which becomes of so dark a tinge in some malignant organic diseases, is due to the admixture of morbid matters absorbed from the seat of local mischief, and which so tinges the serum of the blood that the *rete mucosum* is rendered dark. If, then, it is true that in this cachexia the supra-renal capsules are always diseased, and if it be true, as M. Vulpian has found (*Med. Times*, October 4, 1856), that the *supra-renal capsules* differ from all other organs in the body, in the presence of a substance which has two peculiar re-actions—one with perchloride of iron, and the other with the tincture of iodine, the first of which gives a dark-blue tint—is it not probable that the coloration of the skin in *supra-renal capsule* diseases may result partly from—(1.) The cachectic state; (2.) The organic lesion of the *supra-renal capsules;* and (3.) The re-action of the peculiar substance in these bodies upon the iron of the blood, which the morbid organic changes in them have allowed to mingle with the circulating fluid? It is, unfortunately, the *supra-renal capsules* only which have been carefully examined in the cases recorded; and in connection with the cachexia and the "bronzing," the lesions they exhibit have been described as of a very variable character, consisting of—(*a.*) Acute or recent inflammation ending in abscess; (*b.*) Atrophy with concretions; (*c.*) The conversion of the organs into indurated fibroid enlargements; (*d.*) Tuberculous deposition in various stages; (*e.*) Carcinomatous deposition.

A definite lesion, however, has been recently pointed out by Dr. Wilks, which will be referred to presently.

Sometimes the lesion appears to have been secondary to morbid conditions, apparently of a similar nature, in other parts, all of which must therefore be interpreted as several local expressions of the constitutional disease which prevails: and occasionally the capsules seem to have been the only structures in which lesions were detected; while the degree of "bronzing" of the skin appears to have been proportionate to the length of time which the *supra-renal capsules* are presumed to have been diseased; but neither the time nor the number of cases in which such a proportion can be traced is accurately made out. The general symptoms are those of a person constitutionally diseased, *not* always proportionate in their severity either to the nature or to the extent of the disease in the *supra-renal capsules.* The *post-mortem* appearances seem to have been in some instances associated with the tuberculous diathesis; but in the best-marked cases there has been no appearance of tubercle on any part of the body (WILKS); and in no case of general tuberculosis has Dr. Wilks ever met with an instance where the *supra-renal capsules* were affected in the manner of Addison's disease. In some cases the spleen was much enlarged, the kidneys pale, and in the last stage of fatty degeneration (TAYLOR). Out of 500 cases of *post-mortem* examinations of all kinds of cases, made at Guy's Hospital by Dr. Wilks, only two instances were observed in which, the *supra-renal capsules* being morbid, the skin did not betray the lesion by "bronzing." In some cases the mesenteric glands are stated to have been enlarged; and calcareous concretions have been noticed in the *medulla oblongata.*

Gastro-intestinal disturbance prevails during life; and a condition of mucous membrane is found after death which may be associated in its pathology with this irritation. The stomach is often ecchymosed, and the glands of the intestines enlarged. In several cases Brunner's glands in the duodenum, and the solitary glands in the lower end of the ileum and in the colon, were very prominent.

In one case which, through the kindness of my first teacher, the late Dr. William Monro, of Dundee, I had an opportunity of carefully inspecting after death, in June, 1856, the following lesions existed:— "The body generally was anæmic. The dark colouring of the skin was most conspicuous in the vicinity of the knees, and on the lateral and posterior regions of the neck. The deposits of pigment gave to the parts a dirty sordes-like appearance where the mucous membrane of the skin meets the lips, and especially at the angles of the mouth. The heart was small and flabby. Tuberculous deposits were sparingly disseminated throughout the apices of both lungs. The spleen, the liver, and the kidneys were severally adherent to the adjacent parts, but their structures appeared normal. The supra-renal capsules were morbid, and the sympathetic nerves from the lesser *splanchnic* were greatly increased in size, as were the ganglia of the *solar plexus* towards the side of the organ most diseased and in contact with it. The texture of the nervous parts was of a bright rosy hue, as if under the influence of vascular excitement. The mucous membrane of the mouth was thin, pale, and bloodless, the labial and buccal glands shining prominently through. The stomach and glandular substance of the intestinal tube were uniformly thin throughout. In the stomach the solitary gastric glands were remarkably prominent, while the mucous membrane generally was wasted and atrophic. Microscopic sections from the jejunum and ileum showed the villi remarkably attenuated, and the mucous membrane very readily separated from the adjacent muscular part of the gut. The tubular glands of the mucous membrane of various parts examined were almost entirely gone, and their place supplied by granular amorphous material. The average specific gravity of the mucous membrane of the intestines was 1·040.

There are good grounds for believing that Addison himself entertained the belief that death in such cases may be due to the implication eventually of the ganglionic nerves. Some of the symptoms point to this; and of the special phenomena associated with the cachexia, it may be said that the nervous centres are at least impaired. It has occurred to me also, that, in cases of this disease, all the viscera ought to be examined as to the re-action of their minute blood-vessels with iodine, as there are some phenomena which seem to indicate the not improbable co-existence of *amyloid* degeneration; and the lesion in the *supra-renal capsule* ought especially to be investigated in this direction.

The state of the skin, which has been termed "bronzing," strongly resembles the colour of a mulatto, or of a bronzed statue from which the gloss has been rubbed off. It has been examined microscopically by Dr. Wilks, Mr. Tuffen West, and M. Robin, of Paris. The sections show a layer of very distinct pigment-granules in the *rete mucosum*, limited to that structure, and exactly resembling that of a negro. The pigment is deposited in granules, but in some instances coloured cells are visible.

Before detailing the symptoms observed in the remarkable cachexia from which these lesions result, it is necessary again to draw attention to our very incomplete knowledge regarding many points in its pathology, and especially regarding the state of the blood, and the morbid tendencies of the relatives and progenitors of the patient. While the apparent connection of bronzing of the skin with lesions in the *supra-renal capsules* appears to be evident, our information is of the most deficient kind regarding the chronological sequence of the various symptoms which indicate the establishment of the constitutional disease.

Morbid Anatomy.—The special disease of the capsules in Addison's disease is described by Dr. Wilks as being of one form only—namely, that which was seen in the earlier cases described by Addison, and which he simply styled as "scrofulous." Judging from the material alone, it would by difficult to form an opinion of its nature, seeing that a degenerating inflammatory substance would produce a very similar appearance to a so-called scrofulous one. It is only in exceptional cases that any well-marked tubercular deposit has been found in other viscera. "When the disease is recent, the organ is somewhat enlarged, and changed into a material which is semi-translucent, of a grey colour, softish, homogeneous, and which, when examined microscopically, is found to be without structure, or sometimes slightly fibrillated, or containing a few abortive nuclei or cells. This lardaceous kind of material is the first deposited, and resembles what is often seen in the early stages of scrofulous enlargement of the lymphatic glands; subsequently it undergoes a decay or degeneration, as in these glands, and changes into an opaque yellowish substance; and thus the two materials are constantly found associated. At a later period, as in a scrofulous gland, this may soften into a putty-like matter, " or it may dry up, leaving the mineral part as a chalky deposit, scattered through the organs. These, then, are the changes—first, the deposition of a translucent, softish, homogeneous substance; subsequently the degeneration of this into a yellowish-white opaque matter; and afterwards a softening into a so-called abscess, or drying up into a chalky mass. Occasionally, also, some fibrous tissue may be found round the organs, being the product of an inflammation which has united them to the kidney, liver, and adjacent parts" (WILKS). Some years are necessary for the production of these changes.

Symptoms.—As in the forms of anæmia, already described, so in this disease the patient has considerable difficulty in assigning the number of weeks, or even months, that may have elapsed since he first experienced indications of failing health and strength; and the rapidity with which the local lesions seem to develop themselves varies in different cases—a few weeks being sometimes sufficient to break up the powers of the constitution, or even to destroy life. Dr. Addison believed that this event is the more speedy in proportion to the rapid and extensive destruction of the *supra-renal bodies.* The important features of the disease, as set forth by Dr. Addison, are,—a progressive feebleness of the patient, without any apparent or known cause (*asthenia*), anæmia, general languor and debility, remarkable feebleness of the heart's action, irritability and weakness of the stomach, and a peculiar change of colour of the skin. In most of the cases the early sequence of symptoms appears to have been gradual and almost imperceptible indications of failing health and strength, consisting chiefly of languor and weakness, and indisposition to either bodily or mental exertion, the appetite being impaired or entirely lost, the white of the eyes becoming pearly, the pulse small and feeble, perhaps somewhat large, but excessively soft and compressible. The body wastes, without presenting extreme emaciation or the dry and shrivelled skin usually associated with protracted malignant disease. Slight pain or uneasiness is from time to time referred to the region of the stomach, and there is occasionally actual vomiting. With every sign of feeble circulation, anæmia, and general prostration, "neither the most diligent inquiry nor the most careful physical examination throws the slightest gleam of light upon the precise nature of the patient's malady; nor do we succeed in fixing upon any special lesion as the cause of this gradual and extraordinary constitutional change."

"With a more or less manifestation of the symptoms already enumerated," writes the same distinguished physician, "we discover a most remarkable, and, as far as I know, characteristic discoloration taking place in the skin." It pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis, and scrotum, in the flexures of the axilla, and round the navel. It presents a dingy or smoky appearance, of various tints, or shades of deep amber or chestnut brown: and in one instance the skin was so universally and deeply darkened as to resemble a mulatto. This distribution of pigment is not confined to the skin, but is also visible in the mucous tracts, as well as in some other structures. An interesting case occurred in the practice of Professor Biermer, of Berne (November, 1861), in a weakly girl, eighteen years of age. After continued illness of a tubercular kind, the dark colour of the skin commenced, and increased in intensity with the decrease in the weight of the body, till death followed. The eyes of this patient happened to be examined with the ophthalmoscope, although vision was not disturbed. A bluish-black colour was everywhere visible through the sclerotic, and a peculiar pigment existed in the interior of the eye. In a plane of a uniform red-brown colour there were bluish-black spots, corresponding to the intermediate spaces of the *vasa-vorticosa,* and from which the retina was distinguished by its delicate white appearance. The disease thus originally described by Addison may be associated, like "*morbus Brightii,*" with the expression of several distinct local lesions. The discoloration consists of stains in the lining of the cheeks, and a decidedly blackish tinge of the mucous membrane of the lower lips, as if after eating mulberries. Dark areolæ become developed beneath the orbits, much marked towards the middle line of the face. In one case under Dr. Barlow, loss of consciousness and what are termed "fainting fits" were the earliest symptoms noticed (*Med. Times,* January 24, 1857).

"This singular discoloration usually increases with the advance of the disease; the anæmia, languor, failure of appetite, and feebleness of the heart become aggravated; a darkish streak usually appears upon the commissure of the lips; the body wastes, but without the extreme emaciation and dry harsh condition of the surface so commonly observed in ordinary malignant diseases; the pulse becomes smaller and weaker, and, without any special complaint of pain or uneasiness, the patient at length gradually sinks and expires. In one case, which may be said to have been acute in its development as well as rapid in its course, and in which both capsules were found universally diseased after death, the mottled or checkered discoloration was very manifest, the anæmic condition strongly marked, and the sickness and vomiting urgent; but the pulse, instead of being small and feeble as usual, was large, soft, extremely compressible, and jerking on the slightest exertion or emotion, and the patient speedily died" (ADDISON).

In the volume of *Guy's Hospital Reports* for 1862, Dr. Wilks has given a most able, interesting, and impartial account of the progress of our knowlege regarding this disease since the time Dr. Addison wrote. His own observations entirely uphold the argument which Dr. Addison attempted to develop; and the cases brought forward by him in that report appear to substantiate in a great measure the original facts on which Dr. Addison's history was based. Nevertheless, Addison's views have by no means received the support of the profession at large. Dr. Wilks thinks that Dr. Addison in some measure contributed to this scepticism, by including among its original cases some which did not present the true features of the disease; and the great merit of Dr. Wilks's paper is, that it more clearly defines and renders precise the pathological characters of the disease which Dr. Addison desired to describe. He believed, at the time he published his work, that *any disease* which affected the integrity of the *supra-renal capsules* would be attended by the remarkable phenomena originally described by him. This was an error: for all subsequent observations have shown that no recorded instance of the affection has been connected with cancer, or with any other kind of disease of the organ, than that found in the genuine cases of the disease which he first described, and which constitutes the true form of the malady, as Dr. Wilks has demonstrated.

Treatment.—If the disease be recognized in its earliest stages, its progress may to some extent be delayed. The asthenia, the depression, the evidence of local irritation about the supra-renal capsules, and the pathology of the disease generally, point to the necessity for tonic treatment and nutritive diet, the avoidance of all causes of depression, and the benefit of rest in bed, and of such medicinal agents as may relieve the vomiting. Glycerine in two drachm doses, combined with fifteen or twenty minims of the spirit of chloroform, and of the tincture of the sesquichloride of iron, have been of service (E. H. GREENHOW). This may be varied by the substitution of twenty to thirty minims of the syrup of phosphates of iron, quinine, and strychnia, in place of the sesquichloride of iron. The greatest caution is necessary in using purgative remedies, as fatal collapse is apt to follow cathartic medicine.

CHAPTER XIX.

DISEASES OF THE RESPIRATORY SYSTEM.

The acute inflammatory diseases of the respiratory system are more or less distinctly defined according to the tissue which they implicate and the symptoms to which they give rise. The three structures which mainly take part in the constitution of the lung substance being—(1.) The *bronchial tubes*, terminating in (2.) The *pulmonary air-cells, vesicular structure, proper substance*, or *parenchyma of the lung;* and (3.) *The membrane covering this parenchymatous part*, forming a portion of that serous sac interposed between the lungs and the walls of the thorax. That portion of the serous sac which immediately invests the lungs is known by the name of the *pulmonary pleura*, while that which is applied against and invests the parietes of the thorax is known as the *parietal* or *costal pleura*.

One or more of those pulmonary structures may be associated in the processes and results of inflammation. Thus the bronchial membrane may be inflamed, when the disease is termed *bronchitis;* or the substance of the lung may be inflamed, the disease being then called *pneumonia;* or the pleura may be inflamed, a condition which is described as *pleuritis*. It is rare, however, in practice to find that these morbid states are so completely isolated. More frequently, for instance, with *pneumonia*, or inflammation of the substance of the lung, there is associated more or less inflammation of the air-tubes (*bronchitis*), on the one hand, constituting *broncho-pneumonia;* or there co-exists inflammation of the investing pleura (*pleuritis*), constituting, on the other hand, *pleuro-pneumonia*. The physician can now distinguish each of these elementary conditions by definite symptoms; and by observing the combination of physical phenomena, their association with general symptoms, and the sequence of their occurrence, he is able to determine how far any given pneumonic affection involves one or more of the structures which compose the lung; and to direct the treatment of the case accordingly.

It is therefore necessary to describe, in the following sections, the phenomena of inflammation in each of these structures in detail; although the College of Physicians has not recognized in their nomenclature the combined lesions referred to as distinct diseases (see vol. i., p. 316).

SECTION I.—DISEASES OF THE RESPIRATORY SYSTEM NOT STRICTLY LOCAL.

HAY ASTHMA.

LATIN EQ., *Asthma ex fœnisicio;* FRENCH EQ., *Fièvre de foin;* GERMAN EQ., *Heu-Asthma;* ITALIAN EQ., *Asma dei mietitori.*

Definition.—*A variety of asthma or catarrh occurring generally during the summer months, especially during the inflorescence of the hay crop, or during the drying and conversion of the newly-mown grass into hay in May and June.*

Pathology.—In connection with the development of this affection there is usually a predisposition; and the exciting cause, some palpably minute emanation from the inflorescence of flowering plants, like the grasses, or from flowers of other plants. The exciting cause seems to be in the atmosphere, as the inhalation of minute particles, such as the powder of *ipecacuanha* will produce the affection; so also will fine *dust,* the composition of which is unknown, or at least is not known to contain anything so specific as *ipecacuanha.* The *dust* of grain or *flour* has been known to produce the same effect; and at one time mere effluvia or odours were believed so to affect the nervous system as to cause the occurrence of asthmatic fits. The odours of *mint,* of the *rose,* of *various flowers,* and other strong perfumes, have produced difficulty of breathing, with dry cough, as in hay asthma.

The probability is that the mere mechanical influence of minute particles floating in the air may be sufficient in some to cause this disease; on the other hand, the nature of the particles may be *specific,* as in the case of *ipecacuanha,* or inflorescence particles of certain grasses, or of flowers cut down with the grass in the progress of haymaking.

Symptoms.—The complaint is often very severe, involving the whole of the air-passages in an acute bronchitic attack, with much redness of the mucous membrane of the nose and eyelids.

Treatment.—If the pathology of this disease be accepted, which regards it as the result of irritation (specific or mechanical) from fine particles of matter (indefinite dust or specific powder) floating in the air, the use of a respirator of fine cotton, as shown in Professor Tyndal's interesting expositions regarding dust and disease, would absolutely prevent the disease, and ought to be tried by those who suffer every year about the months of May and June.

CROUP.

LATIN EQ., *Angina trachealis;* FRENCH EQ., *Croup;* GERMAN EQ., *Croup*
—Syn., *Häutige Bräune;* ITALIAN EQ., *Laringitide membranacea.*

Definition.—*A "non-infectious inflammation of the mucus of the trachea, occurring in children, differing from other inflammations in like tissue in the presence of plastic exudation"* (BARCLAY); *or it is a disease, accompanied by the exudation of a fibrino-albuminous material, which rapidly coagulates upon the mucous membrane of the epiglottis, glottis, larynx, or trachea, and sometimes over all of these parts; indicated by accelerated, difficult, wheezing, or shrill respiration; short, dry, constant, barking cough; voice altered by hoarseness, with spasm of the interior laryngeal muscles, and pain and constriction above the sternum; frequently followed, towards the close of the disease, by expectoration of a membranous albuminous substance, or even of a cylindrical cast of some portion of the breathing tube. The disease occurs in children, and may terminate fatally either in suffocation or exhaustion of the vital powers.*

Pathology and History.—It has often excited much surprise that a disease so distinctly marked in its symptoms should not have been accurately described before the middle of the eighteenth century, when Dr. Francis Home published a treatise on the *suffocatio stridula* or *croup,* in 1765, as it was observed in Leith, Musselburgh, and the vicinity of Edinburgh. It has been described under the name of *cynanche trachealis;* and Dr. Farr proposed for it the name of "*trachealia.*"

Before the time of Dr. Home, however, there is reason to believe that the disease was confounded with other affections of the throat and breast, resulting simply from exposure to cold. It was certainly also described and distinguished by Martin Shisi, in 1749, at Cremona, and by Starr, of Liskeard, in Cornwall, in the same year (*Phil. Trans.,* 1750). Many physicians have described the disease since that time, and none with more minuteness than Dr. Cheyne, of Leith, who observed it for several years, and illustrated its pathology by careful dissections.

The most remarkable pathological phenomena of croup are to be observed in the exudative process which attends the inflammation in the windpipe, and the formation of a false membrane, almost peculiar to children, but sometimes seen in adults. In them the disease derives all its importance from the tendency of the inflammation to attach itself to the opening of the glottis. In childhood the *trachea* is the chief seat of the inflammation; and when the larynx and the fauces are involved, they are so secondarily, and to a less degree. The croupous exudation is a *fibrino-albuminous* one, which rapidly coagulates when it is thrown out upon the free surface of a mucous membrane, involving in its lesion the epithelium only, so that when the croup-membrane is detached, the epithelium is quickly reproduced. No loss of substance occurs in the mucous membrane itself, and no scar remains after the membrane is removed or disappears. On the other hand, as Niemeyer points out, the diphtheritic process is also characterized by the production of a similar *fibrino-albuminous* and rapidly coagulable exudation; but differs from croup in the exudation forming not merely *upon* the surface of the mucous membrane, but *within* its substance. It infiltrates the mucous and sub-mucous tissue, and this interstitial exudation, as well as the swollen elements of tissue, exerts a pressure upon the blood-vessels which results in sloughing (diphtheritic) of a portion of the inflamed mucous membrane. A diphtheritic eschar is the result, and on its separation there is loss of substance and a consequent cicatrix.

Every now and then discussions arise as to whether or not croup exists as distinct from diphtheria; and the essential duality of the two diseases has of late been again in dispute. Any one who has seen much of croup in children can have no difficulty in recognizing it as a disease very different from diphtheria in its attack, its course, and its results. A transition from croup to diphtheria is, however, not unfrequent, as Niemeyer also states, when the malady forms part of, or occurs during the course of such acute infectious diseases as *measles, small-pox, typhus, scarlet-fever,* or during an epidemic of diphtheria.

Microscopically, the membrane of croup consists of amorphous or finely fibrilated fibrin, in which numerous young cells have been entangled during the process of its excretion (NIEMEYER).

When death takes place after an illness of four or five days, the windpipe in croup is found to be lined with a white or grey substance. The membranes thus formed vary much in thickness and consistency. Some are so thin that the mucous membrane is readily seen through them, while others are many lines in thickness, exceeding even that of the mucous membrane itself, and consequently opaque. With respect to their consistency, some are so little coherent that they are almost diffluent, while others can be detached for a considerable extent without rupturing. The false membrane, though occasionally only partial, yet more commonly embraces the entire circumference of the trachea, forming a complete hollow cylinder adapted to the walls of the tube. The membrane is in most instances limited by the larynx above, but in some cases it extends down the trachea to the bifurcation, while in a very few cases it reaches even to the minutest branches of the bronchi. M. Hussenot says, of 120 cases he examined in 1778, it did not extend beyond the larynx, while in 42 cases it invaded the trachea or bronchi. The membrane thus formed is in a few instances removed by the cough; but more generally it adheres with so great tenacity that it can only be detached by a thinner and more serous secretion taking place from the mucous membrane beneath it, which loosens and displaces it.

In childhood it is almost exclusively a disease of the trachea, and sometimes of the larynx, and rarely affects the alveoli of the lungs. On the other hand, croupous pneumonia is a common disease of adults, in whom primary croup of the trachea and larynx scarcely ever occurs.

The extent of the exudation, as indicated by the surface covered, is perhaps the most interesting and practically useful part of the pathology of the disease. The place first and most particularly affected is the upper part of the *trachea,* about an inch below the *glottis.* In that part patients complain of a dull pain. External swelling has been observed there; and the morbid membrane is found spreading thence downwards. The back part of the trachea, where there are no cartilages, seems to be its first and principal seat (HOME). According to Guersent, false membrane is never entirely absent from the larynx. Sometimes it is confined to the glottis, and sometimes lines the whole interior of the larynx, including the ventricles; and not unfrequently it extends throughout the trachea, and, for a greater or less distance, into the bronchial tubes. Dr. Wood instances a case in which he saw the false membrane line the upper portion of the bronchia, the whole trachea and larynx, and the pharynx as low down as the œsophagus. More frequently, he says, the exudation is in the form of patches, or long narrow ribbons, and occasionally, in the earlier stages, it has a granular aspect, with the red mucous membrane appearing in the intervals of the imperfectly connected patches. According to Cheyne, in none of the cases recorded by him was membranous exudation observed in the laryngeal mucous membrane; and if the inflammation extended to this part, it was only slight, and its effects were seen in a little puriform fluid in the membrane of the cricoid or thyroid cartilages. Some state that it is essential to the constitution of croup that the larynx should be more or less involved in inflammation, or high vascular irritation, accompanied with spasms of the internal muscles of the larynx (WOOD). Others say that the inflammation in croup is truly tracheal, and even bronchial (CRAIGIE, COPLAND). In the more acutely inflammatory form it may extend to the larynx and epiglottis, in some cases; in others, to the first ramifications of the bronchi; and sometimes in both directions (COPLAND).

Dr. Copland, who has paid particular attention to the pathology of croup, states the following as general inferences from his observations:—"(*a.*) That the mucous membrane itself is the seat of the inflammation of croup; and that its vessels exude the albuminous or characteristic discharge, which, from its plasticity and the effects of temperature and the continued passage of air over it, becomes concreted into a false membrane;—(*b.*) That the occasional appearance of blood-vessels in it arises from the presence of red globules in the fluid when first exuded from the inflamed vessels, as may be ascertained by the administration, upon the approach of the symptoms, of a powerful emetic, which will bring away this fluid before it has concreted into a membrane; these globules generally attracting each other, and appearing like blood-vessels, as the albuminous matter coagulates on the inflamed surface;—(*c.*) That the membranous substance is detached, in the advanced stages of the disease, by the secretion from the excited mucous follicles of a more fluid and a less coagulable matter, which is poured out between it and the mucous coat; and, as this secretion of the mucous *cryptæ* becomes more and more copious, the albuminous membrane is the more fully separated, and ultimately excreted if the vital powers of the respiratory organ and of the system be sufficient to accomplish it;—(*d.*) That sub-acute or slight inflammatory action may be inferred as having existed, in connection with an increased proportion of fibro-albuminous matter in the blood, whenever we find the croupal productions in the air-passages; but that these are not the only morbid conditions constituting the disease;—(*e.*) That in conjunction with the foregoing—sometimes only with the former of these in a slight degree—there is always present, chiefly in the developed and advanced stages, much spasmodic action of the muscles of the larynx, and of the transverse fibres of the membranous part of the trachea, which, whilst it tends to loosen the attachment of the false membrane, diminishes, or momentarily shuts, the canal (of the larynx) through which the air presses into the lungs;—(*f.*) That inflammatory action may exist in the trachea, and the exudation of albuminous matter may be going on for a considerable time before they are suspected —the accession of the spasmodic symptoms being often the first intimation of the disease; and these, with the effects of the pre-existing inflammation, give rise to the phenomena characterizing the sudden seizure;—(*g.*) That the modifications of croup may be referred to the varying degree and activity of the inflammatory action, the quantity, the fluidity, or plasticity of the exuded matter, the severity of spasmodic action, and to the predominance of either of these over the others in particular cases, owing to the habit of body, temperament, and treatment of the patient, &c.;—(*h.*) That the muco-purulent secretion, which often accompanies or follows the detachment and discharge of the concrete or membranous matters, is the product of the consecutively excited and slightly inflamed state of the mucous follicles, the secretion of which acts so beneficially in detaching the false membrane;—(*i.*) That a fatal issue is not caused merely by the quantity of the croupal productions accumulated in the larynx and trachea, but by the spasm, and the necessary results of interrupted respiration and circulation through the lungs;—(*k.*) That the partial detachment of fragments of membrane, particularly when they become entangled in the larynx, may excite severe, dangerous, or even fatal spasm of this part, according to its intensity relatively to the vital powers of the patient; and that this occurrence is most to be apprehended in the complicated states of the malady where the inflammatory action, with its characteristic exudation, spreads from the fauces and pharynx to the larynx and trachea—the larynx being often chiefly affected in such cases—and from its irritability and conformation giving rise to a more spasmodic and dangerous form of the disease;—(*l.*) That the danger attending the complications of croup is to be ascribed not only to this circumstance, but also to the depression of vital power, and the characteristic state of fever accompanying most of them, particularly in their advanced stages;—(*m.*) That irritation from partially detached membranous exudations in the pharynx, or in the vicinity of the larynx or epiglottis, may produce croupal symptoms in weak, exhausted, or nervous children, without the larynx or trachea being themselves materially diseased; and that even the sympathetic irritation of teething may occasion the spasmodic form of croup, without much inflammatory irritation of the air-passages, particularly when the *prima via* is disordered, and the membranes about the base of the brain are in an excited state;—(*n.*) That the predominance in particular cases of some one of the pathological states noticed above (*g.*) as constituting the disease, and giving rise to the various modifications it presents, from the most inflammatory to the most spasmodic, may be manifested in the same case, at different stages of the malady, particularly in its simple forms, and in the relapses which may subsequently take place; the inflammatory character predominating in the early stages, and either the mucous or the spasmodic, or an association of both, in the subsequent periods;—(*o.*) That the relapses, which so frequently occur after intervals of various duration, and which sometimes amount to seven or eight, or are even still more numerous, may each present different states or forms of the disease from the others; the first attack being generally the most inflammatory and severe, and the relapses of a slighter and more spasmodic kind; but in some cases this order is not observed, the second or third, or some subsequent seizure, being more severe than the rest, or even fatal, either from the inflammation and extent of exudation, or from the intensity and persistence of the spasmodic symptoms,—most frequently from this latter circumstance. The above inferences, however minute or trite they may seem, should not be overlooked, as they furnish the safest and most successful indications of cure, and are the beacons by which we are to be guided in the treatment of the disease."

The name of *croup*, by which this disease has hitherto been known in this country, is of Scottish origin. Cullen's *cynanche trachealis*, and the more modern *tracheitis*, are objectionable terms, because they lead to false notions of the pathology of the disease. The "choak," "stuffing," "rising of the lights," and "hives," are all designations by which the disease has been described, and some of them are still names in vogue amongst the common people of the country.

The disease is almost peculiar to infancy and childhood; and there are two forms which can generally easily be distinguished from each other, but which are often confounded. One form is very manageable, the other very fatal. In the former variety the mucous membrane chiefly secretes mucus, pus, or muco-purulent fluid. In the more dangerous form an albuminous, fibrinous, or mucinous exudation grows upon the inner surface of the air-passages, constituting the false membranes already described. The first form seems to be the one common in America, of which not more than *one* in *fifty* dies. The latter is the more common European form, of which the deaths used to be *four* out of *five*, and still are about *a half*. About one child in twelve deaths of children dies from this disease; and the ratio borne by croup to 1,000 deaths from all causes, in 1854, was as 9·249. The disposition to the disease is least during the period of suckling; and again, after the second dentition, cases are rare. The period of life between the second and seventh years furnish the greatest number of cases.

Symptoms and Course.—The mildest form of croup differs from an ordinary catarrh only in the addition of spasmodic symptoms; but this form may run into the more severe form, so that it is not possible to determine, in the first instance, which form the disease may ultimately assume.

The catarrhal croup of Dr. Wood embraces the spasmodic as well as the catarrhal croup of Dr. Copland. Spasmodic action of the laryngeal muscles is, however, common to both, and is characteristic; but the inflammation and exudation is not in general more severe than that which attends a common catarrh.

The disease may be ushered in by sore throat, by catarrhal symptoms, or by a short dry cough; or it may occur *per se*, and without the general health being sensibly impaired. In either case the attack commonly takes place during the night, the sleep of the child, which was perhaps more or less agitated, being interrupted by fits of *hoarse* coughing. These become more frequent, the respiration more difficult, and marked by a peculiar wheezing, which has been described as like the sound of an inspiration forcibly made with a piece of muslin before the mouth, or like to the sound of air passing through a brazen tube. The little patient also feels a sense of restriction about the throat, as shown by carrying the hand often to it, and grasping the larynx. After the paroxysm has lasted some hours, there is an interval of ease, which perhaps lasts for some hours.

By the end of the second or third day, sometimes sooner, the tongue becomes white, the heat of the body increased, the pulse frequent, the face flushed, and the countenance distressed. From this point the disease now rapidly advances, the croupy sound attains its height, and Dr. Home describes it as "*vox instar cantus galli;*" others have compared it to the noise which a fowl makes when caught in the hand; while the child often puts its fingers into its mouth, as if to pull away something which obstructs the passage.

As the disease draws towards a close the paroxysms become more frequent, the cough more severe and with less sound, the pulse more rapid, suffocation more imminent, and the extremities cold and livid. The final close of the disease is often by convulsions, sometimes almost tetanic; and Dr. Ferrier once was present when the struggle was so violent that after death the corpse, in a great measure, rested on the occiput and on the heels.

Often, however, the symptoms are much more moderate; although it not unfrequently happens that symptoms of the severer form come on, indicated by a huskiness of the voice, till no sound can be heard above a whisper, by a muffled cough, and a wheezing noise which attends the inspirations. It is seldom that children expectorate; but in happier cases than the above, mucus, tinged perhaps with blood, is coughed up, and later, perchance, the false membrane is detached and thrown up, and the patient recovers.

The croup which has been described is of the most acute kind; but in many cases its course is much more chronic, the symptoms generally milder, and the intervals of ease longer and more complete, during which the breath is free, the child cheerful, and the appetite good. In the course of a few days, however, a violent paroxysm seizes the child, and destroys him with every appearance of one strangled.

According to Barth, on the stethoscope being applied to the larynx, we hear a sort of "*tremblotement*," as if a moveable membrane was agitated by the air; and he considers this phenomenon as an unerring evidence of the existence of a false membrane in the larynx. Laryngitis in the adult is marked by the same difficulty of breathing, the same lividity of countenance, the same constriction of the throat, the same paroxysmal attack, and by the same exemption from any severe constitutional affection. The voice, however, instead of being sharp and shrill, is generally deep and hoarse, although sometimes altogether lost—differences depending perhaps on the greater size of the glottis, and on the fact of the parts being the seat of ulceration rather than of the effusion of lymph. At length the patient is cut off in one of the paroxysms. The duration of this disease, when acute, is short. The celebrated Dr. Pitcairn died on the fourth day from the first attack; and Sir John Hay, Physician to the Forces, died within the same period. More commonly, perhaps, the disease passes into a chronic state, when the patient may survive many weeks, or even months. Several cases are on record of croup having terminated in twenty-four hours; more frequently, however, the child lives to the third or fourth day, and in chronic cases much longer. From one day to one or two weeks may be given as the variable periods of the duration of this disease.

Diagnosis.—It is generally between croup and the following diseases—namely, the different forms of sore throat, as in *scarlet fever* and *measles, diphtheria, bronchitis, chronic laryngeal and tracheal inflammation,* and *hooping-cough;* and the differential symptoms of each of these from croup must be studied by comparing the definitions, symptoms, and course of each of these diseases, as well as the epidemic constitution as regards *scarlet fever, measles, diphtheria,* and *hooping-cough.* In diphtheria there is a specific fever, and the lesion spreads from above downwards, or may commence in the larynx. In croup the lesion commences in the trachea, and spreads upwards. Hence the two diseases have often been and are apt to be confounded together. Then, as to acute laryngitis, that is a disease of adult life, and croup is a disease of childhood.

Modes of Propagation.—Croup is said to be more frequent in cold and moist climates than in those which are warmer. It is also much more severe in Europe than in America; and its existence and progress is considerably influenced by changes of season, weather, and temperature. It is prevalent in Switzerland and Savoy, in the eastern counties of England and Scotland, the north-west countries of Europe, and in the northern parts of America. While the annals of medicine are rich in descriptions of epidemic and endemic croup, opinions are very much divided as to the nature of the epidemic influence, and whether or not the disease is contagious or infectious.

Age has, perhaps, the greatest influence in predisposing to the disease, and, while rare in adults, it is seldom seen in early infancy. It is most prevalent between the *first* and *seventh* years of life. According to the experience of Dr. Wood, the disease appears to run in families; and vigorous fleshy children, with rosy complexions, are frequently those who suffer most.

Prognosis—"Is never better than doubtful." It is to be determined from the violence of the local symptoms, and the frequency of the paroxysms, rather than from the constitutional symptoms. Children, however, seized with croup are said to recover in a smaller proportion in this country than in America. Death tends to occur by *apnœa.*

Treatment.—*Every case of croup demands the most active, efficient, and energetic treatment.* When the croup in children extends to the larnyx, its course is so rapid and so fatal that the measures for its suppression must be early. Bleeding, and especially local bleeding, should be employed, and in most cases to a considerable extent (an ounce of blood for every year of age);—and two to twelve leeches, according to the age of the patient, should be applied over the larynx. After these have fallen off, the bleeding should be encouraged by the application of a lintseed poultice to the throat. This first bleeding often gives great relief, and sometimes averts the disease; but if not, the leeches, after a few hours, may be repeated. As soon as some relief is obtained, a blister should be applied along the lateral aspect of the neck on each side, and not over the trachea; and after that is removed, the part should be dressed with strong mercurial ointment. In addition to bleeding and blistering, many practitioners prescribe *emetics;* first, because their emetic effects, and the large evacuatious they produce, favour the resolution of the inflammation; and again, because the effort of vomiting may be the means of detaching and of expelling the false membrane, should it have formed. If relief does not ensue on the action of the emetic, Dr. Cheyne recommends *two, three,* or *four* grains of calomel, with *two* or *three* grains of James's powder, to be given at short intervals every two or three hours; and a dose of castor oil is to be given occasionally till the full effect of the calomel as a purgative is obtained. Green fæcal stools, like chopped spinach, are characteristic of this result.

Bleeding, blistering, and mercury, although the rule of treatment in idiopathic infantine croup, are, for the most part, entirely inefficient in those cases in which the affection begins in the fauces, as in the case of many epidemics, and especially after scarlatina. In these cases the best treatment, if the false membrane be not already formed, is to relieve the throat by the application of a few leeches, as in scarlet fever, and then to support the little patient with a moderate quantity of wine diluted with water. If the false membrane has formed, perhaps an emetic affords the only chance of relieving the patient; and, indeed, so soon as croupy cough and dyspnœa occur, an emetic of *ipecacuanha* with *tartar emetic* ought at once to be given, in doses suited to age. Four to six grains of ipecacuanha, combined with a quarter or a third of a grain of tartar emetic, will be found sufficient for a child of two or three years of age. The action of the emetic may be aided, with benefit, by a warm bath of 98° to 100° Fahr. in temperature. If it becomes obvious that the exudation has assumed the form of a membrane, especially if indicated by a diphtheritic coating over the fauces, a solution of the nitrate of silver, varying in strength from forty to one hundred and twenty grains to the fluid ounce of distilled water, should be applied to as much of the fauces and larynx as can be reached. A sponge on the end of a piece of whalebone, as sold ready made by the instrument makers, should be loaded with the weaker solution, and squeezed against the *rima glottidis* two or three times a day. Bleeding has no effect in removing or modifying the false membrane; but the system must be brought as speedily as possible under the influence of mercury. Mercury appears a powerful resource in these cases; and, introduced either internally or by inunction, so as to affect the mouth, but without inducing salivation, uniformly gives relief as soon as the constitutional affection is established. However, the amelioration is too often transitory.

Expectorant medicines should be given with the mercurials, and be continued after them. *Ipecacuanha* and *seneka* have the most efficient influence over the mucous membrane. Five-grain doses of *iodide o potassium* every two hours, and of *chlorate of potassa* have been used with benefit; and the use of a vapour bath from 75° to 80° Fahr. is not to be neglected (BUDD).

The medical treatment of croup is so frequently unsuccessful that tracheotomy has often been had recourse to as the means of prolonging life, and, consequently, as affording an additional chance of the patient's recovery. Guersent has performed this operation repeatedly at the Hôpital des Enfans, but almost always without success. On the other hand, M. Trousseau states that he has saved one-third of his patients by its means; and of twenty cases Bretonneau saved six. Perhaps the experience of the profession, generally, is equally discordant on this point at this moment; for those who operate early contend they save some portion of their patients, while those who wait till a case is advanced, and beyond medical treatment, before they resort to this measure, for the most part lose all their patients. The evidence, however, is daily accumulating which shows that tracheotomy ought to be resorted to much oftener, as a remedy for croup, than it has hitherto been, and that *at a much earlier period in the disease,—not as a last resource, when death from asphyxia appears imminent, and after treatment of the most depressing kind.* That this is the secret of success in France and in this country is shown by the experience of able physicians and good surgeons, of whom the names of M. Trousseau, the late Mr. Jones, of Jersey, Mr. Henry Smith and Dr. Fuller, of London, the late Dr. Cruickshank, of Dalmellington, in Scotland, and Mr. Spence, Professor of Surgery in the University of Edinburgh, Dr. George Buchanan, in Glasgow, and Professor Roser, of Tübingen, may be stated as authorities by experience. In country districts the performance of tracheotomy in a case of croup is almost imperatively called for in the majority of cases, *if some symptoms of amelioration do not follow the steady use of bleeding, emetics, the warm bath, and calomel purgation, pursued for twelve or sixteen hours.* I know that, in a wild country district of Scotland, where croup was very common and fatal, the late Dr. Cruickshank saved eight out of eleven cases during two years. A valuable paper by Mr. Smith, in *The Medical Times and Gazette*, 26th January, 1856; another by the late Mr. Jones, of Jersey, in the 8th November of that year; and, lastly, a paper by Dr. Conway Evans, in the *Edinburgh Medical Journal* for January and May, 1860, go to support the same conclusion,—namely, that an earlier introduction of air, by the operation of tracheotomy, for croup, would not only give a larger per centage of recoveries in this country, but would place the operation in the same favourable light in which it is now regarded in Paris and other parts of France. Tracheotomy in croup is undoubtedly gaining ground; and it cannot be denied that children perish in the first instance almost always from suffocation. Tracheotomy is therefore indicated in croup (as in diphtheria) as soon as there are urgent symptoms of obstruction of the glottis. When the respiration is so impeded that the demand for oxygen is only satisfied by difficult forced respirations, dreadful anguish is depicted on the reddened countenance covered with sweat; there is extreme restlessness; the patient tosses from side to side, gets out of bed one minute and into it the next, clutching spasmodically at those around him, as if seeking everywhere for help. *This is the proper period for the operation of tracheotomy in croup,*—the time when success may be expected (ROSER). Should the operation be longer delayed, symptoms of asphyxia appear, overloading of the blood with carbon ensues, the face suddenly becomes blue, with fixed and staring eyes, convulsive exertions are made, and anxious struggles for breath follow the stage of suffocative agony. In some cases the symptoms of asphyxia come on more slowly, and are apt to make considerable progress before the danger is fully appreciated. This insidious form of asphyxia is denoted by symptoms of great weariness and weakness, restlessness, oppression, anxious startings out of short slumbers, loss of consciousness and of feeling, approaching stupor, the face pale, and tending to become œdematous. If tracheotomy is delayed till these symptoms become expressed, it may still enable the child to breathe more freely, and thereby may promote the chances of recovery; but the child has usually no longer power to resist the advancing bronchitis. The causes of death after the operation are mainly pneumonia, bronchitis, or the severity of the constitutional febrile state (*Syden. Society Year-Book,* 1863, p. 278). Abscesses in the anterior mediastinum, pleuro-pneumonia, and pericarditis have been found after death. The fatal result, therefore, seems in some cases to depend on a cause acting generally on the system, and which ultimately destroys the patient. This cause gives a difference of type to the disease in this country from what it has in France; but as this difference appears, in the first instance, to be aggravated by the obstruction to the passage of air, there is thus a still more powerful reason why the operation of tracheotomy ought to be resorted to early. Age also seems to influence success to a considerable extent. Under *two* years of age few cases recover; but between the ages of *six* and *twelve* nearly one-half are saved (CONWAY EVANS).

SECTION II.—AIDS TO THE DIAGNOSIS OF DISEASES OF THE THROAT AND LARYNX.

THE LARYNGOSCOPE.

For upwards of a century attempts, more or less successful, have been made to examine the lower part of the throat during life. In connection with the unsuccessful or only partially successful efforts may be mentioned the names of Levret (1743), Bozzini (1807), Cagniard de Latour (1825), Senn (1827), Babington (1829), Avery (1844), and Garcia (1854). It was not, however, until 1858 that Professor Czermak, of Pesth, brought to perfection and practical application the laryngoscope of Garcia, which Türck, of Vienna, had unsuccessfully endeavoured to employ a few months previously. In this country, the profession is largely indebted to Drs. Morell-Mackenzie, Walker, Johnson, Gibb, and Sieveking, and to Mr. Durham, for making known the practical application of the laryngoscope as well as for many valuable additions to the literature and the armamentaria of this branch of practice.

The following description of instruments used in laryngoscopy and the method of examining the larynx are taken from Morell-Mackenzie's work, *On the Use of the Laryngoscope,* third edition, published by Longmans:—

(1.) *The laryngoscope* is "an instrument for obtaining a view of the larynx during life. It consists of two parts; first, a small mirror called the *laryngeal mirror,* fixed to a long slender shank, which is introduced to the back of the throat; secondly, an apparatus for throwing a strong light (solar or artificial) on to the small mirror. For thus projecting the luminous rays a second (larger) mirror, which reflects the light from a lamp, or the solar rays, may be employed; or artificial rays may be concentrated by a lens directly on to the small mirror. When artificial light is employed, the illuminating mirror is slightly concave; when sunlight is used, its surface is plane."

(2.) The *laryngeal mirror** is of circular shape, and made of glass, backed with amalgam, set in a German-silver frame, and attached to a shank of the same metal, at an angle of 120°; the shank is fitted into a slender wooden or ivory handle. The mirrors, which are of three sizes—No. 2 being the most convenient for ordinary cases—are about one-twentieth of an inch in thickness; the shank is about four inches in length, and the handle about three inches in length.

(3.) The *reflector* is a circular mirror, about three inches and a half in diameter, with a small oval hole in its centre; it is fixed to the head-piece by a ball and socket joint. The mirror is by some attached to an elastic band, which encircles the head of the operator; by others fixed to a horizontal arm, which is connected with the body of the lamp. Dr. Mackenzie's method of attaching it to a spectacle-frame, from which the upper halves of the rims have been removed, is not only the most comfortable but the most convenient for adjustment and removal.

Light.—It is unnecessary to point out that for practical purposes the solar light is too inconstant to be of service in this country. It is therefore from artificial sources that light must be obtained. Any lamp that gives a bright steady light answers the purpose perfectly well. Many of the most valuable observations have been made with a common "moderator." An argand gas burner, constructed on the reading-lamp principle, with flexible tubing to connect it with an ordinary burner, is very convenient for practitioners who only require to use the instrument occasionally; and this lamp is equally convenient for microscopic or ophthalmoscopic purposes. For a fixture, Mackenzie's rack movement lamp (fig. 44), which ingeniously admits of both perpendicular and horizontal movements, is everything to be desired. In all cases a concentrating lens is required to intensify the light. In country practice, a sheet of white paper placed behind the light is a good substitute for a concentrator. For demonstration and class purposes, the oxyhydrogen light, as arranged at the Hospital for Diseases of the

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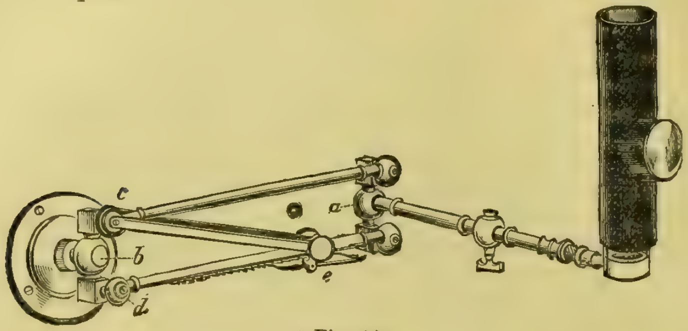
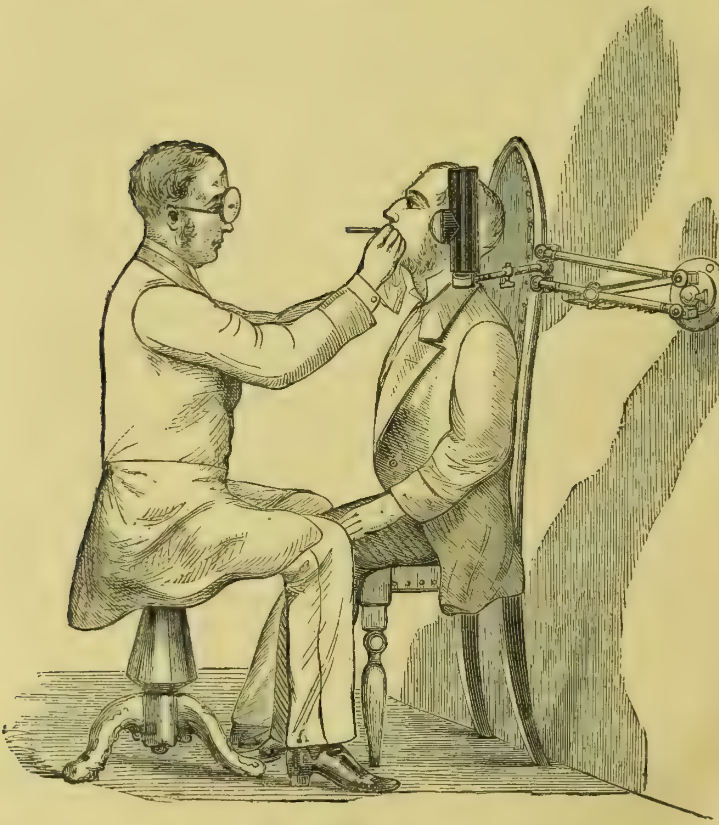
* Square mirrors are used in France, but they are not so convenient as round ones. Oblong mirrors are of service where the laryngoscope has to be used on patients affected with enlarged tonsils.

Throat, is most convenient. The apparatus is so contrived that as many as six people, in addition to the demonstrator, can simultaneously see the larynx of a patient.



* Fig. 44.



† Fig. 45.

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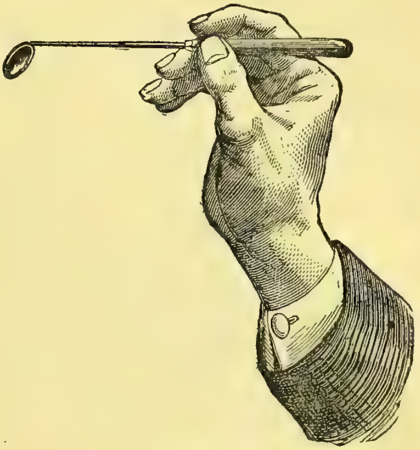
* The Rack-movement Lamp.—At *a* and *b* the horizontal movements can be effected; at *c* and *d* the vertical movement takes place. The gas passes only along the upper horizontal tube from *c*; in the lower tube is a rack which regulates the height of the lamp through the little handle, *e*. The chimney of the lamp is made of metal, a round hole being left where the lens fits in (after Dr. Morell-Mackenzie).

† View of practitioner examining patient. The patient is seen to be sitting up-

The *method of making an examination* is as follows:—The patient should sit upright, facing the observer, with his head inclined very slightly backwards. The observer's eyes should be about one foot distant from the patient's mouth, and a lamp burning with a strong clear light should be so arranged at the side of the patient so that the flame of the lamp is on a level with the patient's eyes.

The observer should now put on the spectacle-frame, with the reflector attached, and directing the patient to open his mouth widely, should endeavour to throw a disc of light on to the fauces, so that the centre of the disc corresponds with the base of the uvula. The patient should be directed to put out his tongue, and the observer should then hold the protruded organ gently but firmly between the finger and thumb of the left hand, previously enveloped in a small soft cloth or towel. Then, holding the mirror (previously warmed to prevent condensation of the moist expired air) like a pen in the right hand (fig. 46), it should be quickly introduced to the back of the throat, and kept as far as possible from the tongue, the face being directed downwards, The posterior surface of the mirror should rest on the uvula, so as to push that part rather upwards and backwards towards the posterior nares.


* Fig. 46.

The exact angle which the mirror should bear to the laryngeal aperture must depend on a number of circumstances, such as the degree of flexion backwards of the patient's head, the particular angle which the plane of the laryngeal aperture bears to the horizon in the case undergoing inspection, and on the position of the observer.

Undue faucial irritability may exist, and there may be other accidental physical difficulties, but in the majority of instances the difficulty is with the observer, not with the patient. For the purpose of investigating the action of the vocal cords the patient should be directed to inspire deeply, or to produce a vocal sound, as "ah," "eh," &c. Where the epiglottis hangs low, it is useful to make the patient laugh or strike a high or falsetto note. The beginner must remember that in examining the larynx the objects are reversed in the mirror, *not as regards right and left,* but with reference only to the antero-posterior direction. The part

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right, with his head thrown slightly backward against a high-backed chair. The rack-movement lamp is seen in use on the left side of the patient. The practitioner wears the large reflecting mirror in front of his right eye; with his left hand (not seen in wood-cut) enveloped in a small napkin, he holds out the patient's tongue; and, with his right hand, he introduces the laryngeal mirror (Dr. Morell-Mackenzie).

* The position of the hand and mirror, when the latter has been properly introduced for obtaining a view of the larynx (after Dr. Morell-Mackenzie).

which in reality is nearest to the observer, the anterior commissure of the vocal cords, becomes farthest in the image, and the posterior or inter-arytenoid commissure, which in reality is farthest from the observer, becomes nearest in the image (fig. 47).

As in the use of every other instrument, it is only the experience of practice that will enable the observer to overcome the various little difficulties that occur in this apparently simple method of examination.

The instrument is absolutely necessary, not only for precision of diagnosis, but for accuracy in applying local remedies to the inferior part of the pharynx, the larynx, and trachea, as well as to the posterior surface of the pillars of the fauces, and the posterior nares. The scientific investigation of the latter part, by means of mirrors, is called Rhinoscopy.

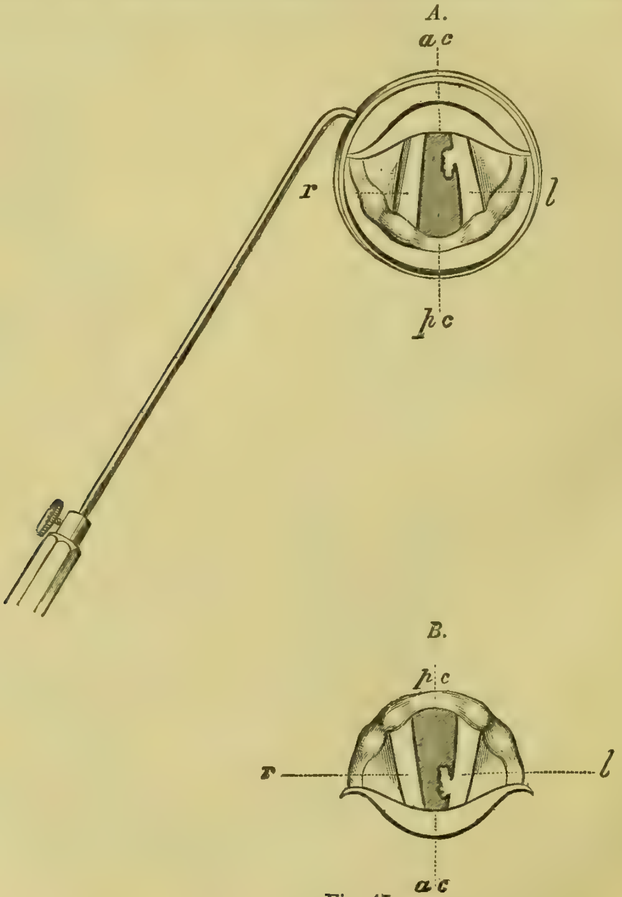

Fig. 47.[*]

The following series of woodcuts (fig. 48), A, B, C, D, E, and F, on the next page, are in illustration of appearances seen by the laryngoscope and rhinoscope:—

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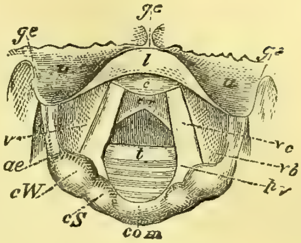
[*] Drawing showing the relation of parts in the larynx (*B*) and the laryngeal mirror (*A*). *a c*, Anterior commissure of the vocal cords; *p c*, Posterior commissure of the vocal cords; *r*, Right vocal cord; *l*, Left vocal cord, with a wart on it (after Dr. Morell-Mackenzie).

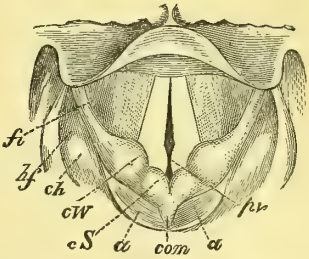
Description of Wood-Cut—Fig. 48.

A



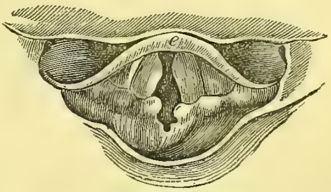
A. Laryngoscopic drawing, showing the vocal cords drawn widely apart, and the position of the various parts above and below the glottis, during quiet inspiration:—*ge*, Glosso-epiglottic folds; *u*, Upper surface of epiglottis; *l*, Lip of epiglottis; *c*, Cushion of epiglottis; *v*, Ventricle of larynx; *ae*, Ary-epiglottic fold; *c W*, Cartilage of Wrisberg; *cS*, Capitulum Santorini; *com*, Arytenoid commissure; *vc*, Vocal cord; *vb*, Ventricular band; *pv*, Processus vocalis; *cr*, Cricoid cartilage; *t*, Rings of trachea (after Dr. Morell-Mackenzie).

B



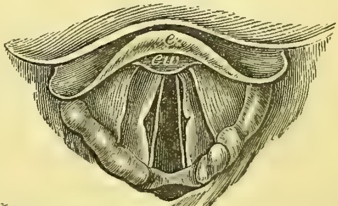
B. Laryngoscopic drawing, showing the approximation of the vocal cords, and the position of the various parts in the act of vocalization:—*fi*, Fossa innominata; *hf*, Hyoid fossa; *ch*, Cornu of hyoid bone; *c W*, Cartilage of Wrisberg; *cS*, Capitulum Santorini; *a*, Arytenoid cartilages; *com*, Arytenoid commissure; *pv*, Processus vocalis (after Dr. Morell-Mackenzie).

C



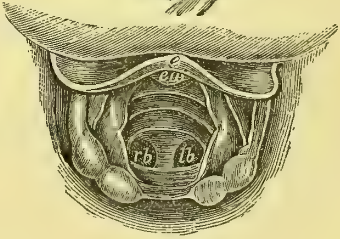
C. Constriction of the glottis, erroneously called *respiratory glottis*, the arytenoid processes converging, and causing the posterior section of the glottis to be almost triangular (after Elfinger and Czermack).

D



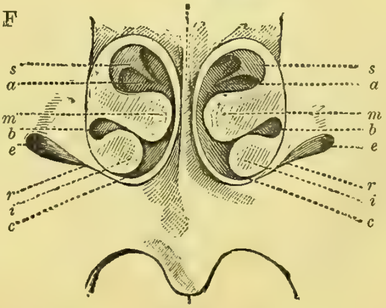
D. Complete relaxation of all the parts, as when the glottis under ordinary circumstances is open for breathing (after Elfinger and Czermack).

E



E. Bifurcation of trachea and commencement of bronchial tubes, seen on widely opening the glottis, as in deep inspiration, and straightening the trachea (after Elfinger and Czermack).

F



F. The Posterior Nares as seen in Rhinoscopy:—*sn*, Septum nasi; *s*, Superior turbinated bone; *m*, Middle turbinated bone; *i*, Inferior turbinated bone; *a*, Superior meatus; *b*, Middle meatus; *c*, Inferior meatus; *e*, Eustachian orifice; *r*, Ridge between the Eustachian opening and lower border of the nasal fossa; *u*, uvula (after Dr. Morell-Mackenzie).

Fig. 48.

SECTION III.—DISEASES OF THE LARYNX.

LARYNGITIS.

LATIN EQ., *Laryngitis;* FRENCH EQ., *Laryngite;* GERMAN EQ., *Entzündung des Kehlkopfs;* ITALIAN EQ., *Laringitide.*

Definition.—*Inflammation of the lining membrane of the larynx.*

Pathology.—Laryngitis may be (*a.*) Acute or œdematous; (*b.*) Subacute or mucous; or (*c.*) Chronic.

(a.) *Acute or Œdematous Laryngitis.*

This disease is essentially an inflammation of the mucous membrane, and sub-mucous tissue of the larynx, its danger being in proportion to the extent that the sub-mucous tissue is affected; in other words, the danger is proportionate to the amount of œdema.

Acute laryngitis not only often threatens the life of the patient, but there are few diseases which can kill more quickly. Death approaches by suffocation, and the glottis may be very rapidly closed by the œdematous inflammation. The risk, however, is not due to œdematous swelling alone, but also to the spasm of the glottis which the infiltration causes—partly by reflex action, partly by direct irritation of the adductor muscles of the vocal cords (MORELL-MACKENZIE).

The following are the modes in which the disease may terminate:—

(1.) By resolution—either spontaneous or the result of remedial measures. (2.) The acute symptoms having passed away, chronic congestion may remain. (3.) Death may suddenly take place from the combined effects of œdematous swelling and spasm of the glottis; less suddenly from the former cause acting alone, or slowly, and often preceded by delirium, from the effects of exhaustion and imperfectly aerated blood. (4.) Threatened suffocation may be averted by tracheotomy.

Symptoms.—The disease may commence as a slight catarrh, and may gradually take the form of acute inflammation, or it may from the first be ushered in by rigors, and rapidly followed by fever and elevation of temperature. Niemeyer, however, thinks that the premonitory and acute pyretic symptoms are often wanting, and observes that "acute catarrh of the larynx rarely begins with shivering; indeed, in most cases, when it does not spread from the bronchi, catarrhal fever is also absent throughout the whole course of the attack." Locally, a sense of uneasiness in the throat, generally referred to the *pomum Adami,* is soon followed by a feeling of constriction and strangulation.

The period at which the symptom of embarrassed respiration first appears, depends very much on the part of the larynx first affected; thus, if the epiglottis or ary-epiglottic folds are first attacked, the feeling of strangulation and difficulty of swallowing will be among the most early phenomena. If, on the other hand, the ventricular bands, or vocal cords, are the primary seat of the disease, loss of voice will precede the other symptoms. In all cases, modifications of the natural functions of the part are the most striking phenomena. Vocalization, cough, and respiration are all more or less modified. The voice is at first hoarse, but as the disease advances it becomes completely aphonic. The cough is at first clear and shrill, then harsh and croupy, and finally aphonic. In a well-marked case, the brassy tone peculiar to the disease terminates in a hissing noise, and begins similarly by a hissing inspiration in a muffled manner, because the lips of the glottis being thickened, irregular, and rough, cannot be sufficiently closed to begin a sharp sound (HYDE SALTER). As to the respiration, there is a peculiar noise, like a loud whisper, which accompanies both inspiration and expiration. Inspiration is from the first laborious and wheezing; afterwards it is very much lengthened and stridulous, and starts sharp from the conclusion of the previous expiration. Mucous râles are heard over the whole larynx.

In cases where œdema takes place, and the calibre of the larynx is consequently narrowed, the respiratory process becomes most laborious and painful, and the anxiety of the patient extreme.

"When catarrh is confined to the larynx, the expectoration is scanty. At first it is either absolutely wanting, or else it is clear or glairy. . . . As the disease progresses and begins to abate, the expectoration becomes thicker and more rich in young cells, which rather resemble pus-cells" (NIEMEYER).

With the laryngoscope the mucous membrane, at first only hyperæmic in appearance, is soon seen to become œdematous; and from the swollen condition of the epiglottis, the rest of the larynx is often hidden from view. If the vocal cords are visible, they are generally congested and slightly swollen, but not œdematous; the ventricular bands, however, generally quickly take on the œdematous character.

In addition to all these signs, there are the usual febrile symptoms of acute inflammation. In advanced stages the countenance becomes pale, the lips purple, and all the symptoms of embarrassed respiration are exhibited in a marked degree.

Prognosis.—In giving an opinion, the age of the patient is the most important consideration. In early life the disease is alway attended with great danger; and even in adults a very guarded opinion should always be expressed. Not only does the danger depend on the amount of œdema present, but, where tracheotomy has been performed for relief of the symptoms, there is still the risk of extension of the disease to the trachea and bronchi.

Causes.—Relaxing habits and previous attacks are the strongest *predisposing* causes of this affection. Males are more liable to it than females; and though adults are more frequently attacked than children, it is far more fatal in the young—more than 80 *per cent.* of the mortality occurring befor the tenth year.

Amongst *exciting* causes may be mentioned exposure to cold draughts of air; and irritating drinks, pungent vapours, and a dusty atmosphere may be considered as *traumatic* causes.

Laryngitis may also be caused by extension downwards of a similar affection from the pharynx. It rarely occurs from spreading of inflammation upwards from the trachea and bronchi.

Treatment.—In its early stages a warm, moist, and uniform tempertaure, with complete rest of the parts, is of the first importance. Not only should the voice be rested, but the tendency to cough should as much as possible be arrested by the administration of small doses of morphia, especially in those cases in which paroxysmal cough is a prominent symptom. Inhalations of hot steam, or of steam impregnated with the volatile principles of *benzoin, hops*, or *conium*, are of the greatest service. Hot poultices and fomentations may also be ordered. Neither local nor general blood-letting, blistering, *mercury*, *antimony*, nor other lowering remedies are to be recommended; but where the œdema is slight, non-depressing emetics, as *sulphate of zinc* or *copper*, dissolved in large quantities of warm water, are frequently of great service. Where the œdema is considerable, and, in spite of the foregoing remedies, does not appear to diminish, scarification of the larynx, by means of Mackenzie's laryngeal lancet, is of paramount value. Where, however, from circumstances this measure cannot be carried out, and the disease is advancing, tracheotomy should be performed without delay. All experience points to the fact that, where this operation is performed sufficiently early—especially in the adult—life is almost always saved.

In children, a warm moist temperature, poultices, warm emulcent drinks, and, if there is œdema, emetics of *sulphate of copper* are the most serviceable methods of treatment.

Although in very young subjects the laryngoscope sometimes cannot be used, scarification is often practicable, and not less effective than in adult patients.

It is necessary after an attack to caution a patient that he is very liable to a recurrence of this affection, and that he should therefore be very careful to guard against all preventible causes of the disease.

As laryngitis is more common to those engaged in in-door than outdoor occupations, and to those living in towns than in the country, Niemeyer recommends out-of-door exercise, with proper precautions, as a valuable prophylactic measure.

Acute laryngitis sometimes occurs as a complication of erysipelas; but *erysipelatous* inflammation of the larynx originating in that part is rare. The local treatment is the same as that required for ordinary acute laryngitis. The constitutional treatment should be that applicable to other forms of erysipelas. Tracheotomy in these cases, although offering a less favourable chance than in simple laryngitis, should be performed if the symptoms are urgent.

Mr. Durham states that he knows of five recoveries out of fifteen cases in which this operation was performed for erysipelatous laryngitis.

Of other *catarrhal* forms of laryngitis may be mentioned those of measles and scarlatina.

In *measles* the affection of the larynx may be either a simple catarrh, or a severe croupous affection. The catarrhal form may occur before the eruption appears, a day or two after the rash has come out, or when it is beginning to decline. Although the inflammation is often severe, it is seldom sufficient to cause anxiety.

The croupy or diphtheritic form is much less common, and "seldom begins until the eruption of the measles is on the decline, or the process of desquamation has commenced."

"Its appearance is most frequent from the third to the sixth day after the appearance of the eruption; but it oftener occurs at a later than an earlier period" (West). The treatment should be the same as that for primary croup.

In *scarlatina* the laryngitis may be either œdematous or croupous: they are fortunately both rare complications. The œdematous form may be one of the manifestations of the exanthem or may be dependent on debility, or it may be due to the renal affection, which is so common a sequel of scarlatina. The croupy form is not common, and seems to have been peculiar to some epidemics.

In all cases of laryngitis, associated with scarlatina, there is a great tendency to the ulcerative process. The treatment required is of a tonic and nourishing character, with the free use of well-diluted stimulants.

Tracheotomy may be necessary when there is œdema; but scarification should always be first tried.

(b.) *Sub-acute or Mucous Laryngitis.*

Pathology.—The disease is essentially a catarrhal inflammation of the mucous membrane, and is usually accompanied by a similar condition of the trachea.

The hyperæmic condition of the mucous membrane is followed by a slightly increased secretion; and, cell-growth taking place at the surface, the mucous membrane becomes swollen and sodden. There is a very slight, and, indeed, scarcely perceptible deposit in the sub-mucous tissue. The dysphonia or aphonia which is present, is due, partly to the altered density of the vocal cords, and partly to imperfect muscular action; for the slight pain which is occasioned by the use of the muscles causes them to be insufficiently employed.

Symptoms.—Congestion of the larynx may come on suddenly or gradually, and may disappear without spreading to the deeper structures. The symptoms are similar to those of the acute disease in the earlier stages, and the hyperæmic condition is at once seen with the laryngoscope; but there is seldom much constitutional disturbance. The affection is often associated in a slight degree with common faucial catarrh.

It may subside either spontaneously or under treatment, or it may become chronic.

Causes.—The disease is almost always catarrhal, or at least is attributed to taking cold. Those who have suffered once or twice from this affection become afterwards very liable to it. Any of the causes referred to under acute laryngitis may give rise to it.

Treatment.—In addition to functional rest, and an equable, warm, and moist temperature, a regular system of inhaling should be prescribed. Any of the appended forms may be employed; and there are many others contained in the very useful Pharmacopæia of the Hospital for Diseases of the Throat, published by Churchill, 1871.

For the administration of the following remedies, The Eclectic Inhaler possesses great advantages; for whilst the vapour to be inhaled is thoroughly saturated with the volatile principles employed, and is kept at a uniform temperature, the patient is able to inhale without effort.*

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* It can be obtained of Messrs. Bullock & Reynolds, 3 Hanover Street, Hanover Square, London; or of Maw, Son, & Thomson, London.

℞. Creasoti fl. dr. iij.; Glycerine fl. dr. iij.; Aquæ ad. fl. oz. iij.; *mix.* A tea-spoonful to be added to a pint of water at 150° Fahr., and inhaled for five minutes twice or thrice daily.

Ol. Pini. Sylvestris, fl. di. ij.–iij.; Mag. Carb. Lev., gr. 60–90; Aquæ ad. fl. oz. iij.; *mix.*, and use as above.

In addition, Oil of Juniper, ♏xx. ad. fl. oz. iij.; Oil of Santal, ♏xv.; Oil of Myrtle, ♏xx., are all of service.

It will be found convenient to prescribe light carbonate of magnesia as a medium, in the proportion of one grain to every two minims of the oil; and the addition of twenty grains of camphor to any of the above makes the inhalation additionally stimulative.

When mucous laryngitis is associated with inflammation of the pharynx, lozenges containing guaiacum are very useful in removing the hyperæmic condition. Even when the pharynx is not affected, the experiments of Fournié have proved that lozenges have a decidedly beneficial effect on diseases of the larynx.

(c.) *Chronic Laryngitis.*

Pathology.—This disease is essentially a chronic inflammation of the mucous membrane of the larynx, accompanied with slight enlargement and tortuosity of the vessels of the areolar tissue. Cell proliferation takes place at the surface; and instead of the pale semi-transparent mucus usually secreted in small quantities, a more abundant discharge occurs, in which pus is mingled in varying quantities with true mucous corpuscles.

Causes of Chronic Laryngitis.—In addition to those causes which predispose to, or excite the acute disease of which this is often a sequel, the abuse of alcohol and tobacco, especially the former, sometimes gives rise to chronic inflammation.

Symptoms.—The principal symptom is modification of the voice; the respiration is generally little affected; but there is often a tickling cough, with expectoration of small pellets of mucus, and a frequent desire to clear the throat.

Occasionally the patient complains of a pricking or tingling sensation in the throat. With the laryngoscope, general or partial congestion of the mucous membrane is at once visible. There is occasionally a certain amount of thickening when the chronic affection has originated in acute œdema. It is of great importance in these cases, by comparing the local manifestations with the constitutional condition, to decide whether the thickening be simply the result of inflammatory tumefaction, or œdematous infiltration, or whether it be due to tuberculous exudation. In all cases of chronic laryngitis of long standing the lungs should be carefully examined.

Treatment.—Applications of mineral astringents to the larynx, either with the laryngeal brush, or in the form of atomised inhalations, are of the greatest service; and the "alternation of topical remedies is often as efficacious in the cure of chronic laryngitis as it is in the treatment of chronic inflammation of other mucous passages."

Although Niemeyer is of opinion that "induration of the sub-mucous tissue (of the larynx), is incapable of resolution," the daily experience of all laryngologists affirms local treatment of chronic thickening of this region, unless, indeed, it be due to phthisis, to be attended with the very best results.

Stimulating inhalations may also be used with great benefit.

Dr. Morell-Mackenzie's experience is related in the "Use of the Laryngoscope," third edition, page 98, as follows:—"Amongst the remedies I have found most efficacious are solutions of perchloride of iron (gr. 60-120 ad. fl. oz. i.), chloride of zinc (gr. 20-30 ad. fl. oz. i.), sulphate of copper (gr. 15 ad. fl. oz. i.), sulphate of zinc (gr. 5 ad. fl. oz. i.), alum (gr. 30-60 ad. fl. oz. i.), chloride of aluminium (gr. 30-60 ad. fl. oz. i.) The perchloride of iron and chloride of zinc are the solutions I use most largely. Glycerine will also be found a most useful solvent for these agents, as its consistence is better calculated than that of water to keep up prolonged and close contact between the remedy and the affected membrane. I seldom employ solutions of nitrate of silver as applications to the larynx; for whilst I have not found them more beneficial than other mineral astringents, they are much more likely to produce spasm and nausea."

Of atomized solutions, those of tannin (gr. 5 ad. fl. oz. i.), and perchloride of iron (gr. 3 ad. fl. oz. i.), are most useful. Bergson's atomiser, known in this country under the name of Andrew Clark's Spray-producer, Dr. Richardson's Ether Spray apparatus, and Siegle's Inhaler, are the most convenient for administering atomized liquids.

Functional rest is, of course, of great importance in those cases of chronic laryngitis in which the voice is affected. If complete silence cannot be enforced, the patient should be recommended to speak only in a whisper.

Relaxation of the uvula, being a frequent cause of irritation of the larynx, should be treated by astringent lozenges of tannin, rhatany, or zinc; and if necessary the elongated part should be amputated.

A warm dry climate is of essential service in cases of chronic inflammation of the larynx; as are also some mineral waters, particularly those of Ems, Ober-Salzbrunnen, Les Eaux Bonnes, Luchon, Cauteret, Aux-les-Bains.

The principal varieties of chronic laryngitis are—first, Those due to a morbid state of some special tissue of the larynx; and, secondly, Those due to a morbid condition of the general system, and mainly dependent on constitutional causes. Of the former, we have Glandular Laryngitis and Phlebectasis Laryngea; of the latter, Syphilitic Laryngitis and Laryngeal Phthisis.

Chronic glandular laryngitis, or chronic inflammation of the minute racemose glands of the larynx, is generally associated with disease of the follicles of the pharynx and tonsils; but, as the glandulæ of the larynx are all of the racemose character, it is better to use the generic term, "glandular laryngitis." It is this disease which has most improperly received the term of *dysphonia clericorum*, or clergyman's sore throat. The most common throat affection of the clergy is not, however, glandular pharyngitis or laryngitis, but merely chronic inflammation of the pharynx and larynx. On the other hand, glandular laryngitis attacks other people just as much as the clergy, and is not at all peculiar to those whose profession requires them to make sustained use of the voice.

Chronic glandular laryngitis generally occurs in people of feeble constitutional powers.

The *symptoms* are very similar to those of ordinary chronic laryngitis, but more troublesome and more intractable. There is a constant sensation of a foreign body in the throat, and a corresponding disposition to hawk and clear the throat.

Pathologically the disease is essentially one of the secretory system, the normal secretion of the minute racemose glands, instead of being clear and transparent, becoming thick, white, and opaque.

By some observers it is thought to be due to imperfect or perverted digestion; but it is more probable that the glandulæ of the stomach are simultaneously affected.

The treatment is the same as that recommended for simple chronic laryngitis, except that in many cases it is necessary to entirely remove the follicular matter, and then apply the solid nitrate of silver to each pit in which the unhealthy secretion has been formed. It is often very useful to destroy the unhealthy surface by means of the laryngeal scraper. There is frequently considerable feebleness of the voice in these cases; and when this exists, lozenges of benzoic acid, stimulating inhalations, and even Faradization are indicated.

The second variety, that of *phlebectasis laryngea*, first described by Dr. Morell-Mackenzie (*Lancet*, July 6th, 1862), consists of a venous congestion of the larynx. It is very uncommon. The symptoms are slight, and usually consist in no more than an uneasy sensation in the larynx, and a more or less frequent cough.

With the laryngoscope, dark vessels may be seen on various parts of the larynx, particularly on the ventricular bands. The treatment consists in the local application of strong astringents, and of vigorous constitutional remedies.

The varieties of syphilitic laryngitis, and phthisical laryngitis depend on a special constitutional condition, and will be treated under the next subject, that of ulcer of the larynx.

ULCER OF THE LARYNX.

Latin Eq., *Ulcus;* French Eq., *Ulcère;* German Eq., *Geschwür;* Italian Eq., *Ulcera.*

Definition.—*Loss of substance of the larynx, rarely the result of simple inflammation, but usually caused either by syphilis or phthisis.*

Pathology.—(a.) *Syphilitic Ulceration.*—The great frequency with which the larynx is affected in both the secondary and tertiary forms of syphilis does not appear to justify the belief of Gehrardt and Roth, that the localization of syphilis in this organ is in some degree determined by fortuitous catarrhal inflammation. Mr. Durham states "that, according to his own experience, a very large proportion (from 30 to 40 *per cent.*) of the cases of laryngeal disease met with in hospital practice among the surgical out-patients, are of syphilitic origin (Holmes's *System of Surgery*, second edition, vol. iv., page 556).

Nor can the laryngeal complications in secondary syphilis be ascribed solely to extension of the disease from the pharynx, as not only do cases frequently occur in which the larynx is affected without the mouth, fauces, or pharynx having any signs of the malady, but also the larynx is frequently attacked some weeks after the disease has disappeared from the pharynx.

Ulceration of the larynx is exceedingly rare in secondary syphilis; but there is frequently a sort of mottled congestion of the vocal cords, which has some resemblance to the roseolous eruption so common on the skin.

The ulceration, when present, is generally quite superficial. The parts most frequently affected are the epiglottis and the inter-arytenoid commissure.

Condylomata are also rare in the larynx. Dr. Mackenzie estimates the proportion of condylomata in the larynx at about 4 *per cent.* of all well marked secondary syphilitic cases. Gehardt and Roth, on the other hand, give the proportion as eight out of fifty-four cases—that is, about 16 *per cent.*

Secondary laryngeal affections are sometimes difficult to cure, but being, in fact, of exanthematous character, after undergoing certain phases of evolution they disappear spontaneously. If allowed to run their natural course, condylomata generally disappear at the end of two or three months; but chronic congestion is more intractable. Stimulating inhalations, and the local application of astringents are, however, sometimes of great service. Secondary ulceration of the larynx is seldom so severe as to require the application of solid nitrate of silver, as recommended by some authors.

Tertiary syphilitic ulceration of the larynx always occurs in a late stage of the disease. Ulceration may extend from the palate or pharynx to the epiglottis; but much more frequently when a patient seeks relief on account of recent laryngeal disease the scars of former ulceration will be seen in the pharynx.

The character of the ulceration is its disposition to cause permanent loss of substance. It most commonly attacks the epiglottis; but the arytenioid cartilages and ary-epiglottic folds are often affected, and the vocal cords and ventricular bands not unfrequently suffer. The destructive process destroys both the mucous and sub-mucous tissues, and large portions of the epiglottis are frequently completely eaten away. (Fig. 49, A. and B. *).

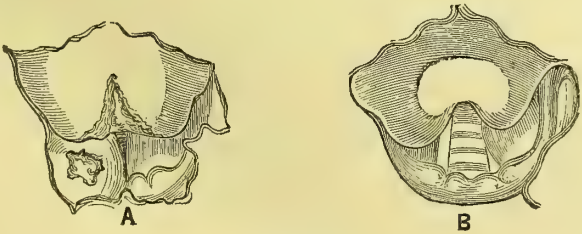

Fig. 49. *

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* A. Active Syphilitic ulceration of the epiglottis and right arytenoid cartilage, with œdema and general thickening. A "false excrescence" or cicatricial outgrowth projects across the left ary-epiglottic fold (View on Jan. 6, 1868).

B. The same larynx, eighteen days later, after treatment by iodide of potassium

Ulceration of the larynx occasionally originates from gummatose deposits. These are most frequently seen on the posterior wall of the larynx as large swellings, which become inflamed, burst, and leave a large open ulcer, which, if not checked, will speedily spread.

(b.) *Pathology of Phthisical or so-called Tubercular Ulceration of the Larynx.*—It is very doubtful whether laryngeal phthisis is in any way actually *caused* by disease of the lungs, but as a rule the pulmonary disease precedes the throat affection; and though Dr. Mackenzie states that "numerous cases of laryingeal phthisis have come under his inspection, where the most experienced stethoscopists have been unable to discover a trace of lung disease," he nevertheless confesses that, "on the other hand, he has seldom met with a case of laryngeal phthisis in the dead subject without finding pulmonary disease." The course of events of this disease is generally—first, hyperæmia; secondly, thickening; thirdly, ulceration. The ulcerations vary in size from that of a pin's point to a threepenny piece. The character and situation of the ulceration will be described under laryngoscopic appearances.

One of the common sequelæ of tubercular disease of the larynx is, caries of the cartilages.

Prognosis.—This must, of course, be most unfavourable. A patient in whose case there is pyriform swelling of the ary-epiglottic folds very rarely recovers, even if there be no evidence of lung disease. The symptoms may, however, be greatly mitigated and life prolonged by treatment. Those cases generally terminate most rapidly in which the epiglottis is much affected.

Phthisical ulceration of the larynx is generally preceded by thickening, and usually occurs as a complication of pulmonary phthisis. It may, however, be present before there is any discernible evidence of lung disease.

Causes.—"The causes are the same as those which give rise to other laryngeal affections (such as exposure to cold, functional excesses, &c.) —*plus* a special constitutional condition either inherited or acquired, through which cell-proliferation takes place in the sub-mucous tissues" (Morell-Mackenzie; see also article on *Scrofula*, page 1, vol. ii., and *Pulmonary Phthisis* at a subsequent page).

The Symptoms are very similar to those of chronic laryngitis. When the thickening is great, dysphagia is the most prominent symptom—solid food frequently getting into the larynx, and drink being violently ejected through the nares. The voice is affected at a very early stage. This is generally caused by change of structure in the vocal cords; but it may be the result of weakened approximative action of the cords. The cough is generally violent and paroxysmal at first, and subsequently aphonic; occasionally it is but a slight tickling cough, with but little expectoration. Respiration is always affected in the later stages of the disease, and dyspnœa is sometimes so extreme as to demand tracheotomy.

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and local remedies. The œdema has subsided, the ulcers are healed, and the right vocal cord has come into view. Some general thickening, however, especially of the epiglottis, still remains, and the left vocal cord is still hidden by the swollen ventricular band on the same side (View on Jan. 24, 1871).

With the *laryngoscope,* the first thing that often strikes the observer is the extremely pale and almost ashy colour of the mucous membrane.

When thickening has already commenced, the ary-epiglottic and inter-arytenoid folds are, as a rule, seen to be first affected. There may be only thickening of one of the ary-epiglottic folds, but later, both of the folds are generally affected, and the posterior portion of the larynx is seen to be swollen, and to have the appearance of one or two pear-shaped semi-transparent bodies, according as one or both sides of the larynx are diseased.

Although, for the sake of comparison, the accompanying diagrams represent only one side of the larynx as affected, the appearance is far more frequently symmetrical or nearly symmetrical. (Fig. 50, A, B, and C.)

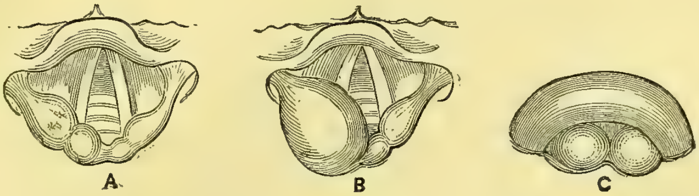

Fig. 50. *

When the epiglottis is attacked, the normal contour of the valve is often lost, and a turban-like appearance is produced.

The ulceration of phthisis, if it attack the mucous membrane, is generally of a molecular worm-eaten character. The edges of the epiglottis are often eroded and the cartilage exposed, and numerous small ulcers are often found at the root of the epiglottis, on a level with the vocal cords. Ulcers never commence on the lingual aspect of the epiglottis, although the ulceration may extend to the anterior surface of the valve. The most common situation for deep ulcers is the cartilaginous portion of the glottis; penetrating ulcers, which affect both the vocal cords and ventricular bands, being almost invariably present, in advanced cases, just behind the posterior extremity of the ventricular orifice. These ulcers appear to be due to hypostatic causes. It is in this situation that the arytenoid cartilages become affected, first by ossification and subsequently by caries.

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* A. *Incipient Laryngeal Phthisis.*—Deposit has taken place around the cartilage of Wrisberg and the Capitulum Sontorini, but the normal contour of the parts is not yet lost.

B. *Advanced Laryngeal Phthisis* affecting the right side of the larynx, and producing a characteristic pyriform swelling.

C. *Laryngeal Phthisis* affecting the epiglottis, and producing the turban-like swelling of the valve. The interior of the larynx is hidden by the thickened epiglottis, but the arytenoid cartilages can be seen as two round balls. The round appearance is due to the ary-epiglottic folds being hidden by the epiglottis. If the whole of the orifice of the larynx were visible, the "pyriform" appearance would be present.

Diagnosis.—The thickening of laryngeal phthisis may be differentiated from acute œdema by the rapid occurrence of the latter disease, and the much greater transparency of the simple œdematous swelling. The thickening of syphilis is much less symmetrical and much more irregular in every respect, and it is rapidly followed by destructive ulceration; or, if this be averted, by resolution and absorption. On the other hand, the thickening which succeeds extensive syphilitic cicatrization always gives rise to considerable distortion—a distortion which never occurs in laryngeal phthisis. In phthisis, thickening is an early symptom, and generally exists for a long time before deep ulceration takes place.

Dr. Mackenzie considers the pale pyriform swelling of the ary-epiglottic folds and mucous membrane covering the arytenoid cartilages, when present, as pathognomonic of phthisis in the larynx. Although, however, these pyriform swellings are typical of laryngeal phthisis, it must be clearly understood that the disease may present itself under other forms. Thus, there may be, as already remarked, turban-like thickening of the epiglottis, or molecular ulceration of the general mucous membrane or diffused deposit.

Treatment—(a.) *Syphilitic.*—Large doses of *iodide of potassium,* in combination with ammonia and freely diluted with water, are of the greatest use; but local treatment is also of the first importance. The application of solid nitrate of silver, or of strong solutions of the same salt (60 gr. ad. fl. oz. i.), or of sulphate of copper (15 gr. ad. fl. oz. i.), is attended with the most satisfactory results. For applying the solid nitrate the method of fusing a very small portion of the salt on to a curved aluminium wire far surpasses, in simplicity and safety, any other form of laryngeal *porte-caustique.*

The great danger of all forms of tertiary syphilitic disease is the narrowing of the glottis from thickening and œdema. The latter subject is treated at p. 714, under "*contraction of the larynx,*" but in these days of the laryngoscope, the cases should be very rare in which ulceration cannot be arrested and healed.

(b.) *For phthisical ulceration,* remedies similar to those recommended for chronic laryngitis may sometimes be employed with advantage. Mineral astringents have a decidedly beneficial effect in the early stages, and are also useful in relieving the dysphagia when ulceration of the epiglottis has taken place. Sedatives, whether in the form of inhalations or as medicines, which allay the cough, and so keep the larynx at rest, are also to be recommended.

Treatment, however, when the disease is well manifested, is at the best but palliative. Every effort should therefore be made to carry out preventive measures. Congestion of the larynx, therefore, in patients of a phthisical tendency should be treated with the greatest promptitude—first, By proper local treatment; secondly, By complete rest of the voice; thirdly, By a suitable climate—a warm, dry, and uniform temperature being most to be recommended.

The constitutional treatment required is, of course, similar to that which will be found described under Pulmonary Phthisis.

ABSCESS OF THE LARYNX.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abcès;* GERMAN EQ., *Abscess;* ITALIAN EQ., *Ascesso.*

Definition.—*Inflammation resulting in a circumscribed collection of pus, generally associated with disease of the cartilages.*

Pathology. — Circumscribed abscess of the larynx is rare, and generally originates in chronic inflammation of the cartilages. The perichondrium separates from the cartilage, and, pus collecting beneath the investing membrane, a circumscribed abscess is formed. Most commonly, however, a communication is established (through the perichondrium) between the cartilage affected and the areolar tissue of the larynx, and the abscess becomes diffused. Diffuse intercellular suppuration of the larynx is not very uncommon. It may occur as a sequel to chronic disease of the cartilages, or it may arise as an idiopathic affection. In the latter case, it occurs as an acute affection, and is, in fact, a most dangerous variety of laryngitis. When following necrosis of the cartilages, the origin of the disease may generally be traced to *phthisis* or to *syphilis.*

Much less uncommon are abscesses connected with the larynx, in which the effusion takes place in parts external to the larynx. In these cases it is generally the external cartilages of the larynx that are the seat of the disease, and a laryngeal fistula is frequently produced.

ŒDEMA OF THE GLOTTIS.

LATIN EQ., *Œdema glottidis;* FRENCH EQ., *Œdème de la glotte;* GERMAN EQ., *Oedem der Glottis;* ITALIAN EQ., *Edema della glottide.*

Definition.—*Effusion of serum which accumulates in the folds of the mucous membrane of the larynx.*

Pathology.—This subject has already been treated under the head of acute laryngitis; and in this place it will be principally considered as a secondary phenomenon.

It sometimes occurs in exanthematous laryngitis; and though not found in small-pox or measles, it is occasionally a complication of scarlatina, erysipelas, and typhus. In typhoid, the predisposition to œdema is even greater. In all these cases the œdema is more likely to be followed by ulceration than in ordinary acute laryngitis, where resolution is not unfrequently the termination. Œdema is also an almost constant consequence of disease of the cartilages, and may then also be considered as secondary. As a complication or sequel of renal disease, œdema of the larynx is occasionally seen, but not so frequently as to justify the specific form described by Fauvel as "*aphonie albuminurique.*" Œdema is, of course, likely to occur in lax tissues, in which the areolar tissue is abundant. Hence, we most frequently find it in the folds of the mucous membrane of the larynx, viz.:—the glosso-epiglottic, ary-epiglottic, inter-arytenoid, &c. The mucous membrane over the vocal cords being immediately in contact with the elastic tissue of the cords—there being no submucous areolar tissue in this situation—the œdematous fluid cannot pass the barrier of the vocal cords. Hence, we have a supra and a sub-glottic œdema. The favourable results likely to accrue from tracheotomy in the former case will be at once apparent when the anatomical arrangement is taken into consideration.

Treatment.—In all cases of œdema of the larynx, whatever the cause, scarification and hot steam inhalations, as recommended in acute laryngitis, are the most serviceable aids. When suffocation is imminent, tracheotomy should be performed.

NECROSIS OF THE CARTILAGES OF THE LARYNX.

LATIN EQ., *Necrosis cartilaginum;* FRENCH EQ., *Nécrose des cartilages;* GERMAN EQ., *Necrose des Knorpels;* ITALIAN EQ., *Necrosi delle cartilagini.*

Definition.—*Death of the cartilages, by a molecular or truly carious process, usually preceded by separation of the perichondrium.*

Pathology.—This disease—which should more properly be termed caries—is in most instances a sequel of *laryngeal phthisis* or *tertiary syphilis.* It may, however, depend on a gouty diathesis, or it may originate as a purely local disease—the lungs not being in any way affected.

It is now generally accepted that death of the cartilage of the larynx most commonly originates in separation of the perichondrium. Ossification of the cartilage generally precedes its death by caries.

Two very instructive cases, bearing on this point, are related by Dr. Mackenzie, in the 21st vol. of the *Pathological Transactions.* In the first (p. 46), severe pain in the left side of the throat, preceded any swelling in the larynx, or in the tissues external to it, for many months. After death, a circumscribed abscess, the size of a hen's egg, was found, which almost completely blocked up the œsophagus; and at the bottom of the abscess a small portion of the cricoid cartilage—about the size of a threepenny piece—was found to have been completely removed by molecular destruction.

In the second case (p. 56), there was from the commencement extreme dysphagia; and this was the prominent symptom. After death "a large abscess was found in the thickened tissues, covering the anterior surface of the spinal column, and extending from the fourth cervical to the second dorsal vertebra. The vertebræ themselves were not exposed or diseased. The abscess involved both the lateral walls of the œsophagus, the sides of the pharynx as high as the upper border of the cricoid cartilage, and the anterior wall of the œsophagus, from the middle of the cricoid cartilage downwards for two inches. The posterior surface of the lower third of the cricoid cartilage was completely exposed, and had not undergone ossification. At the lower border of the cricoid cartilage was a vertical fissure, about half an inch long, communicating between the trachea and œsophagus." In this case the disease of the cartilage may fairly be said to have been secondary to the abscess.

Whenever the cartilage is exposed to the air, through ulceration of the superjacent tissues, it will be seen to be blackened or greatly discoloured.

The cartilages most frequently affected are—firstly, the arytenoid; next, the cricoid; and, lastly, the thyroid.

There is a rare form of disease of the cartilages of the larynx, first described by Rühle, in which the cartilages are found to be increased in volume rather than diminished. In this case, in place of ossification, the cartilages undergo fibrous degeneration, and become quite soft. A case of this kind is also reported by Dr. Morell-Mackenzie in the 21st vol. of the *Pathological Transactions*, p. 58.

The Symptoms of necrosis of the cartilages are similar to those of chronic laryngitis and phthisis; but great pain is generally experienced, and there is almost always considerable dysphagia.

With the laryngoscope, a slight swelling in the region of the affected cartilage is first seen. This swelling, which is generally of a semi-œdematous character, gradually increases, and subsequently either pus slowly exudes from the surface of the swelling, or the pent up matter suddenly escapes. In those cases in which the external cartilages of the larynx are affected, pus forms in the tissues of the neck external to the seat of disease. It rarely happens that the necrosed cartilage is exposed during life. Hunter recorded a case in which the cricoid cartilage was expectorated, and the patient subsequently made a good recovery; and several cases are on record in which the arytenoid cartilages have been expelled during life.

Treatment.—The practitioner's treatment must clearly be directed to the relief of the pain and dysphagia. For the former, sub-cutaneous injections of morphia should be administered; and for the latter, as nothing can be done to actually dilate the narrowed œsophagus, the patient must be fed by enemata or the œsophagus tube. The preferable way, in cases which will admit of it, is to use an œsophageal tube with a feeding-bottle attached.

By means of an instrument of this kind, in one of the cases referred to, the patient's life was sustained for more than three months; and after death there was a considerable layer of fat covering the body. Of course, great care must be taken in passing an instrument in cases of this kind; but, where practicable, this method of feeding is far more satisfactory in every respect than the use of enemata per rectum.

CONTRACTION OF THE LARYNX.

LATIN EQ., *Coarctatio;* FRENCH EQ., *Rétrécissement;* GERMAN EQ., *Verengerung;* ITALIAN EQ., *Contrazione.*

Definition.—*Narrowing of the passage of the larynx the result of various lesions.*

Pathology.—This condition is most frequently due to tertiary syphilitic ulceration, and the contraction is caused by cicatrization and puckering of the mucous membrane. Cases have occurred in which the glottis has become narrowed by adhesion of the vocal cords after ulceration. In these cases there is generally a sort of web between the two cords, which, when divided, has a great disposition to form again.

The contraction may, however, be due to the chronic thickening of laryngeal phthisis or to simple chronic ulceration. The canal of the larynx is also sometimes narrowed by the pressure of a tumor external to it. (See an interesting pamphlet on this subjeet by Dr. Massei.*)

The treatment of narrowing of the larynx resolves itself sooner or later into tracheotomy.

In cases of syphilitic cicatricial thickening, iodide of potassium does little good, and local treatment is of no avail. In chronic tubercular narrowing, treatment is equally futile. In all cases, if the dyspœna is extreme, tracheotomy must be performed at once.

BENIGN GROWTHS IN THE LARYNX.

LATIN EQ., *Polypus laryngis;* FRENCH EQ., *Polype du larynx;* GERMAN EQ., *Larynxpolyp;* ITALIAN EQ., *Polypo del laringe.*

Definition.—*New formations of benign character, forming projections on the mucous membrane of the larynx, generally giving rise to aphonia or dysphonia, often to dyspnœa, and occasionally to dysphagia.*

Pathology.—Chronic congestion of the mucous membrane of the larynx is, far above all other causes, the most important œtiological feature in the production of simple morbid growths in the larynx. The most common cause of hyperæmia is catarrh, and catarrh must therefore be looked upon as the great predisponent. The various other influences such as acute diseases, the inspiration of irritating vapours, and particles of matter, and various occupations, probably only act through establishing a condition of hyperæmia. Neither syphilis, nor phthisis, nor any constitutional condition appears to favour the growth of these neoplasms.

Growths in the larynx are most frequent in the middle period of life. They occur oftener in the male than in the female sex; and are especially to be looked for in persons following those occupations in which constant use of the voice is necessary.

Benign growths are essentially of local origin, the result of a perverted nutritive process, in which growth is excessive and development imperfect.

Dr. Morell-Mackenzie gives the following as the various kinds of benign growths in the larynx:—

Papillomata and *benign epithelial growths, fibromata, fibro-cellular tumors, cystic tumors, fasciculated sarcomata, lipomata,* and *angeiomata,* and they are here enumerated in their order of frequency.

Papillomata are by far the most common of benign laryngeal growths, and occur in about 70 *per cent.* of all cases. They vary in size from a grain of mustard to that of a walnut, but they do not often attain the latter dimension. Their most common size is that of a large split pea. They may have a mamillary or cauliflower configuration. They are generally of a pink colour, but may be white or even bright red. " In their general form and arrangement they have many points of resemblance, but on an enlarged scale, to the papillæ, which in various localities constitute natural projections from free surfaces, more especially from the skin and mucous membranes. Their basis-substance is formed of connective tissue, which is continuous with that which normally exists in the part; whilst the free surface is covered by an epithelium, which may vary in thickness and its number of layers according to the seat of the tumor. Blood-vessels and even nerves enter into the interior of the papillæ." * Papillomata grow rather quickly, are generally situated on the vocal cords, and show a greater disposition to recur than other growths.

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* Sui Restringimenti Laringei, del Dottor F. Massei. Napoli, 1871.

Benign epithelial growths may be considered as a sub-variety of papillomata. They constitute a very small proportion of laryngeal neoplasms. In these tumors the epithelial scales do not clothe papillæ, but form continuous layers of more or less undulating character. These growths generally occur in the neighbourhood of the cords, and are of a white or pale red colour. In nearly all cases of laryngeal growth, the epithelium is of the tesselated variety. Simple epithelial formations are not very common, but occur most frequently in children.

Fibromata are present in about 11 *per cent.* of all cases, and are seldom found before adult life. The rate of growth of these neoplasms is very much slower than that of papillomata. Their structure is that of simple fibrous tissue. They are usually of smooth appearance, round or oval in form, and of a bright red colour. They show no disposition to recurrence.

Fibro-cellular growths, or *mucous polypi,* consist of more or less perfectly developed fibro-cellular tissues, and have diffused through their substance a greater or less quantity of serous-like fluid. They are often semi-transparent in appearance, and resemble nasal polypi; but differ from them in showing no disposition to recur. The other kinds of growth are too rare to require any detailed description. Their structure is generally that of similar tumors in other parts of the body.

Symptoms.—The subjective signs of growths in the larynx may be very slight, or they may be very severe. A feeling of uneasiness, a desire to clear the throat, and, very rarely, a slight pricking pain, are perhaps the only sensations experienced by the patient; and in many cases these are altogether absent. On the other hand, there may be extreme dyspnœa, and even suffocation may occur. When the growth is connected with the epiglottis, or with the posterior wall of the larynx, so as to press upon the food tract, dysphagia is the most prominent symptom.

The most important objective signs are aphonia, or alteration of the voice, and dyspnœa (for this symptom is objective as well as subjective). There is almost always some modification of the voice. Dysphonia is more common than aphonia. Out of 100 cases treated by Dr. Morell-Mackenzie, and analyzed by him, there was modification of the voice in ninety-two instances, complete aphonia being present in fifty-five cases. Impairment of the voice was the *unique* symptom in no less than 52

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* Lectures on Surgical Pathology, by Sir James Paget, F.R.S., 3rd ed., p. 591.

per cent. of all the cases (*Essay on Growths in the Larynx*, Churchill, 1871, p. 19).

Cough is not a very frequent sign of growths, occurring, as it does, in only about 8 or 10 *per cent.* of Dr. Mackenzie's tables of all published cases. It is but very occasionally of a very violent or paroxysmal character.

Difficulty of breathing depends entirely on the size and situation of the growth. In the accounts of the specimens collected in the various hospital museums in pre-laryngoscopic times, almost all the patients died from suffocation. The dyspnœa is not unfrequently of a severe and paroxysmal character; the attacks of suffocation generally occur at night; and it almost invariably happens that inspiration is much more difficult than expiration.

The laryngoscope at once reveals the presence of a growth in the larynx. The situation can almost always be ascertained by this means; but in a few cases, when the growth is very large, the exact seat of origin may be hidden. It may also happen that a growth in the upper part of the larynx may conceal others situated lower down.

The general health is, as a rule, little affected by the presence of benign growths, unless the difficulty of breathing has existed so long as to give rise to constitutional disturbance.

Course and Termination.—The various symptoms previously described may develop themselves slowly, and, after a time, remain stationary without causing serious inconvenience, unless an attack of catarrh or some other accidental circumstance arise. Amongst the lower classes patients will suffer from loss of voice for many years without seeking medical aid; but if dyspnœa or dysphagia occur, they will at once apply for relief. Some growths are exceedingly rapid in their development, and may attain such a size in a few weeks or months as almost entirely to occupy the larynx.

The tendency to death in all cases of growths in the larynx is by suffocation; but such a result should never occur in the adult, unless the patient refuses to submit to tracheotomy. In children this termination is more likely to take place, because the difficulties of diagnosis and treatment are much greater.

In the young, also, the chances of recovery from the palliative treatment of tracheotomy are considerably less.

Two cases are on record in which growths in the larynx have been cured spontaneously by becoming separated and expelled by coughing; but it is scarcely necessary to remark that such a fortunate accident cannot be anticipated.

Diagnosis.—Tumors of the larynx cannot well be mistaken for any other disease. In carcinoma there is usually ulceration; and in phthisis there is a general thickening—not a defined growth as in the case of a true neoplasm. In both these diseases there is the constitutional marasmus. In syphilis "false excrescences"—the result of ulceration—are sometimes met with. Eversion of the ventricle is a very rare occurrence; but when present it has very much the appearance of a laryngeal polyp.

Prognosis must be considered in relation to life and voice. A benign growth is seldom fatal to life in the adult, if treatment be adopted; but in children a more cautious opinion must be given. The presence of a growth in the larynx of a young subject may at any time cause suffocation; and the only treatment which in some cases is expedient—viz., tracheotomy, is in itself attended with much greater risk in young patients. With reference to restoration of voice, an opinion must be formed according to the size, situation, and nature of the growth.

Treatment.—In cases where the growth is small, does not appear to increase, and does not give rise to functional derangement, no treatment need be adopted. Where the opposite conditions, however, exist, means should be at once taken to eradicate the growth, or to place the patient out of danger by performing tracheotomy.

Radical treatment may be divided into *internal*—that is, removal or destruction of the growth through the mouth with the aid of the laryngoscope; or *external*, by operations through the neck. Internal treatment may be farther subdivided into *Mechanical* and *Chemical.* Mechanical treatment consists in evulsion, by common laryngeal forceps, tube forceps, *écraseurs*, or guillotines. Added to this, there are a few cases in which crushing or incision of the base of the growth is effectual. Chemical treatment consists in the application of various caustic solutions,—a process seldom satisfactory, and in galvanic cautery. This last method has been very successful in the hands of some Continental practitioners; but it cannot be recommended, on account of the complicated and unwieldy apparatus required by the practitioner, and of the great pain often experienced by the patient.

External treatment may be divided into thyrotomy, or division of the thyroid cartilage, supra-thyroid laryngotomy, or division of the thyro-hyoid membrane, and infra-thyroid laryngotomy, or removal of the growth through the crico-thyroid membrane, or by an opening made in the trachea. In addition to these various surgical procedures, there is the combined method, in which tracheotomy having been called for, on account of the urgency of the symptoms, the neoplasm may afterwards be removed *per vias naturales.* All external measures are attended with less satisfactory results, both as to life and restoration of function, than those resulting from laryngoscopic treatment; and it should be borne in mind that the more serious surgical methods *should never be resorted to unless the tumor in the larynx produces symptoms endangering the life of the patient.*

APHONIA.

LATIN EQ., *Aphonia;* FRENCH EQ., *Aphonie;* GERMAN EQ., *Stimmlosigkeit;* ITALIAN EQ., *Afonia.*

Definition.—*Loss or modification of the voice.*

Pathology.—Loss of voice, or at least modification of voice, is such a prominent symptom in almost every laryngeal disease, that to describe it as a separate affection would be not less unscientific than inconvenient. It will now be considered chiefly in relation to neuroses or nervous affections of the larynx.

Neuroses may be primarily divided into (1.) Diseases of the motor system; (2.) Diseases of the sensory system.

Diseases of the Motor System.

This division embraces "*Paralysis of the glottis*," or more properly speaking, *paralysis of the muscles of the vocal cords*, and "*Spasm of the glottis*," or *spasm of the muscles of the vocal cords.*

This subject has been investigated by Gerhardt, Türck, Ziemsen, and others; but Dr. Morell-Mackenzie was the first to differentiate and analyze the various paralyses of the vocal cords, and to point out the treatment suitable for each variety. The remarks on this subject are extracted from his "Essay on Nervo-Muscular Affections of the Larynx,"—Churchill, 1868—to which the reader must be referred for fuller information.

Paralysis of the Muscles of the Vocal Cords.

The paralytic affections of the muscles acting on the vocal cords may be sub-divided as follows:—

1. Bilateral paralysis of the adductors. 2. Unilateral paralysis of the adductors. 3. Bilateral paralysis of the abductors. 4. Unilateral paralysis of an abductor. 5. Paralysis of the tensors. 6. Paralysis of the laxors.

These last two may also be either bilateral or unilateral.

Bilateral paralysis of the adductors (*crico-arytenoidei laterales, and arytenoideus proprius*), is a condition in which there is "inaction of the adductors on to the sides, preventing approximation of the vocal cords on attempted phonation, and consequently giving rise to loss of voice." This, the most common form of laryngeal paralysis, is known as nervous, functional, or hysterical aphonia.

Pathology.—The disease is essentially one in which the nerve-force is feebly or imperfectly evoked, or is not directed in the proper channel.

Causes.—Debility and hysteria are undoubtedly the most common causes of this disorder. It occurs much less frequently in association with uterine disturbance than is commonly supposed. Women are, however, far more subject to it than men, but the affection occasionally occurs in the male sex.

Loss of voice originating in hyperæmia not unfrequently remains as a functional phenomenon after the congestion has disappeared.

Symptoms.—The essential symptom is loss of voice; though, however, the power of voluntary phonation is lost, the reflex vocal acts are not generally affected, so that the cough, sneeze, and to a less extent the laugh, are accompanied with a distinctly laryngeal sound. With the laryngoscope it is seen that, on the attempted phonation of a vowel sound, as "*ah*," "*a*," "*o*," the vocal cords do not approximate. The laryngeal mucous membrane is generally pale.

The **Prognosis** is very favourable.

Treatment should be directed towards stimulating the larynx so as to produce a mild spasm.

Inhalations of creosote, pungent applications, &c., may be used, but the most certain method of effecting this object is Faradization of the larynx by means of the "laryngeal electrode,"* by which the electric current can be easily passed through the affected muscle.

The most obstinate cases succumb in the end to this treatment, and frequently a few applications of the current are sufficient to restore the voice to its full power. Constitutional treatment of a tonic character is also frequently indicated.

Unilateral paralysis of the adductors (of a vocal cord) depends on "inaction of the adductors on one side preventing the approach of the corresponding vocal cord to the median line, and consequently giving rise to hoarseness or loss of voice."

Pathology.—The disease differs from the bilateral paralysis of the adductors in never being of an emotional character. There is, probably, in some cases a special morbid condition of some filaments of the recurrent laryngeal nerve, but the actual lesion found after death is often merely more or less complete atrophy of the affected muscles. The disease, in many cases, probably consists, from the very beginning, in idiopathic disease of the muscle; in other cases there is probably an inflammatory exudation, either of simple or dyscrasic character.

Causes.—The condition may be due to chronic toxœmia, (lead, arsenic, diphtheria, &c.,) may result from cerebral disease, or may be caused by cold, or muscular strain. Aneurism of the arch of the aorta, or of the right subclavian or carotid, may cause paralysis of the left or right adductor, but the really important muscular affection in these latter cases is the associated paralysis of the *abductor.*

Symptoms.—The laryngoscope at once reveals the nature of the paralysis. On attempted phonation the affected vocal cord remains at the side of the larynx, whilst the healthy one is well adducted to the median line. Occasionally the mucous membrane is congested. There is aphonia or dysphonia, and the character of the cough, sneeze, and laugh are greatly altered in tone. *This circumstance enables those not using the laryngoscope to differentiate this affection from the functional aphonia or bilateral paralysis of the adductors.* There is not unfrequently slight dysphagia. In cases in which the paralysis is dependent on cerebral disease, there will be loss of power on the same side of the tongue and palate.

Prognosis.—Serious apprehensions need not arise in those cases in which the condition is due to local causes, but in those rare cases in which the disease is cerebral the prognosis is serious.

Cases due to blood poisoning and catarrh, are favourable for treatment, but the cure is often tedious.

Treatment.—Faradization, as recommended in bilateral paralysis of adductors, is the most appropriate treatment in those cases in which the cause is local, but the affection is much more obstinate. The Faradaic current must, in these cases, be applied more frequently, for a longer time at each sitting, and must be continued for many weeks. Constitutional remedies may be used with advantage in cases of chronic toxæmia.

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* Made by Mayer & Meltzer, 59 Great Portland Street, London.

Bilateral paralysis of the abductors of the vocal cord (*crico-arytenoidei postici*) depends on "inaction of the abductors on both sides preventing the outward movement of the vocal cords on inspiration, and consequently giving rise to dyspnœa and stridulous breathing."

The *causes* are sometimes cerebral; but morbid influences, which affect both pneumogastric or both recurrent nerves, may give rise to it. Of these, bronchocele, scrofulous deposits in the bronchial and cervical glands, and cancer of the œsophagus, are the principal. It sometimes depends on simple degeneration of the muscles, without there being any evidence of implication of the nerves. The condition is rare.

Symptoms.—With the laryngoscope it is at once seen that the vocal cords, instead of being drawn widely apart on inspiration, remain nearly approximated—the opening of the glottis being in proportion to the degree of the paralysis.

The voice is not generally much affected, but it may be slightly hoarse. The least exertion brings on stridor, and in sleep the breathing is invariably stridulous. The cough is croupy. The affection may be mistaken for spasm of the adductors; but in spasm the vocal cords are constantly varying in the degree of adduction, whilst in the cases of paralysis, the cords are never thrown back beyond a certain point.

Pathology.—The disease consists essentially in a loss of power of the *crico-arytenoidei postici*, caused by interception or non-generation of the nerve current. After death these muscles are generally found to be considerably atrophied.

The **Prognosis** is very serious, both on account of the immediate danger of suffocation, implied by the condition of the larynx, as well as on account of the probable existence of some serious disease in the brain, or in the neighbourhood of the pneumogastric nerve or its branches.

Treatment.—No medical treatment is of any avail, but when the breathing is at all seriously embarrassed, tracheotomy should be performed in all cases without delay.

Unilateral paralysis of the abductor of a vocal cord may be defined as "inaction of an abductor on one side, preventing the outward movement of the corresponding vocal cord on inspiration, and consequently giving rise to more or less dyspnœa and stridulous breathing."

Pathology and Causes.—Where the disease is of any long duration, the abductor of the affected side, when examined after death, is always atrophied, and in cases of long standing, will often have undergone fatty degeneration.

The causes are the same as those which produce the bilateral form, but the condition now under consideration is more often due to peripheral influences,—that is to say, to pressure on one pneumogastric, or one recurrent nerve. Aneurism of the arch of the aorta, and other intra-thoracic tumors, cancerous or glandular, not unfrequently involve the left recurrent nerve. Cases in which the right nerve is involved are rare. Cancer of the œsophagus in its last stage often affects one recurrent nerve. Strumous glands and tumors along the trachea may also involve one of the recurrents. The condition may also be due to cerebral disease.

Symptoms.—The condition is at once recognized with the laryngoscope; the affected cord is seen to remain fixed in or near the median line when the patient takes a deep inspiration. Stridor, though not so severe as in the bilateral form, is a constant symptom.

The voice is shrill. The constitutional symptoms vary according to the cause of the paralysis; but when the disease has existed for some time there is usually more or less irritative fever.

Prognosis.—As this condition generally indicates serious disease elsewhere, a very unfavourable opinion must, as a rule, be given.

Treatment.—Little can be done curatively, but tracheotomy should be performed, if the dyspnœa is at all urgent. In cases of aneurism of the arch of the aorta, it is most important to ascertain whether the dyspnœa is due to direct pressure on the trachea, or to pressure on the recurrent nerve. The laryngoscope at once clears up this point. Where the symptom is due to nerve pressure, tracheotomy is indicated, whilst, if the dyspnœa be due to pressure on the trachea, it would be useless.

In addition to the foregoing more evident and serious forms of paralysis, there are others in which the tensors and laxors of the vocal cords are affected. The loss of power is generally caused by a too violent or too prolonged use of the voice, and occurs in singers, clergymen, and military or naval officers. The most suitable treatment is Faradization, with complete functional rest.

Spasm of the tensors of the vocal cords is an affection in which, the vocal cords being unduly and irregularly stretched, the voice is feeble, jerky, unsteady, and constantly rises to a high key.

Pathology.—The condition appears to be due to constant perverted action of the vocal cords. The cerebro-spinal system does not appear to be at fault; but the affection is in all probability simply one of spasm of the expiratory act, in which the thoracic and abdominal muscles participate.

The **Cause** is probably to be found in an artificial and strained use of the voice.

The **Symptoms** are those described in the definition. The varying voice differentiates it from the constantly high pitched tone of paralysis of the laxors.

Prognosis as to recovery of the natural voice is always unfavourable in cases of long standing. There is, however, no danger whatever to life.

Treatment consists in complete rest of the voice for a lengthened period, soothing inhalations, and the application of belladonna to the larynx externally. When the patient is allowed to use his voice again, he should receive instruction to speak slowly, and take a full breath at very short intervals, so that the pauses for rest are much more frequent than is usual. He should also be instructed to speak with a "full chest"—that is, with a good volume of air to set the vocal cords in vibration.

Diseases of the sensory system of the larynx.—Hyperæsthesia, or increased sensibility, occurring independently of inflammatory disease or structural alteration of the tissues of the larynx, is undoubtedly a rare morbid condition, but several cases have been reported. The inhalation of hot sedative vapours and anæsthetics does good in some cases, and narcotics are also indicated. Neuralgic affections should be treated on the ordinary principles which regulate the therapeutic management of such cases.

Anæsthesia.—Although there is a great difference between the sensibility of the glottis in different people, anæsthesia rarely occurs as a distinct morbid affection. Disease affecting the origin, or trunks of the pneumogastric nerves, or their superior laryngeal branches, would be likely to diminish the sensibility of the larynx in proportion as the function of the nerves was interfered with.

Impaired sensibility of the mucous membrane of the larynx has been observed by Romberg in cholera.

SECTION IV.—DISEASES OF TRACHEA AND BRONCHI.

BRONCHIAL CATARRH.

LATIN EQ., *Catarrhus bronchiorum;* FRENCH EQ., *Catarrhe bronchique;* GERMAN EQ., *Bronchialcatarrh;* ITALIAN EQ., *Catarro bronchiale.*

Definition.—*Infiltration and œdema of the mucous membrane of the bronchial tubes, so that the tumefaction diminishes their calibre, attended first with dryness of surface, followed by a watery secretion, which subsequently becomes turbid and yellow; and sometimes attended with chilliness and the discomfort of what is called a "common cold."*

Pathology.—The disease is extremely common, and certain conditions are known to predispose towards the affection, as pointed out by Niemeyer, namely:—(1.) Childhood; (2.) Badly fed flabby individuals, the walls of whose capillaries are probably weak, running through soft and yielding tissues, with a tendency to hyperæmia and increased transudation. The anatomical constitution associated with scrofula and rickets is of this description; (3.) A previous attack predisposes to others; (4.) Effeminate modes of life.

The directly exciting causes which Niemeyer also enumerates are—

(1.) Impeded evacuation of the bronchial veins.

(2.) Obstruction to the current of the blood through the great branches of the aorta below the origin of the bronchial arteries, such as may be caused by the compression of liquid in the peritoneum, accumulation of excrement or of gas in the intestines.

(3.) Irritants acting directly on the mucous membrane, such as dust, vapours, hot and cold air. The irritant materials from certain trades, such as that of bakers, millers, and stone-cutters.

(4.) Chilling of the skin and the influence of sudden changes of temperature, as sitting in a draught of air when perspiring freely.

(5.) As a result of the morbid states of the blood, as when bronchial catarrh is a premonitory symptom of enteric fever, measles, and small-pox. Under this head might be noticed the opinion of Dr. Hyde Salter, that bronchial catarrh depends on a specific animal poison, whose material presence circulates in the blood, and whose irritation acts on the mucous membrane.

Symptoms are generally combined with catarrh of the *larynx*, of the *nasal mucous membrane*, and that of the *frontal sinus* and *conjunctiva;* but which rarely spreads to the smaller bronchi. Pains "all over," aching of the joints, limbs, and back, with a sense of tightness across the forehead, and chilliness, usually usher in a "common cold." Then follow discharge from the nostrils, at first watery, with copious flow of tears, hoarseness, or rough voice, dryness of throat, furred tongue, thirst, loss of appetite, and quickened pulse.

Treatment.—When the symptoms of a "common cold" first express themselves, and even when the sensations have extended to the chest, as indicated by the hoarseness and tendency to cough, the disease may be at once subdued in a healthy person by a full stimulant (but not narcotic) dose of *opium* or *morphia* (i. e., *one grain of opium, or a fourth of a grain of morphia*) at bedtime; or by an alcoholic diaphoretic drink; or by *five* grains of *carbonate of ammonia,* or *ten* to *twenty* grains of *muriate of ammonia;* or, if the appetite is unimpaired, a full supper, followed by a hot alcoholic stimulant, may have the same effect.

When the attack is sudden, and attended with marked depression, associated with aching pains in the head, back, and limbs, and much febrile disturbance, opium, if taken early enough, is a perfect remedy. "One-third of a grain of *muriate of morphia* ought to be taken at bedtime, and its influence will begin to be felt in from twenty minutes to half an hour, by the gradual disappearance of the sense of intense weakness, the relief of the pains, and that peculiar feeling of thorough and evenly distributed warmth of the whole body which is so different from fever on the one hand or chilliness on the other" (ANSTIE, *op. cit.*, p. 120). Natural sleep ought to supervene, and the morning ought to find the patient well.

If such remedies are delayed too long, the object next to be aimed at is to induce a copious perspiration and a continued action of the skin and kidneys, in the first instance by small doses, frequently repeated, of *antimonial* and *ipecacuanha wines,* or *nitrate of potash,* or *acetate of potash,* as well as *bicarbonate of potash* and *aqua potassæ,* combined, at a much later period, with tinctures of *squills* and *hyoscyamus.*

In the early stage of the disease the bronchial membrane has its normal moist condition altered to a very dry state; and the object of treatment is to bring about a return of the naturally moist condition. This is best effected by the inhalation of the vapour of hot water, the use of *tartar emetic* in doses of *one-twelfth* to *one-sixth* of *a grain,* the use of *ipecacuanha* in *one-quarter or half-grain doses;* and these may be aided by *hemlock, henbane, aconite,* or *hydrocyanic acid* in appropriate doses. From the frequent inhalation of steam great benefit is derived (EASTON).

Opium has often been regarded as hurtful in the first stage, because it has been taught that it interferes with the free secretion of mucus, so as to render expectoration difficult. This is not quite true. The onset of the catarrh and free expectoration is in consequence of the abatement of the inflammation,—opium tends to subdue the inflammation, and in this respect acts as an expectorant. Thus it is often of great service in the first stage of a common cold, as it is in most acute inflammations (Flint).

The inhalation of iodine vapour every three or four minutes for an hour is said to be of service, each inhalation lasting one minute, by merely holding a bottle of tincture of iodine in the warm hand under the nose (Tanner). Total abstinence from liquids for forty-eight hours, originally recommended by Dr. Richard Lower one hundred and forty years ago, is a practice recently revived by Dr. C. J. B. Williams.

Muriate of ammonia, ʒss. in solution, is a remedy much in use also, combined with *liquorice* or syrup, with one grain of *tartar emetic* and one or two drachms of *antimonial wine,* dissolved in six or eight ounces of water. Of this a table-spoonful for a dose, frequently repeated, may induce relief, when the discharge from the nose and trachea is very viscid. Copious warm drinks and abundant warm bed clothing, to induce diaphoresis, are also efficient remedies to be used early.

The object aimed at in the treatment of a "common cold" is to establish a free catarrh—to soften the expectoration and make the coughing "loose" and easy. Niemeyer lays down some practical rules, based on symptomatic indications, to accomplish these ends.

(1.) In chronic colds or catarrhs, where cough is incessant and distressing, and out of proportion to the expectoration, narcotics are indicated—such as ten grains of Dover's powder at bedtime, or the *camphorated tincture of opium* (*paregoric elixir*). External counter-irritants are also of service in the form of mustard poultice, "mustard leaves," or blistering substances, short of vesication, such as stimulating liniments. *Chlorate of potass* is also a useful remedy (Flint).

(2.) Dyspnœa, with extensive sibillant rhonchus and irritability of the mucous membrane, are also relieved by narcotics, combined with *ipecacuanha,* or with *tartar emetic.* But narcotics are contra-indicated if, owing to the feebleness of the patient, the efforts of expectoration are inadequate to prevent accumulation in the bronchial tubes.

Alkaline carbonates possess the property of thinning and rendering easier of expectoration the viscid bronchial secretion, and are on that account to be recommended, especially where the condition of the urine suggests such remedies; but if care be taken to watch the remedy, and that nothing contra-indicates its administration, *opium* in its many forms is the most useful remedy, so long as the first stage of inflammation exists. Preparations of *conium, hyoscyamus,* or *hydrocyanic acid* must be used in cases where opium is contra-indicated.

(3.) When the secretion is profuse, with relaxation of the mucous membrane of the bronchi, when large, soft, moist *râles* are heard, *stimulant* expectorants are indicated,—*senega, squills, carbonate of ammonia, benzoin,* and *oil of cubebs.* Remedies in the form of gas are also of service, such as the fumes of tar, by boiling it alone or mixed with water; also vapour of turpentine, half a drachm being put into a bottle of hot water, the patient inhaling for a quarter of an hour, four times a day, through a mouthpiece attached to the bottle.

In all cases absolute rest, vegetable diet, demulcent drinks, and small doses of antimonials will greatly tend to bring about relief. A pint of any demulcent fluid (such as lintseed tea), with two drachms of *antimonial wine*, taken in small quantities, so as to be consumed in twenty-four hours, will be found a good standard prescription. *Gum arabic*, in the proportion of *one ounce* to the pint of water, or decoction of *marsh mallow*, or of *dried fruits*, is a good substitute for the *lintseed*, and may be flavoured with anything agreeable to the taste of the patient, such as orange or lemon syrup.

If much fever exists, a very hot foot-bath just before going to bed has a soothing effect. The air of the bed-room must be kept at about 60° Fahr., not below, and the patient must remain as quiet as possible.

Generally, in prescribing cough mixtures, which ought if possible to be avoided (seeing that they so impair the functions of the stomach), the following conditions ought to be adhered to in devising the mixture :—

(1.) That the prescription should be in the form of *fluid* remedies, —solution, or infusion of wines or syrups of the drugs.

(2.) That the dose should not exceed one tea-spoonful.

(3.) That the proportions contained in the mixture should be so arranged that a dose may be given every two, three, four, or six hours, according to the urgency and acuteness of the symptoms.

Such a mixture may contain in each dose, for an adult, from ten to thirty minims of *antimonial wine*, or of *ipecacuanha wine*, or of *tincture of sanguinario*, or of *syrup of squills*, or of *vinegar of squills*, with *two minims* to four minims of *hydrocyanic acid*, or thirty to sixty minims of *tincture of hyoscyanus* or of *conium;* or from two to four minims of *tincture of opium;* or from thirty to sixty minims of *camphorated tincture of opium.*

BRONCHITIS.

LATIN EQ., *Bronchitis;* FRENCH EQ., *Bronchite;* GERMAN EQ., *Bronchitis;* ITALIAN EQ., *Bronchitide.*

Definition.—*Inflammation of the air-passages leading to the pulmonary vesicles, characterized by hoarseness, moderate cough, heat, and soreness of the chest anteriorly—symptoms which are more and more intense according to the severity of the disease. The natural mucous secretion is at first arrested, but subsequently it becomes increased in amount and altered in quality, tending to assume the corpuscular character.*

Pathology and Morbid Anatomy.—The mucous membrane lining the bronchial tubes may undergo the inflammatory process, followed by results peculiar to the texture of the part affected, and the dimensions of the tubes.

In diffuse bronchitis we find the inflamed portions of the mucous membrane of a deep venous red, and this redness may be general or partial, in spots, streaks, or arborescent forms, the result of injection and of ecchymosis. The more asthenic the inflammation, or the more feeble and cachectic the patient, the more livid and purple is the redness, and the greater the ecchymosis. The substance of the mucous membrane also is softened, swollen, and easily torn, the result of infiltration, causing an œdema which reduces the calibre of the tubes. The secretion of the tubes, at first arrested, is eventually increased in quantity and variously altered in quality. It becomes thin, watery, and frothy, and subsequently thicker and more consistent, assuming the appearance of pus (see vol. i., p. 23). Abortive or young epithelial cells, loaded with serous effusions, and losing their vital cohesion with the basement membrane, are rapidly and easily discharged, constituting the thin, watery, frothy, serous expectoration of bronchitis in its early stage. Fibrinous exudation subsequently abounds, and the expectoration becomes tenacious, more opaque, and even pus-like. Occasionally it has occurred that the expectoration of bronchitis is of a very fetid character, so that the case simulates gangrene of the lungs. An instance of this description (with other analogous cases) has been carefully described by Dr. Laycock, Professor of the Practice of Medicine in the University of Edinburgh. A chemical analysis of the expectoration in this case demonstrated the presence of *butyric* and *acetic acids;* and the odour was characteristic of the *butyrates of ethyl.* It resembled the smell of the May-flower or apple blossoms, but was combined with an odour of fæces (*Med. Times and Gazette*, May, 1857, p. 480). This condition is generally associated with dilatation of the tubes (*Bronchiectasis*).

When the chest is opened after death in cases of acute bronchitis, where the finer or capillary bronchial tubes have been implicated so as to be obstructed or occluded by mucus, the lungs do not collapse as they ought to do in health; but, on the contrary, they bulge out of the opened thorax with considerable force, so as to convey the idea that they were too bulky for the cavity which contained them; and which did not contain them without compression of the air contained in the air-cells; and although the inspiratory expansion of the chest ceased with death, the lungs do not collapse on account of the obstructed bronchi preventing the escape of the air in the alveoli (NIEMEYER).

Bronchitis may affect one lung, or both lungs, or a part of a lung, and the upper lobes are more commonly affected than the lower ones. The larger bronchi are also supposed to be more commonly inflamed than the smaller ones. Hence it is that the more marked primary effects of bronchitis are most obvious in the bronchi towards the roots of the lungs; while the secondary effects which are apt to follow a prolonged or severe attack, such as *vesicular emphysema,* are mostly developed towards the pleural surfaces of the lungs, and especially towards those surfaces most directly under the influence of the expansion of the more moveable portions of the thoracic parietes.

A result of bronchitis which in its morbid anatomy may be mistaken for tubercle of pulmonary phthisis has been recently demonstrated by Zenker, of Dresden. It is described as catarrhal induration of the pulmonary air-vesicles, by the accumulation of epithelium carried into them from the adjoining smaller bronchi, and which eventually fills up and consolidates the vesicular structure of the lung where this takes place. Zenker is one who does not believe in the possession of epithelium by the air-vesicles; and the tortuous vessels seen in them like varicose veins, especially in *stenosis* of the mitral valve, he regards as the normal state of vessels which are naked and uncovered by epithelium.

One most direct, invariable, and important result of bronchitis in the adult is condensation of the vesicular substance of the lung, of a peculiar type, as a result of mucous or other obstruction in the air-tubes leading to the condensed portion. Professor W. T. Gairdner has shown this, and that such a condensation is most apt to occur in bronchitis associated with asthenia, although in such cases the amount of obstruction may be small, as in patients debilitated by disease or by age. Such condensation is also produced by collapse of the pulmonary vesicles, the boundaries of this local condensation being distinctly mapped out by the interlobular divisions of the pulmonary lobules implicated. Such collapse, when extensive and sudden, is not only a frequent cause of death, but at the same time it is a fleeting temporary condition of immense frequency, and important practically to distinguish from the condensation of pneumonia.

The morbid anatomy of bronchitis, which an examination of the air-passages discloses, shows that obstruction in the tubes may be due to fluid mucus, or even to solid fibrinous coagula, or more or less prolonged spasm of the circular muscular fibre, and that such obstructions tend to the production of pulmonary collapse; and if the obstruction be considerable and persistent, large portions of the lung may be emptied completely of air in the course of a few hours. The mechanism by which an obstruction, such as mucous fluid or more solid substances, brings about this collapse, appears sufficiently obvious when it is observed that such obstruction is never absolutely complete. The air gradually finds its way out by the edges of the obstructing substance, impacted as it is more or less in a series of diminishing tubes, such as the bronchi, and acting the part of the ball-valve of a syringe, so that when the obstruction is driven onwards towards the narrower tubes by the force of inspiration, occlusion is more or less perfect only in the one direction. The expiratory force, however, so long as there is air in the vesicles, constantly tends to dislodge the obstructing body by pushing it towards the wider end of the tube. While, therefore, the entrance of the air is constantly and more or less effectually opposed, its exit is always permitted, so that ultimately the air-vesicles of the tubes beyond the seat of obstruction become completely emptied, and they collapse. The result of such collapse is a condensation of the tissue of the lung—a condition which had previously been ascribed to a limited inflammation of the pulmonary tissue, known as *lobular pneumonia,* and which was commonly believed to be peculiar to infants. Such collapse and condensation of the lung, however, whether in the lobular or diffused form, is an exceedingly common lesion in the adult, as shown by Dr. West and confirmed by Dr. W. T. Gairdner, especially in old persons, in typhus fever and in fatal dysentery, and is always associated with a certain degree of bodily weakness. Dr. Gairdner has further shown, however, that it is almost invariably found as a concomitant of fatal bronchitis, and that it depends on the obstruction of the tubes, as just described. When this collapse becomes permanent, it leads to still more obvious and important results:—

1. Like other parts which become useless, the collapsed portions become atrophied, leaving only a small amount of fibrous tissue in its place, the proper and special elements of the pulmonary tissue having disappeared. Such an atrophy causes a diminution of the volume of the lung at the place where the collapse occurs.

2. By a definite law (which may be expressed thus:—*That a compensation by increased volume in one or more parts of the thoracic viscera invariably occurs to make up for diminished bulk in another, the internal capacity of the chest remaining the same*) this pulmonary collapse and atrophy invariably leads to vesicular emphysema of the lung, and may even tend to dilatation of the heart itself. The most constant result of collapse, however, is emphysema, so much so that the one almost never occurs without the other.

Bearing upon these statements, several general phenomena have been noticed, which may be thus shortly enunciated:—

1. That emphysematous portions of lungs are almost invariably free from every diseased appearance, except the dilatation of the air-vesicles, and the consequent stretching and disorganization of their parietes.

2. That the bronchi leading to them are usually quite free from obstruction.

3. That vesicular emphysema by *increase of volume* of those portions of the lung to which the air has access, compensates for the *diminished volume* of the collapsed portion.

4. That the vesicular emphysema prevails in the opposite parts of the lung to those in which the direct effects of bronchitis are observed.

The following are the forms of bronchitis which may be clinically recognized:—(1.) *Acute bronchitis* (a.) *of the larger and medium-sized tubes*—(b.) *Capillary bronchitis, and of the tubes generally—the "peripneumonia notha" of the older writers;* (2.) *Chronic bronchitis;* (3.) *Plastic bronchitis;* (4.) *Mechanical bronchitis,* such as knife-grinders' disease—*carbonaceous bronchitis,* or black phthisis; (5.) *Bronchitis secondary to general diseases,* such as typhoid fever; (6.) *Bronchitis secondary to blood diseases;* (7.) *Syphilitic bronchitis.*

(a.) *General Symptoms of Acute Bronchitis of the Larger Tubes.*

Bronchitis, of whatever kind, is generally preceded by fever, but more commonly by symptoms of what is commonly called "a *cold*," or "a cold in the *chest*," as already described. It often commences without any previous illness, and the uneasy sensations—frequently commencing about the region of the frontal sinuses, gradually pass down the nasal mucous passages, and thence by the trachea and windpipe—are experienced in the chest, especially over the anterior region. The symptoms of bronchitis becoming developed are expressed by the hoarse altered voice, the cough and expectoration, and are too palpable to allow us to mistake the nature and existence of the disease. In a very few instances of diffuse inflammation, especially in *capillary bronchitis*, the cough is dry and without expectoration; but far more generally it is accompanied by sputa. The sputa vary greatly according to the different degrees of inflammation, or according as that inflammation is acute or chronic, sthenic or asthenic. In acute cases it is at first a thin mucus, sometimes streaked with blood, then more opaque, and lastly purulent; in more chronic cases it may be merely a muciform saliva, or a gelatiniform mass; or it may be like the unboiled white of egg, so tenacious that it may be poured from one vessel into another without separating. In other instances it is puriform, varying from a laudable pus to a red or green putrilage. When purulent, it is usually formed into sputa, but in a few cases it is thrown up in large quantities unmixed, as from an abscess. The quantity of matter expectorated also varies greatly; sometimes only a few sputa in the morning, at others half a pint or a pint in the twenty-four hours, while other patients actually die suffocated from the immense quantity which is suddenly poured out, causing obstruction of tubes and collapse of the vesicular structure of the lungs.

The cough is seldom accompanied by any pain in the inflamed membrane, and has many degrees of violence. It may occur in paroxysms, and the sputa be discharged after a violent effort, at night or in the morning, or at other definite intervals. Again, it may be incessant, harassing the patient at every instant, causing a sense of soreness or constriction of the chest, and sometimes severe pain at the ensiform cartilage, in consequence of the mechanical exertion of coughing.

The urine of bronchitis varies greatly, as the grades of the disease are almost infinite, from a very slight affection of the larger tubes to a disease involving all the smaller tubes of both lungs, accompanied, perhaps, by collapse of the air-cells in some cases, and impeding aëration to a great degree in all. In these cases the urea is in large amount, and the pigment is increased, while the chloride of sodium is sometimes as deficient as in the height of extensive hepatization; and it appears to Dr. Parkes that those cases in which the urinary ingredients are in extremely small amount are more commonly those of severe and general capillary bronchitis. The retention or non-excretion of the urinary substances seem to be more common in diseases attended with a considerable impediment to aëration than in other affections in which the absorption of oxygen is presumably less interfered with (Parkes, *On the Urine*, p. 282).

With respect to the effects of the cough on the constitution, the patient, (supposing the disease to be unconnected with any morbid poison or organic affection of the substance of the lung,) suffers little in his general health, and often feels he would be well if he could get rid of "the cough." In other cases he loses flesh, ejecting every meal, from the violence of the cough, or he sinks into a state of marasmus simulating phthisis. His pulse is generally natural, although in some cases it is frequent; his bowels also are regular. In bad cases, however, the patient's nights are broken, and he sleeps towards morning; while in slighter cases he sleeps through the night, but is disturbed early in the morning by cough and expectoration.

The duration of bronchitis is uncertain. It sometimes terminates in a few hours, sometimes in a few days, ceasing with the cold weather that ushered it in. In other cases its duration is long, and it is with difficulty recovered from; thus often laying the foundation of other formidable diseases which may ultimately destroy the patient. In old persons it generally returns every winter, or lasts, with intermissions, during the whole year.

Physical Symptoms.—The natural and healthy respiratory *bruit* of an adult has been compared to the sound heard during the calm sleep of a healthy child. In bronchitis this sound in the adult is changed, and varies, in different cases, from a tolerably sharp sound which, when multiplied from a number of bronchi similarly diseased, resembles a chirping sound, or the bass notes of the violoncello. The sounds thus embrace a musical scale of considerable compass, the principal and more marked division of which compose the *sonorous* and *sibilant* râles, as they are termed. The cause of the higher notes has been supposed to be owing to a thickening of the mucous membrane at the orifices of the various bronchial tubes, so that the natural *embouchure* is narrowed, and a musical wind instrument is thus formed. To those who have observed in the dead body a swollen state of the bronchial membrane, this explanation may seem satisfactory; but to those who have not seen such a phenomenon, it seems more easy to explain this morbid sound by the different degrees of contraction of the circular and longitudinal fibres of the bronchi, in the same manner as we observe contraction of the muscular fibres of the œsophagus, or of the small intestines, causing a constriction. Besides the alteration of tone of sound in bronchitis, its quality is also often affected by the presence of liquid matters within the cavity of the bronchi; and hence we have it interrupted and modified by the air passing through bubbles of mucus; and as the size of these bubbles and their viscidity vary, so the sounds vary. Hence a scale was established by Laennec, whose extremes are the "râle muqueux," and the "râle tracheal;" the former representing the bursting of small slightly viscid bubbles, the latter larger ones of greater tenacity, and yielding a gurgling sound. Sometimes this mucus, instead of being fluid, hardens so as occasionally to adhere and play as a valve, giving rise to a clicking noise. These are the various morbid sounds heard in bronchitis; and the danger of this disease is denoted by the quantity of fluid effused, and may be judged of by the nature of the sound. The sharp chirping sound is more to be feared than the graver and deeper notes; for grave sonorous notes originate in the larger tubes, chirping whistling notes in the smaller; and the danger in bronchitis increases in proportion as the finer bronchial tubes become involved (*Edin. Med. Journal*, 1864, p. 1114). When the sounds of expiration are also much prolonged, severe bronchial inflammation is indicated.

Percussion generally returns a healthy sound in bronchitis. An important physical sign is one which indicates a sudden disappearance of the respiratory murmur over a definite part or parts of the lung. Percussion sometimes shows, however, that the part still contains air; and therefore it is presumed that the disappearance of the murmur is due to obstruction of one or more of the bronchial tubes; and which, as already shown, may lead to *collapse*, *condensation*, *atrophy*, and *emphysema*. In some cases the respiration is greatly accelerated, varying from 36 to 50 or 68 in a minute, especially in cases of capillary bronchitis. The pulse at the same time rapidly loses its strength, and becomes excessively frequent—120 to 150. The ratio of the respiration to the pulse is therefore greatly altered from the normal standard—that is, about 4 or or $4\frac{1}{2}$ beats of the pulse for every respiration; and thus *pulse-respiration* ratios of 3·0, 2·5, or 2·25 to 1 are not uncommon.

Diagnosis.—It is hardly possible to confound bronchitis with any other disease; but there is often much difficulty in assigning its cause and distinguishing it from phthisis. The absence of great emaciation, and the clear resonance returned on striking the chest, are the most salient points in diagnosis.

(b.) *Acute Catarrh of the Smaller Bronchi* (*Capillary Bronchitis*).

Here the cough is of far more violent character from the commencement, and the sputa scanty. The seat of pain in the chest is mainly at the points of insertion of the muscles upon the chest and epigastric regions, from the spasmodic jerking movements of the act of coughing, which is of a violent character, and continues in long paroxysms.

The sputa from these smaller bronchi contain no air, and are specifically heavier than water. They are extremely tenacious, and retain the shape of the small thread-like tubes from which they come. They, therefore, cling to the lighter and frothy secretion of the larger tubes, which, being full of air, floats on the surface of water. Thus, when the expectoration of acute capillary bronchitis is cast into water, the fine filaments of the adhesive thread-like exudation can be seen hanging in the water, suspended from the frothy mucus which floats on the surface.

Differences of opinion prevail as to the frequency of this form of bronchitis, and also as to the ages at which it prevails. Niemeyer describes it as common in the adult and in children, and in old persons debilitated in health. It has been generally regarded as most common at the two extremes of life—infancy and old age,—its occurrence in youth or adult age as exceptional, and then only amongst the weakly. Dr. Clymer's experience leads him to believe that it is more common in adults of both sexes than is generally admitted, and is not limited to those of feeble health or the infirm from previous disease. It may happen in persons of vigorous and sound constitution. He cites the death of the late Emperor Nicholas, of Russia, in the full strength of manhood, and refers to a fatal case, related by Dr. T. K. Chambers, of a clergyman "of large frame and great height." Dr. Clymer's observations have also satisfied him that capillary bronchitis was not uncommon among the American soldiers during the American war, especially the coloured troops—and that it was often mistaken for pneumonia or ordinary acute bronchitis.

It is sometimes met with as a secondary or intercurrent affection in the course of exanthemata in children, particularly of measles (*Broncho-pneumonia* of Trousseau).

It has also been described as lobular pneumonia when coincident with collapse of the lobules.

The following excellent account of the remaining features and treatment of this disease is given by Dr. Clymer:—

" **Symptoms.**—An attack of capillary bronchitis may begin with shivering, though an initial chill is much less frequent than in pneumonia. Headache is often complained of, the tongue is furred, the face pallid, and the expression anxious, often from the outset. There is a feeling with an aspect of general debility, frequently from the beginning, and out of proportion to the objective symptoms, the patient having the appearance of being in the first stage of a continued fever, though the peculiar besotted look is wanting. The breathing soon becomes hurried and somewhat laborious; an incessant hacking cough sets in, which is generally dry, with the occasional expulsion of one or more greyish, or yellowish-grey, stiff pellets; or there is scarce, frothy expectoration. The pulse-rate is quickened, the skin dry, and its temperature variable. (There is yet no reliable record of the body-heat.) These symptoms soon grow worse; the breathing becomes more accelerated and difficult, and the breathlessness more harassing. The ratio of the respiration to the pulse is always high, the respirations going up to sixty or even seventy in the minute, with a pulse-rate of 120 or 130, the pulse respiration ratio being changed from 4·5 to 2. The supplementary respiratory muscles are brought into vigorous action, and the supra-sternal, mastoid, scalenal, and infra-xiphoid spaces, and the base of the chest along the insertion of the diaphragm, are forcibly sucked in at each inspiratory effort. The face betokens great suffering and is of a bluish hue, the lips are purple, the conjunctiva has a bluish-grey tint and is turgid, and the skin of the extremities is of a dusky hue, and frequently at times cool and moist. There is great exhaustion. In many cases these symptoms are for a while paroxysmal, with intervals of comparative comfort between the exacerbations. If the disease progresses, all the phenomena due to deficient oxygenation of the blood are aggravated; the cyanosis deepens, the facial anxiety is extreme and distressing, the surface is livid and damp, words are spoken with great difficulty, the dyspnœa is intense, and all the respiratory muscles are forcibly working; the patient is unable to lie down, though completely worn out. A frequent, short, moist cough, coming on in spells, replaces the ceaseless hack, and expectoration becomes free, the sputa being brought up during or after each fit; they are frothy and aërated throughout, or spongy at the top, ropy, and adhering together as a single mass when turned out of the vessel. As death approaches the pulse usually becomes more rapid, and is large and compressible, and then small and thready. The respiratory efforts are less violent, the number of respirations begin to lessen, and the pulse-respiration ratio falls, and approaches to a more natural standard—the deepening asphyxia probably making the patient less sensible of the respiratory wants—and carbonic narcosis may possibly, in some cases, retard the heart's action, and decrease the pulse-rate. Towards the fatal end, both the cough and the expectoration cease, and death happens from apnœa.

"The *physical signs* of capillary bronchitis are, *percussion-sound*, at first natural over the whole chest; subsequently it may be slightly exaggerated over the superior and interior regions, from the emphysematous state of the air-cells; and in some cases there are limited spaces of dulness, owing to collapse of the air-cells of one or more lobules, caused by obstruction of a bronchial tube: towards the close there may be diminished percussion-resonance on account of pulmonary œdema, and excessive accumulation of the morbid products in the air-cells and terminal air-tubes. *Auscultation* in the beginning shows the respiratory murmur clearer and somewhat exaggerated in the upper portions of the lung, with diffused dry rhonchi over the chest, the sonorous rhonchus being often quite loud and musical and the sibilant of high pitch; if the respiratory murmur is not masked by the rhonchi, it is in these regions feeble; as we proceed downwards, and as the disease progresses, the distinctive rhonchus of capillary bronchitis—the subcrepitant—is heard, particularly in the posterior inferior regions. This fine moist rhonchus is mainly an inspiratory sound, though audible in expiration, and is described as resembling the continuous bursting of innumerable small unequal bubbles, or the sound produced by squeezing a not too wet sponge close to the ear. Heard at first about the base of the lungs, it soon becomes audible over nearly the whole chest, above and below, front and back, moving always upwards; it is from the beginning symmetrical. If the larger air-tubes are more involved, a mucous or submucous rhonchus may replace the dry rhonchi in the middle regions. When the lobules are collapsed, the signs of localized condensation of the lung-tissue can rarely if ever be made out, on account of the existing rhonchi. *Vocal resonance* is increased. There is, no doubt, bilateral local expansion of the chest, often of large extent; and Dr. Flint says that he has known the anterior, superior, and middle regions, in a young child, to become largely dilated, presenting the heteromorphism of long-continued and great emphysema, which disappeared on recovery; but owing to the violence and labour of the respiratory act it is very difficult, and generally impossible, to make any accurate measurement.

"The course of the disease is rapid; in children generally lasting from three to six days; in adults from a few days to two weeks; in old persons it rarely exceeds a week after the acute symptoms have set in. In young children capillary bronchitis sometimes comes on very insidiously, the symptoms being for a while those of an ordinary catarrhous affection, and exciting little attention, although there are really distinctive traits present almost from the outset,—drooping, pallid face, quickened breathing, and spells of coughing, preceded and followed by wheezing. Dyspnœa suddenly happens, and chest-exploration shows extensive capillary bronchitis. In pulmonary emphysema of long standing, with extensive vesicular dilatation, intercurrent localized capillary bronchitis is very apt to happen; but it, so far as the writer's observations go, never becomes general, and, though for a time aggravating the symptoms of the original disorder, is not a serious affection.

"**Prognosis** is unfavourable, especially when capillary bronchitis supervenes upon any existing disorder which limits the respiratory area, and when in such cases it is general. In infancy and old age it is very fatal.

"**Diagnosis.**—From simple acute bronchitis it can always be easily distinguished, both by the general symptoms and physical signs. Acute bronchitis is nearly always preceded by symptoms of "a cold," and its course is downwards and peripheral, the proximal larger bronchi being first invaded, and afterwards the smaller distal ones. In capillary bronchitis the morbid action begins in the minute tubes near the external surface of the lung, and extends in an inverse direction,—towards the large bronchial branches. The extreme frequency of the respiration in capillary bronchitis is another distinction, for in ordinary bronchitis it is not usually much hurried. The sputa, too, are characteristic. But it should be remembered that bronchitis of the larger bronchi may extend to the smaller ramifications, and that capillary bronchitis may supervene upon and complicate simple bronchitis. The diacritic phenomena, both objective and subjective, of capillary bronchitis and pneumonia are so well marked that it is difficult to account for the frequent errors of diagnosis. It is of much practical importance to distinguish the two disorders, both as regards prognosis and therapeutics. The initial chill is much less constant in capillary bronchitis than in pneumonia, rarely amounting in the former to more than shivering, and there is reason to believe that the body-temperature is never so high. In the bronchial disorders the debility is earlier, the physiognomy different—more anxious and livid—the respirations quicker, the febrile re-action greater, and the sputa, when present, distinctive. Pneumonia is commonly unilateral, capillary bronchitis bilateral. The physical signs of the two affections are unlike, especially those shown by auscultation, the characteristic rhonchus of each affection having individual qualities. When collapse of the lobules has taken place in capillary bronchitis, the differential physical diagnosis may be less marked; but even here the rational symptoms and the presence and diffusion of the symmetrical pathognomonic rhonchus should prevent error. Occasionally a case of acute phthisis is met with that at first sight bears some resemblance in the objective symptoms to capillary bronchitis—one is mentioned by Dr. Flint—but physical exploration and a history of tuberculosis will indicate the real nature of the disease. In plastic bronchitis there is subcrepitant rhonchus, but it is limited and never general, and the expectoration is characteristic.

"**Treatment.**—If a happy issue of this disorder is to be looked for, the treatment must be strictly conservative; spoliative remedies lessen, if they do not destroy, the chances of recovery. Like similar pathogenetic diseases, it is what is styled self-limited, having an inevitable cycle to travel over. The tendency to death is by apnœa, from the imperfect oxygenation of the blood—the area of available respiratory surface being greatly diminished, both from the morbid state of the minute bronchi and the accumulation of diseased products in them. The patient must be properly and adaquately nourished, and stimulants are often required, at least at times. Tonics should be early administered. The chest may be covered with hot poultices, and an oil-silk jacket worn over them. Dry cupping, in some instances, has seemed to give relief to the urgent symptoms. The writer's (Dr. Clymer's) experience is favourable to the use of the muriate of ammonia —two grains every two hours—either alone or in combination with the chlorate of potash. The carbonate of ammonia has long had a certain reputation in capillary bronchitis, especially in the later stages. Whatever effect it may have is probably not due to its supposed stimulant properties, but to specific action upon the diseased tissue and its products."

(c.) *Chronic Bronchial Catarrh.*

The basis of all the symptoms are those of catarrh already described; the form of bronchitis is an extensively prevalent one; and the changes pass from the trachea into the ramifications of the bronchi. A frequently relapsing and protracted catarrh, recurring habitually once or twice a year in spring or autumn, or both, commences the chronic affection, the patient generally remaining exempt during the summer, till at last the symptoms and the catarrh are more or less constant all the year round.

Treatment.—Abundant experience has shown that general bleeding in acute *bronchitis* uniformly weakens the patient, without greatly influencing the disease. Neither has medicine any very marked effects in the cure; for although some persons rapidly get well under a given treatment, yet many similar cases, under exactly the same treatment, will run on for weeks, and perhaps for months, without any amendment. In the most acute cases of bronchitis, however, some blood may be taken from the chest, with great caution not to take too much, either by cupping between the shoulders or by leeches, and in general from ten to twelve ounces are sufficient. It is only in cases of congestion of the brain, heart, or venous circulation threatening asphyxia, that general blood-letting is imperative. Next to blood-letting, *tartarized antimony* administered in solution in doses of *a sixth* or *a quarter of a grain, every three or four hours,* conduces to free secretion, and generally to mitigate the symptoms of the disease. Dr. Fuller regards *digitalis* as a useful adjunct to the antimonial treatment; and the air-passages should be fomented by the inhalation of moist warm air, as by the steam of hot water, the secretions being at the same time stimulated, so that the bowels act freely. When the expectoration becomes thicker and less copious, the antimony may be decreased, and *squills,* or *ipecacuanha,* with *paregoric,* given. After this a blister should be applied to the chest; and on its being removed, a large lintseed poultice should be placed over the blistered part, and be continued for many hours, which will not only keep the ulcerated surface open, but gratefully foment the part and relieve the patient. The bowels should be freely evacuated by a purgative dose of *calomel,* combined with *compound jalap powder,* and they should subsequently be kept in regular and gentle action by some neutral salt, such as the *sulphate of magnesia* in the *liquor ammoniæ acetatis,* combined at the same time *with some nitrate of potass.* The *compound jalap powder* is a most useful remedy when the system will bear it. Œdema is greatly relieved by its use, whether of the lungs or of the body generally. The neutral *citrates, tartrates,* or *acetates* of the alkalies are useful eliminating remedies.

After these means have subdued the severity of the symptoms at the outset, expectoration should be promoted by such remedies as *squills, ipecacuanha,* and *tartar emetic,* combined with *hyocyamus* or *conium.* Opium in narcotic doses (*i. e.,* above a grain) is inadmissible if the evacuation of the loaded air-passages is to be promoted, because its tendency, in a narcotic dose, is not only to diminish the secretion, but to paralyze the action of the mucous passages in eliminating that secretion. When large doses of opium have been given at this stage, death has been known to follow, and necroscopic examination has revealed the air-passages loaded with frothy serous mucus, and the air-cells congested and collapsed. It is not till after secretion has begun to diminish, in acute cases, that opium may be prescribed with benefit in stimulant doses, and it is then to be given in the form of the solution of the salts of morphia, added at bedtime to the doses of the *cough mixtures* so usually administered. If the disease shows a disposition to pass into the chronic stage, *quinine,* with *squills* and *conium,* may be administered, or a draught containing full doses of *cinchona, with five grains of carbonate of ammonia,* or *ten to twenty grains of the muriate of ammonia, and thirty or forty minims of the compound tincture of benzoin or of the balsam of Peru,* will generally facilitate expectoration and relieve the dyspnœa. If a "*hearty cough*" is not attended by easy and free expectoration, but the chest remains loaded, powerfully stimulant expectorants may be given in aid of other remedies, such, for example, as the *decoction of senega* or the *misturæ ammoniaci* (FULLER). The patient throughout the treatment should remain in a room where the air is kept moist by the evaporation of boiling water from large flat dishes near the bed, and the temperature of the air should be maintained at 63° to 68° Fahr.

In chronic cases of bronchitis, especially in patients "who have made considerable progress in the journey of life, a lower tone of the system generally prevails, and a greater laxity of aërian membrane—particularly with excessive secretion, often muco-purulent"—characterizes such cases; and in them, after blistering, and perhaps poulticing the chest repeatedly with mustard poultices, the treatment in general should be more tonic. The *camphorated mixture* or *paregoric* and stimulant expectorant remedies are indicated for occasional but not for constant use. It was in these cases that the late Dr. Easton, Professor of Materia Medica in the University of Glasgow, so well showed the necessity of using remedies which will stimulate expectoration of the secretion which accumulates in the bronchial tubes. Such remedies are those which tend to invigorate the general system. Besides the selection of a beneficial climate, and the use of nourishing, easily-digested food, stimulating embrocations may be rubbed not only over the chest, both before and behind, but along the sides of the neck.

In the Meath Hospital at Dublin a liniment composed of the following ingredients is extensively employed by Drs. Graves and Stokes, and is recommended by them, as well as by Dr. Maclachlan:—

℞. *Spt. Terebinthinæ*, ℥iii.; *Acid. Acet.* ʒiv.; *Vitelli Ovi*, i.; *Aq. Rosæ*, ℥iiss.; *Ol. Limon.*, ʒi.; *misce.*

As a rubefacient, it is to be applied morning and evening, when it generally reddens the skin and produces small pimples. In several cases the secretion of the kidneys is increased during its use (MACLACHLAN).

Of tonic remedies, which are invaluable, my friend the late Professor Easton, from his extensive experience, put most reliance on the influence of *nux vomica, iron, and cinchona.* He was in the habit of prescribing them in the form of a syrup composed of the *phosphates of strychnia*, of *iron*, and of *quinia*, so that, in doses of a tea-spoonful three times a day, each dose shall contain the *thirty-second part of a grain of phosphate of strychnia*, and *one grain respectively of the phosphates of iron and quinia.* (Formula for the preparation of this compound, see page 86, *ante.*)

Combined with these remedies, the inhalation of *slightly* irritant vapours has a beneficial effect, as of *vinegar, turpentine, chlorine, and iodine.* Inhalation of vapour is often unsatisfactory, on account of the difficulty of getting an apparatus to hold a sufficiently large volume of boiling water. In the practice of my friend Dr. Fergus, of Glasgow, I have seen an admirable arrangement for inhalation, which overcomes this difficulty. It consists of a globular glass flask, about eight inches diameter, and six inches deep, having a wide mouth, a closely fitting cork, carrying an inlet tube which descends to the bottom of the vessel, and an outlet tube, to which a flexible mouth-piece is fixed. It is used as an Eastern uses a *nargilé* for smoking through water.*

Of the fetid gums, *ammoniac* in particular is a useful remedy. An emulsion of *gum* ammoniac in *diluted nitric* acid is a combination from which decided beneficial results are obtained. Professor Easton gave the following formula for its prescription—namely, *an hundred and twenty grains of the gum ammoniac dissolved in two fluid drachms of diluted nitric acid and twelve ounces of water*, compose a mixture of which an ounce may be given in gruel or barley-water three times a day (*Glasgow Med. Journal*, Oct. 1, 1863). On the contrary, it is often advantageous to administer astringent remedies, and one of the most useful is *tannic acid* in doses of one to three grains two or three times a day, as originally recommended by Dr. Alison, of Edinburgh; or the *oil of cubebs* to the extent of ten drops three or four times a day on a piece of sugar.

Acute bronchitis is very apt to be latent in old people, and to be complicated with gastric or gastro-enteric inflammation (MACLACHLAN). The treatment must therefore be modified to meet such a contingency. When there is tenderness at the pit of the stomach, nausea, and failure of the appetite, with the general condition approaching a typhoid state, in persons beyond the meridian of life, the pectoral symptoms are often apt to be marked by such associated disorder. The stimulants of food (by *enema* in the form of soup, without salt, if unable to be taken by the mouth) and of alcohol are the main remedies necessary from the very beginning. Abstinence cannot be enforced with safety. Leeches must be applied to the pained gastric region.

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* These inhalers are made of glass, so prepared as to receive boiling water without breaking; and are fitted up with tubes ready for use by Mr. Hugh Reid, chemist 423 Argyle Street, Glasgow.

When the disease is associated with a tendency to gout, *colchicum* must be given. "It allays the cough, promotes the flow of urine, keeps up a regular alvine discharge, and can be given much more generally than squills, because it does not produce that feverishness which results from the use of the latter remedy, and can therefore be employed where there is considerable fever" (FORBES). It requires to be administered with great caution in the aged and infirm (MACLACHLAN).

In the protracted bronchitic affections of the aged, diuretics are of great service; and the following formulæ are recommended by Drs. Maclachlan and Stokes, as well suited in a variety of cases of senile chronic catarrh:—

℞. *Decocti Senegæ*, *f*ʒvij.; *Potassæ Nitratis*, gr. iii.; *Tinct. Camph. Comp.* vel *Tinct. Conii*, ♏xx.; *Spiriti Ætheris Nitr.*, *f* ʒss.; *Oxymellis Scillæ*, *f* ʒss. *Fiat haustus ter die summendus* (MACLACHLAN).

℞. *Liq. Ammon. Acet.*, ʒiii.; *Potassæ Acetatis*, gr. xx.; *Aceti Scillæ*, *f*ʒss.; *Spirit. Æth. Nitr.*, *f*ʒss.; *Tinct. Camph. Co.*, ♏xx.; *Mist. Camph.* ʒvi.; *Syrupi Aurant.*, ʒi. (MACLACHLAN). *Fiat haustus ter die summendus.*

℞. *Decocti Senegæ*, ℥v.; *Tinct. Camph. Comp. Scillæ*, ā ā ʒii.; *Syrupi Tolut.*, ʒiv. *Sumat* ℥ss. vel. ʒi. *ter die* (STOKES, MACLACHLAN).

When gastric irritation prevails, the administration of balsams, gum resins, and terebinthine remedies must be suspended.

CASTS OF THE BRONCHIAL TUBES.

LATIN EQ., *Plasmata bronchiorum;* FRENCH EQ., *Bronchite pseudo-membraneuse;* GERMAN EQ., *Abdrücke der Bronchialröhren;* ITALIAN EQ., *Getti membranosi de'tubi bronchiali.*

Definition.—*A lymphy exudation, thrown out on the mucous surface of the bronchial tubes and their ramifications, forming false membranes or casts, which are sometimes expectorated, with catarrhal symptoms more or less acute. The disease happens at all ages; but more usually between twenty and fifty years of age, and generally associated with some constitutional disorder, such as rheumatism, gout, or scrofula, or with an aneurismal or other tumor pressing on the bronchi* (CLYMER).

Pathology.—Since Dr. Baillie first described and figured these tubular expectorated products, cases have been minutely described by many observers, and especially by Dr. T. Peacock, of St. Thomas's Hospital, in the *Transactions of the Pathological Society of London.* Figures of such casts are also to be seen in the description of a case published by him in the *Medical Times and Gazette* for 1854, p. 659. Such a form of bronchitis is known and described by the various names of *plastic bron-*

chitis, bronchitis crouposa, or *bronchité pseudo-membraneuse.* It is not a common form of bronchitis, and ought not to be confounded with cases of diphtheria or of croup. The following bibliographical notice of the disease is given by Dr. Clymer:—"The first recorded case of plastic bronchitis is in the *Acta Eruditorum,* Leipsic, 1682. Tulpius mentions it (1685); and also Clarke (*Phil. Trans.,* vol. xix., 1697). It has been described by Morgagni, Senac, Michaelis, Cheyne (*Edin. Med. and Surg. Journal,* 1803); Iliff (*London Med. Report,* vol. xviii., 1820); Starr (*Med. Gazette,* vol. xxv.); Cazeaux (*Bul. de la Soc. Anat.,* 1836); Nouat (*Archiv. Gen.,* 2 ème serie, 1837); Watson, Fauvel, 1840; Thore, fils (*Archiv. Gen.,* 4th serie, 1849); Fuller, Peacock (*Trans. of the Path. Soc. of London,* vol. v., 1854, and *Medical Times and Gazette,* vol. ii., 1854, p. 659); Michel Peter (*Gaz. Héb.,* 1863); Valleix (*Guide du Méd. Prat.,* 4th ed., 1866); and Dr. Stephen Rogers of New York (*Trans. of the State Med. Society of the State of New York,* 1866)."

Dr. Clymer has made an analysis of forty-four cases, and the following account is a summary of his results :—

The disease is most common in middle age, and happens oftener in males than in females. Morgagni mentions a case in a man seventy-eight years of age. The health in some instances is excellent until the sudden onset of the disorder; but generally it occurs in persons of delicate constitution who have suffered and are suffering from pulmonary disease, *inherited* strumous diathesis, a liability to cutaneous affections, to asthma, or hæmoptysis. Some had suffered from scurvy or had been gouty. It is not unfrequently associated with rheumatism, or been referred to the pressure of tumors, malignant or benign, upon the lung-tissue; or to the presence of aortic aneurism. [I once dissected a case in which an aneurism pressing on the trachea and lungs proved fatal (but not by rupture), in which the whole ramification of the bronchial tubes were occupied by an exudation of a reddish lymph which had formed a continuous coagulum of the consistence of jelly.]

The characteristic expectoration (sometimes called bronchial polypi) was described by Ruysch and Morgagni, and is figured by Cheselden in his *Anatomy* (1722).

Two classes or forms of the expectoration are described by Michælis and Cheyne (*Edin. Med. and Surg. Journal,* vol. iv., 1803).

(1.) *A moulded coagulum of blood.*

(2.) *A condensed, lammellated, solid,* or *tubular membrane;* and this second class belongs to that form of fibrinous exudations where the blood-corpuscles escape along with the *liquor sanguinis,* as in some cases of *pericarditis* and *pleurisy* (WATSON, PEACOCK).

The expectoration may be expelled in shreds, or in the form of oblong or rounded bodies, sometimes as large as a filbert; which, when macerated in water for a short time, gradually expand into a stem and branches; or they are expelled as cylindrical casts of the bronchi, varying in diameter from a crow-quill to a writing-pen or drawing-pencil, and from one to three or four inches in length, with small, divergent branches and minute terminal points, resembling some vegetable roots and their radicles or rootlets. As many as ten distinct subdivisions have been seen. They come from as low down as the second, third, and fourth bronchial ramifications. They are of a dull white colour, or occasionally brownish, from the admixture of blood. These casts seem to have been deposited or exuded in successive layers; being composed of tough, concentric, fibro-membranous laminæ, between which a fine probe can be passed. The main stem is sometimes solid and sometimes hollow; or portions may be solid and others hollow.

In the case described by Dr. Stephen Rogers, of New York (of which a figure is given by Dr. Clymer), the number of casts expectorated was from one to half a dozen a day, through a period of about three and a half months. In the opaque semi-fluid mass the arborescent character of the casts was quite distinct, and was thoroughly brought out by an admixture with a gentle agitation in water. They were always cylindrical or solid, and varied from $\frac{1}{8}$th to $\frac{3}{8}$ths of an inch in diameter at the base of the cast, the extreme length of some of the larger ones reaching nearly four inches in length. The case was one of malignant disease of the lungs.

Microscopically, these casts are of uniform structure, of parallel and nearly straight fibres, which are the edges of laminæ closely and concentrically arranged and intermixed with numerous rounded bodies about the size of blood-corpuscles. Acetic acid produces expansion and partial solution of the laminæ, but has no evident action upon the corpuscles. Towards the termination of the minutest ramifications of the casts there are found compound granular cells mixed with irregular clusters of oil globules and ovoid bodies, containing pigmentary matter (Bristowe, *Trans. Path. Soc.*, vol. v., 1864), showing that the lymphy exudation has commenced to undergo degenerative changes.

The nature of the disorder is to be sought for in some peculiarity of constitution—a specific morbid process which yet requires investigation.

Symptoms—Are those of slight catarrh, which do not attract attention till the appearance of the peculiar sputa. Sometimes the symptoms are more acute, like those of pneumonia or bronchitis. Hæmoptysis may also be the initial symptom.

The symptoms which immediately precede the characteristic expectoration are:—(1.) A sense of constriction about the chest, with short breath, and a dry, hard, paroxysmal cough, which may last for several days. (2.) Breathlessness suddenly becomes urgent and alarming, the lips are blue and the face swollen and livid, the extremities cold and discoloured. (3.) A fit of coughing, severe and strangling, brings forth the membrane usually along with the white, glairy, adhesive sputa of simple bronchitis, or there may be a little blood.

Relief is immediate, and lasts till there is a fresh accumulation and another exacerbation, followed by the same phenomena of detachment, expulsion, and relief. Several such paroxysms may occur in the twenty-four hours.

"In some cases the expectoration of plastic membrane ceases after a few days, and recovery is rapid and good. In others, the acute symptoms abate, but false membrane continues to be occasionally expelled, after fits of cough and breathlessness, for several weeks, with intervals of comfort, and, in some instances, of apparent good health. The coughing paroxysms are often brought on by excitement or exertion. Cases are recorded where this disorder has lasted during one, two, three, or more years, either continuously or at certain seasons. One instance is related in which there was catarrhal fever for four successive winters, each attack lasting five weeks, and ending in the expulsion of false membranes, the patient during the intervals being quite well. In Dr. Fuller's case there was a recurrence of catarrhous symptoms every winter for eleven years, accompanied by the expectoration of small pieces of plastic matter; after which time the disease was not limited to the winter months, but followed any exposure to cold, or damp, or change of weather. Relapses may happen. In the chronic form the general appearance is unhealthy. The subjects of this disorder are habitually short-breathed and pallid, with livid cheeks and lips, and incurvated nails" (CLYMER).

"**Prognosis** depends upon circumstances, and upon the presence and nature of any complicated pulmonary disorder. In thirty-four cases analyzed, twenty seemed to have ultimately recovered perfect health, and ten to have died sooner or later;—in ten it is stated that false membranes continued to be expectorated at intervals for a long period. The acute form of plastic bronchitis runs its course rapidly to a happy or fatal issue; and in adults, however alarming or urgent the symptoms, usually ends in recovery, particularly when the false membrane is freely expectorated; or it may pass into chronic bronchitis, with occasional exacerbations from fresh cold, from time to time, which subside after membranous expectoration. When the general and local symptoms are slight at the outset, the disorder is apt to be of longer duration."

"**Diagnosis.**—The expectoration of plastic membrane in rounded pellets or cylindrical casts is really the only distinctive symptom of this disorder. The diacritic signs between plastic and capillary bronchitis are usually sufficiently marked, the former being generally localized in one lung, and one region, while the latter is symmetrical, and shows a great tendency to extend; the subcrepitant rhonchus, when met with in plastic bronchitis, is limited; in capillary it is diffused From pseudomembranous croup and diphtheria it is distinguished by the local symptoms and physical, as well as by the character of the false membranes expelled" (CLYMER).

Treatment.—In the acute form, *muriate of ammonia* and the alkalies and *iodide of potassium* may be given, with an occasional emetic, and inhalation of the vapour of hot water. Dr. Clymer recommends that the Turkish bath should be tried. The patient should also be carefully protected against damp and sudden changes of weather.

DILATATION OF THE BRONCHI.

LATIN EQ., *Dilatatio;* FRENCH EQ., *Dilatation;* GERMAN EQ., *Erweiterung;* ITALIAN EQ., *Dilatazione.*

Definition.—*A cylindrical, fusiform, or saccular dilatation of a bronchial tube, or of several tubes, at one or more points, or through a considerable portion of the second, third, and fourth divisions of the air-tubes in one or more lobes, generally the lower and middle, with atrophy of the muscular and elastic coats. The expectoration is muco-purulent and fœtid.*

Pathology.—True bronchiectasis may arise quite independently of all other pulmonary affections; but there are several minor varieties which result from other changes in the lungs, such as from *hooping-cough; suffocative capillary bronchitis; stricture of bronchi; long-standing indurations of lung-substance, tubercular or inflammatory; the remains of chronic tubercular cavities,* or *abscesses in the lung-tissue.*

The most important forms of *true bronchiectasis* are—(1.) *The general or uniform,* in which there is a cylindrical or fusiform dilatation of a tube, or of several tubes throughout considerable lengths of their extent; (2.) *The saccular,* or *ampullary,* in which there occurs an abrupt dilatation of a tube at a particular point, or at several points. The disease is not uncommon; and is of interest and importance on account of its alliance with forms of pulmonary consumption.

When dilatations exist in neighbouring bronchi, communications may become established between them, and to such an extent that several bronchial tubes open into one common cavity. Wasting of the muscular and elastic coats is a usual result, and there is degeneration of their tissue. Thus the dilated tubes open into each other without any truly ulcerative process, as shown by Dr. T. G. Stewart, from whose excellent description of "*Dilatation of the Bronchi,*" in the *Edin. Med. Journal* for July, 1867, this account of *Bronchiectasis* is taken. In a small number of cases ulceration of the bronchial membrane occurs from within outwards, frequently associated with dilatation of the bronchial tubes, and constituting characteristic bronchial abscesses. Copious yellow or inspissated mucus, sometimes with casts of tubes, and often very fetid, exists in the dilated tubes, associated with crystalline fats and fungi. In some cases the tissue of the lung is condensed, from cirrhosis or fibroid degeneration of the lung, which may proceed to more intense induration, ulceration, and even gangrene. The site of *bronchiectasis* is usually the lower lobe and the middle lobe of the right lung. It occurs towards the apices sometimes. Usually it affects many bronchi and occurs in both lungs. The essential element of the lesion in bronchiectasis is atrophy of the bronchial walls, which rapidly yield to the pressure of air. The enfeebled and dilating condition of the bronchi favour the accumulation of the mucus secreted by the mucous membrane, which, accumulating and undergoing decomposition in the dilated cavities, leads to inflammation and the formation of villous processes, to increase of connective tissue, and to further consolidation of surrounding lung.

The disease is to be regarded as a lesion following "a cold" and catarrhous affections, collapse of the pulmonary lobules, pneumonia, and pleurisy. It is met with at all ages, but is most frequent about and after forty. Pulmonary emphysema is found in a large proportion of the cases.

Symptoms and Clinical History.—The disease comes on insidiously; but gradually symptoms of bronchitis become well marked. Breath and sputum become fetid, and general health is impaired. Decomposition of the secretions is followed by lung consolidation, ulceration, abscess, or gangrene. Perforation of the pleura, empyema, or pneumothorax may prove fatal; or death may result from exhaustion due to the constant discharge of the sputum. A peculiar febrile disturbance, resembling *septicæmia,* may also terminate life. Unfortunately, the tendency is to a fatal result; but recovery may take place—(1.) *From cretification of the contents of the dilated tubes, and the conversion of their walls into a sort of fibrous capsule;* (2.) *From penetration of the pleura and thoracic parietes, and discharge of the contents outwards.* Dr. Stewart considers the disease as probably hereditary and constitutional.

The most characteristic symptoms are *the odour of the breath,* the *characters of the sputum,* and the *cough.* The fetor of the breath is sometimes distinct and different from that of gangrene, and is not present in all cases, and not till decomposition of the bronchial secretion has taken place. *Blood* is sometimes expectorated with the sputum, sometimes in large quantities, as in phthisis, at other times merely in streaks over the expectoration. *Cough* is frequent, and occurs in paroxysms, but is moist, soft, and usually quite painless; and after the fit of coughing, large quantities of the sputum are brought up with difficulty. Exertion induces dyspnœa.

The physical signs depend on the form, size, and extent of the dilatations. *Inspection* may show depression over the site of the affected lung. *The application of the hand* may detect increased fremitus. *Auscultation* gives varied sounds—cavernous moist râles, sometimes even gurgling—and the site of these sounds aids to distinguish them from those of phthisis, also the history and progress of the case. Auscultation must be frequently repeated.

Treatment.—Opiates, to relieve cough; balsamic remedies (*tolu, tar, turpentine, copaiba, cubebs*), and astringents, like *catechu* or *rhatany*, with the use of counter-irritants and the inhalation of variously medicated vapours, are all useful aids in ameliorating the condition of the patient. *Muriate of ammonia* and the alkalies are also to be recommended. Inhalation of disinfectants, capable of being so used, such as *creosote, carbolic acid, sulphur vapour, turpentine,* and the like, are of special service.

(SPASMODIC) ASTHMA.

LATIN EQ., *Asthma;* FRENCH EQ., *Asthme;* GERMAN EQ., *Asthma;*
ITALIAN EQ., *Asma.*

Definition.—*A disease which culminates in paroxysmal attacks of difficult breathing, of longer or shorter duration. The dyspnœa seems to be immediately dependent on more or less extensive contraction of the smaller bronchi, and due to tonic spasm of their circular fibres. The breathing is accompanied by a wheezing sound, a sense of constriction in the thorax, great anxietas, and a difficult cough. The attack usually terminates by the expectoration of a quantity of mucus from the lungs, which varies considerably in appearance and in amount. In some instances the mucus is thick and heavy, in others it is light and frothy, whilst in the severer forms of the disease there may be only a few dark pellets coughed up before relief is obtained* (PRIDHAM). *In the hours immediately succeeding the fit a remarkable diminution of the urea and chloride of sodium may occur, which would imply a considerable arrest either of formation or elimination, probably the former* (RINGER, PARKES); *or to the starvation that is generally enforced at that time* (SALTER).

Pathology.—Few diseases have been the subject of greater doubts and differences of opinion as to its nature than asthma. Not unfrequently it has been confounded with dyspnœa; and the terms *dyspnœa*, *asthma*, and *orthopnœa* were formerly employed to designate different degrees of difficulty of breathing. Their signification must now be much more precisely defined. *Dyspnœa* is a term which is now used to denote difficulty of breathing generally, and may be due to various causes. The significance of *asthma* is defined above, and its pathology is about to be considered; while the term *orthopnœa* signifies that great difficulty of breathing in which the patient is incapable of respiring except in the erect posture.

When asthma has once expressed itself, it seldom fails of recurring, though the intervals between the paroxysms are of very uncertain duration. In severe cases the fits will return periodically every ten days or a fortnight; and in still more severe cases they will recur every night or early morning, at exactly the same hour.

It has been observed to recur in females just after the menstrual discharge, or immediately before it. It is also apt to recur in the spring and autumn, and after exposure to cold and wet. The disease is not only paroxysmal, but often periodic—by days, weeks, months, or even years. Diurnal asthma is very common, especially when associated with chronic bronchitis, heart disease, and impaired digestion. Asthma occurring once a year is usually a winter affection complicated with bronchitis.

The stomach and bowels are extremely liable to disorder in asthmatic persons; colic-like pains, flatulence, loss of appetite, and an irregular state of the bowels, are not uncommon.

There appears to be no period of life at which asthma may not make its appearance—from the earliest infancy to old age. In thirty-eight cases noted by the late Dr. Hyde Salter—himself a great sufferer—the first access of the paroxysm occurred in *seven* during the first year of life. In one of these, symptoms of asthma were noticed at *fourteen days* old; in another at *twenty-eight* days; in another at *three months;* in another at *one year;* and in three "during the first year." Many of the best marked and purest asthmatic cases date from early infancy—so early in some that it were difficult to say the disease was not truly congenital. It is evidently very often dependent upon hereditary transmission and conformation; and in all such cases of its early development there was a history of its inheritance (HYDE SALTER). Mr. T. L. Pridham, of Bideford, has traced its hereditary origin in nine out of ten cases, and relates a case of asthma which commenced as early as seven years of age. Out of *thirty-five* cases in which Dr. Salter noted this circumstance, he found distinct traces of inheritance in *fourteen.* The melancholic temperaments, the sanguineo-melancholic, the nervous, and the irritable, are most liable to the affection; and the male sex is much more disposed to it than the female (WOOD). According to the experience of Mr. Pridham, about 80 *per cent.* of the cases are men.

There are good reasons for regarding asthma as a general or constitutional disease; and it is believed by not a few to be connected with the gouty or rheumatic diathesis. The disposition of the attacks to recur at distant but gradually diminishing intervals; the division of each attack into nightly paroxysms, with marked remissions during the day; the duration of the earliest fits for several days or a week, are all circumstances which point to the constitutional nature of the affection. Many of the cases recorded by Mr. Pridham inherited gout; and in some instances both diseases could be traced in the family; and in more than one instance, where gout and asthma had prevailed in previous generations, there were alternate attacks of asthma and of gout. He found that when women were the subjects of asthma, gout prevailed in their families in a larger proportion than in men.

The best descriptions of the phenomena of asthma have been given by medical men who have themselves suffered from it—for example, Dr. Bree and Sir John Floyer, both of whom were asthmatics (GOOD). Asthmatic patients generally live to a good old age. The lungs undergo dilatation of the air-cells, which dilatation does not much interfere with their normal action when free from attack. It produces emphysema in the same way that bronchitis does, and leads to hypertrophy and dilatation of the right side of the heart. In those cases of asthma which have been described as dyspeptic, the powers of digestion are insufficient to assimilate the food taken, and such patients can never with impunity eat and drink as other people do; and wherever the disease has occurred in any member of a family for one, two, or even three generations back, such exciting causes as imprudence in eating or drinking, an attack of bronchitis or influenza, atmospheric influences, certain odours, mental excitement, and the like, may at any time or period of life bring to light this peculiar disease; but unless in constitutions predisposed to the disease, such exciting causes have no influence upon its development. Asthmatic people, for the most part, are said to be gifted with great energy, and talents far beyond the lot of ordinary people. They are generally courageous and resolute; and those in humble life possess intellectual attainments far beyond their station; and all generally excel in whatever subject of study they are disposed to follow (PRIDHAM). The experience of Dr. Hyde Salter, however, did not bear out this statement. A knowledge of the state of the blood and of the physiology of digestion, embracing a knowledge of the amount of the urinary and other excreta, and especially of the peculiar expectoration, in relation to diet and temperature, is of great importance to be known in cases of asthma. Dr. Sidney Ringer, the Professor of Therapeutics in University College, has made one series of accurate observations relative to the urinary excreta in a case of asthma. He found in the hours immediately succeeding the fit that a remarkable diminution of the urea and chloride of sodium occurred, which implied a considerable arrest either of formation or of elimination, probably the former (PARKES, l. c., p. 319). The urine at the commencement of an attack is generally abundant and colourless, like what is known by the name of "nervous urine;" later in the attack it is often extremely dark and scanty, and is sometimes almost suppressed (HYDE SALTER).

Mr. Pridham observed in one of his cases that the urine was suspended during an attack, on the subsidence of which there occurred an enormous secretion of it, of a dark colour, and loaded with lithates.

It has been clearly shown that the dyspnœa of the asthmatic paroxysm is due to spasmodic contraction of the bronchial tubes; that the phenomena of the paroxysm are in a great measure those of an excito-motory kind—in other words, due to reflex action (Dr. Hyde Salter).

The exciting causes of the paroxysms are mainly due to fatigue and physical exhaustion—sudden or violent mental emotion—certain conditions of the digestive organs—gastric irritation—the irritation of a loaded rectum—irritation of an eruption on the skin and its sudden subsidence—the irritation of certain substances and articles of food, such as cheese, nuts, almonds, and raisins, sweet things generally, salted meats, condiments, preserved and highly seasoned foods, fermented liquors, especially malt liquors, and sweet wines.

Forms of Asthma have been variously described under the following names:—*Peptic* or *dyspeptic asthma, congestive asthma, hay asthma, hysteric asthma,* and *spasmodic asthma;* but seeing that all true asthma is spasmodic, the classification proposed by the late Dr. Hyde Salter is perhaps the best, inasmuch as it suggests the necessity for examining into the various influences which act on the nervous system in each case, and enables the physician to arrange the basis of treatment accordingly. It is as follows:—

Class I. Those cases in which the provocatives of the attack are manifest, in which the lungs seem to be mainly concerned, the source of irritation being applied to them, as some material respired which provokes the bronchial tubes to spasms by direct contact with their mucous surface (*e. g.*, asthma from fog, smoke, fumes of various kinds, *ipecacuanha powder*, or that of hay; from animal emanations; from certain atmospheres; and, lastly, from blood-poisoning, as after beer, wine, and sweets).

Class II. Those cases which acknowledge a reflex source of their development. Of these, five varieties are easily indicated:—(1.) Those in which the asthma follows an error in diet, or supervenes on a full meal; (2.) Those in which the source of irritation is transmitted from a loaded rectum or from uterine irritation; (3.) Those in which it arises from the sudden application of cold; (4.) Emotional asthma; (5.) Periodic asthma.

Class III. Asthma complicating bronchitis, heart disease, or pulmonary emphysema.

Symptoms.—On inspecting the chest of a patient labouring under a severe paroxysm of asthma the whole upper part seems almost motionless, while the inferior portions are acting within a very confined range. All the muscles passing from the head to the shoulders, clavicles, and ribs are rigid. The abdominal muscles, however, act most powerfully to increase the capacity of the chest, and its walls are kept fixed in a condition of extreme inspiration. The chest is enlarged in every way, the diaphragm descends, the abdomen seems fuller, and its girth is increased. The stethoscope teaches us that the whole of the lungs, but particularly the posterior lungs, are labouring with a loud and deep sibilous sonorous wheeze, accompanied with a mucous rattle, sometimes loudest on inspiration and sometimes on expiration. No respiratory murmur exists. Dry tube sounds alone are heard—rhonchus and sibilus of every variety, note, and pitch. There is complete stagnation of air in the chest. The sounds are so small that they seem to indicate spasmodic constriction of the smaller tubes; and the universal diffusion of the sound shows that the constrictions are universal over the smaller tubes. These spasms may also be observed to be constantly changing their place, disappearing in one place and making their appearance in another, so that the sounds are continually changing their character and their site. Percussion shows that the lungs are distended with air; and should an air-cell have burst, a rubbing sound will be heard, denoting the effusion of air into the cellular substance of the lung. As the fit subsides, the respiration becomes puerile, and by degrees the breathing returns to its usual state. In fatal cases the respiration becomes tracheal, slight hæmorrhage perhaps takes place, and after a severe struggle the patient dies; but this is an event so extremely rare that, from Dr. Hyde Salter's very extensive experience of this disease, he believed that death never takes place immediately from uncomplicated asthma.

The duration of the fit varies. In some cases it lasts a few minutes, in others two or three hours, in others the whole night, in others three or four days, and in others as many weeks.

When the expressions of the disease are fully marked, the appearance of an asthmatic person is very characteristic. The countenance often bears the signs of distress. The shoulders are more or less elevated. The stomach is apt to be greatly distended after eating; the tongue becomes coated, and there is a tendency to fissures in it; the eyes red and prominent. Emaciation generally progresses, and there is inability to rest horizontally in bed, or to walk up a hill. The secretions from the bowels are more or less abnormal, and the urine passed generally shows a variable deposit. In extremely severe cases, when remedial measures are not taken to subdue the constitutional affection, the nights may be passed by the patient in a state of great distress. Unable to lie down in bed, his paroxysms may sometimes be so severe that he almost anticipates death before morning—were it not that he becomes accustomed to the severe nature of the symptoms—till a copious heavy expectoration is with difficulty thrown off from the lungs; and as the day advances he becomes somewhat relieved, although it may be passed still in great discomfort. Asthmatic patients are generally large feeders, although the desire for food may not be remarkable, believing that it is necessary to eat and drink well, in order to sustain the strength to encounter the paroxysms of the disease. In some the difficulty of breathing is constant, and always worst after a meal; and, as a rule, they can never, without aggravation of the disease, eat and drink as other people.

The pulse increases in frequency towards night, and subsides in the morning.

Many premonitory phenomena indicate the approach of a paroxysm; but the precursory symptoms are liable to great variation in different persons, according to the proximate cause of the paroxysm. Some patients suffer from fearful headaches, the approach of which they dread; and the heart labours with so much palpitation, and such irregularity of action, that rupture of a blood-vessel seems imminent. Eruptions on the skin sometimes lessen, and even disappear. There may be also some warning during the night of the immediate approach of an attack, in the shape of huskiness of the throat; and during the middle of the night, or towards early morning, the patient is awakened by an oppression which renders it impossible for him to lie down again. In a short time the paroxysm gains strength, and the patient breathes as it were by jerks, each aspiration being accompanied by a spasmodic effort, which seems as though it would burst open the chest. The contractions of the muscles in the neck and below the ribs in front of the chest are very great, and often at the same time most painful. The attacks are not of equal violence at all times.

The majority of asthmatics know that an attack is coming on by certain feelings in themselves, or by certain conditions of the system, of which they are aware. The precursory symptoms generally show themselves on the night previous to the attack, or sometimes for two nights before it, or even for a longer time. Extreme drowsiness and sleepiness are common precursory phenomena which indicate the approach of a paroxysm—the commencement of the nervous condition of which the succeeding respiratory phenomena are the more complete development; and such precursory phenomena must be looked upon as an integral part of the paroxysm (HYDE SALTER).

Extreme wakefulness, unusual mental activity, and buoyancy of spirits, constitute another set of premonitory phenomena seen in some other asthmatics; and Dr. Salter mentions a case in which ophthalmia always ushered in the paroxysm of asthma. At other times, and with other patients, the premonitory symptoms are connected with the stomach, and consist especially of loss of appetite, flatulence, costiveness, and certain peculiar uneasy sensations in the epigastrium.

The time at which a paroxysm commences is almost invariably in the early morning, from three to six o'clock. In some the usual time is the evening, just after getting into bed, and before going to sleep. Even in such cases as night watchmen, who turn day into night and night into day, though the ordinary times of sleeping and waking were transposed, the paroxysm came on at the usual time—from five to six in the morning, towards the end of the vigil, when the patient was up and awake (DR. SALTER).

Profuse *diuresis* not unfrequently attends the first stage of a paroxysm of asthma. The urine is then a pale limpid water, exactly like the urine of hysteria. This abundant watery secretion comes on soon after the paroxysm commences, and generally lasts for the first three or four hours, when it ceases altogether.

Neuralgic pain constitutes another early symptom, in the form of deep-seated, aching, constant, and wearying pains in the limbs, joints, or testicles.

The characteristic wheezing of a commencing paroxysm generally commences while the patient is yet asleep; and as the difficulty of breathing increases, he gradually or partially awakes, and sits up in bed "in a miserable half-consciousness of his condition." A temporary abatement occurs, and sleep may again overpower him, to be again awoke, and again to sit up. By and by the struggle ceases between sleep and the full expression of the paroxysm. The *dyspnœa* increases, so that he can lie back no more. He throws himself forwards, plants his elbows on his knees, and, with fixed head and elevated shoulders, labours for his breath like a dying man. A most distressing spectacle is now presented by the asthmatic. If he moves at all, it is with the greatest difficulty, creeping by stages from one piece of furniture to another. Most commonly he sits fixed in a chair, immovable, unable to speak, or even perhaps to move his head in answer to questions. His back is rounded, his gait stooping. His chest, back, shoulders, and head are fixed; and, when he looks from object to object, he merely turns his eyes like a person with a stiff neck. His shoulders are raised almost to his ears, and his head thrown back and buried between them. The better to raise his shoulders, and to spare muscular effort, he fixes his elbows on the arms of the chair; or he plants his hands on his knees; or he leans forward on a table; or sits across a chair, and leans over the back of it; or he stands grasping the back of a chair, and throwing his weight upon it. In this latter attitude Dr. Salter has known a patient stand for two days and nights unable to move. Sometimes the patient may lean against a chest of drawers or some piece of furniture sufficiently high to rest his elbows upon in a standing position. At every breath the head is thrown back, the shoulders still more raised, and the mouth a little opened, with a gasping movement. The expression is anxious and distressing. The eyes are wide opened, strained, turgid, and suffused. The face is pallid; and if the dyspnœa is extreme and prolonged, it becomes slightly cyanotic. The labour of breathing is so great that beads of perspiration stand on the forehead, or even run down in drops upon the face, which the attendant must constantly wipe off; for the patient is so engrossed with his sufferings and the labour of breathing that he is almost unconscious of what is going on around him; or he is impatient and intolerant of the assiduities of those who are in vain trying to give him some relief.

If the bronchial spasm is protracted and intense, the body-temperature falls: the oxygenation of the blood is so imperfectly performed, from the sparing supply of air, that it is inadequate to the maintenance of the normal temperature. The extremities especially get cold, blue, and shrunken; but while the temperature is thus depressed the perspiration may be profuse. This union of coldness and sweat, combined with the duskiness and pallor of the skin, gives to the asthmatic so much the appearance of a dying man that sometimes even the initiated may fear that death is impending.

The pulse during the paroxysm is always small; and small and feeble in proportion to the intensity of the dyspnœa—due to pulmonary capillary arrest; and immediately the paroxysm yields, the pulse begins to resume its normal volume.

Itching under the chin is a common symptom of an approaching paroxysm of asthma. This itching is incessant, of an indefinite creeping character, and scratching does not relieve it. It often extends over the sternum and between the shoulders. It appears the moment the first tightness of breathing is felt, and subsides when the paroxysm has become confirmed.

The most exhaustive monograph on this disease is that by the late Dr. Hyde Salter, F.R.S., Physician to the Charing Cross Hospital; and the reader desirous of more full details will do well to consult his work on *Asthma: its Pathology and Treatment.*

Diagnosis.—The preceding account of the pathology and symptoms of asthma renders it obvious that it may not easily be confounded with any disease of the chest or larynx, if auscultation and percussion are carefully attended to, and with a due regard to the history of the case. The sudden attacks of the paroxysms, the short periods of their duration, the violence of the symptoms at the time, their returning after intervals of comparative ease and of tolerable health, are sufficient to characterize the disease.

It is only when asthma complicates other diseases that its diagnosis may be obscure and its treatment uncertain.

The diseases with which it has been confounded are,—(1.) Spasmodic affections of the larynx; (2.) Severe cases of sudden and *acute bronchitis;* (3). *Angina pectoris;* (4.) *Hydrothorax.*

The disease is also sometimes associated with the development of lesions of the heart and great vessels, and ultimately leads to them.

The character of the *dyspnœa in asthma* is also quite peculiar. It is unlike the dyspnœa of heart disease, or of that of bronchitis, or of that of emphysema; the distinctive features of each of which are as follow (Salter):—

Heart dyspnœa is intolerant of the slightest exertion, or of the recumbent position; and sitting up or stillness may cure for the time the most violent paroxysm: the breathing, too, of heart dyspnœa has a panting and gasping character, and not the wheezing labouring character of asthma.

Bronchitic dyspnœa is short, crepitous, and accompanied with cough.

The *dyspnœa of asthma* is often long-drawn, dry, and without cough; it gives the most positive evidence of narrowing of the air-passages, and is of such a nature as to shut off the air-supply. The wheezing, or shrill sibilant whistle, is positive evidence of bronchial contraction, which is ever changing its place. Spasmodic stricture of the minute air-tubes thus explains, as Dr. Hyde Salter clearly demonstrates, the sudden access and departure of the dyspnœa in asthma.

The *dyspnœa of emphysema* is abiding, varies but little, and has no wheeze.

The Treatment of Asthma comprises what should be done during the fit, and what should be done during the intervals, with a view to correct the constitutional state which every now and then culminates in a paroxysm of asthma.

When the patient is labouring under a fit of either of the forms of asthma, our efforts must be directed to tranquillize his suffering and shorten the attack; but so capricious is this disease that what will benefit the patient in one attack may be of little use in another. As a general rule, however, any exciting cause actually present and in operation must be removed: an undigested meal, or constipation, must be got rid of—relieved by an emetic in the one case, or by an enema in the other; the patient should be supported by strictly tonic regimen; and *camphor mixture,* to the extent of about an ounce and a half, combined with a drachm of the *spirit of nitrous ether* and some *morphia,* may be given every hour, or every two hours, for a short time. If the head should be affected by the opium, some milder narcotic should be substituted, as *tincture of hyoscyamus,* to the extent of about fifteen drops for each dose. In other cases, or in other attacks, *asafœtida, castor, musk,* or *hydrocyanic acid,* to the extent of ♏iij. every six hours, may be substituted. Again, if the fit should occur after a hearty meal, and after an emetic has been given to empty the stomach, the *tincture of rhubarb* or the *sulphate of magnesia* should be continued in repeated small doses. If the attack be long, arrow-root or sago, with small quantities of wine or brandy, should be given to support the patient under his laborious and exhausting sufferings. *Ipecacuanha, tartar emetic,* and *tobacco* are the drugs which most rapidly relax spasm as direct depressants. There is, however, great danger in the use of the latter, from unmanageable and dangerous collapse. And tobacco ought never to be indulged in by the asthmatic, except as an agent in the cure of his disease; for then only can he look to it for relief. *Ipecacuanha* is the most manageable of these remedies, and ought to be given in a dose of twenty grains at the onset of the paroxysm. The tobacco should be smoked from a pipe.

The feelings of the sufferer should be consulted as to the temperature to which he should be exposed during the paroxysm. Where there is organic lesion of the heart and large vessels, the fresh air is extremely grateful and reviving, its coldness giving power to the circulating organs, and, by lowering the temperature of the body, enables the patient to live on a smaller quantity of oxygen. It is on this principle that the dog, barbarously asphyxiated by the effluvium of the Grotto del Cane, for the amusement of travellers, is thrown into the water, where he is able to breathe at the temperature of the water, when he would have died at the temperature of the atmosphere. The toad, also, when cooled down, will live for an incredible length of time encased in plaster of Paris; but if his body has a high temperature the experiment is soon fatal. On the contrary, when the paroxym is purely a spasm of the bronchial tubes, warmth, by relaxing the spasm of the bronchial tubes at their ultimate divisions, is generally more useful than cold.

But it is the treatment during the interval which is all-important; so much so, that few cases will be found of true spasmodic asthma which are not entirely under the control of well-regulated dietetic management. "More is to be done for *asthmatic patients* on the side of the stomach than in any other direction;" and by many observing and thoughtful physicians dietetic treatment is regarded as the only certain treatment of asthma. Mr. Pridham, of Bideford, in Devonshire, has been very successful in the management of asthmatic cases, by extremely strict dietetic treatment and sedatives during the intervals of the paroxysms (*Brit. Med. Journal,* June 9 to December 29, 1860). His plan of treatment is somewhat as follows:—The secretions from the bowels are first of all to be corrected by the following pill, at bedtime, followed by a saline aperient in the morning:—

℞. *Pilulæ Aloes cum Myrrhâ*, gr. iii. ; *Pilulæ Hydrargyri*, gr. i. ; *Extracti Taraxaci*, gr. ii. ; *Extracti Strammonii*, gr. ss.; M. Fiant. pil. ii.

Or giving, every alternate night, in the form of a pill,—

℞. *Pil. Hyd.*, gr. iv. ; *Pulv. Ipecac.*, gr. i.

And on the following morning,—

Mist. Sennæ comp., ℥i. ; *Bicarbonatis Magnesiæ*, gr. x. ; *Bicarbonatis Sodæ*, gr. viii. ; and during the day small doses of Compound Rhubarb Powder.

After having thus attended to the general secretions for about ten days, the strict dietary system is to be commenced.

The diet must be regularly weighed out, and adhered to with the greatest strictness, the hours of meals being most rigidly fixed as follow:—

Breakfast at *eight* A.M., to consist of half a pint of green tea or coffee, with a little cream, and two ounces of dry stale bread.

Dinner at *one* P.M., to consist of two ounces of fresh beef or mutton, without fat or skin, and two ounces of dry stale bread or well-boiled rice; three hours *after* dinner (not sooner) half a pint of weak brandy and water, or whisky and water, or dry sherry and water, may be taken, or toast-water *ad libitum.*

Supper at *seven* P.M., to consist of two ounces of meat as before with two ounces of dry stale bread.

The patient is not to be allowed to drink any fluid whatever within one hour *before* his dinner or supper, and not until three hours after either of these meals. At other times he is not limited as to drinks, otherwise than that all malt liquors are to be prohibited. *Soda* or *seltzer*-water may be indulged in at other times when thirsty.

With this dietetic treatment sedatives are to be given as follow:—

Three grains of the *Extract of Conium* are to be taken four times a day—namely, at the hours of seven, twelve, five, and ten,—the dose to be gradually increased to five grains four times a day. To each of these pills a *fourth* of a grain of the *Extract of Indian Hemp* may be added, which may be gradually increased to one grain in each dose.

Under this treatment in a few days the distressing symptoms may be expected to subside; and after the regimen has been strictly persevered in for at least a month, two ounces more of meat may be permitted, if digestion is found to be sufficient. The stools must be repeatedly seen by the physician, and the stomach must not have more to do than it can accomplish. The powers of digestion are known to be recovering when the stomach craves for food as the hour of nourishment arrives. If flesh is gained, strength improves; and while the tongue cleans, the appetite improves, the distension of the stomach lessens, and there is sufficient evidence that the powers of digestion are recovering. Great encouragement is then given for the physician and for the patient to follow up the line of treatment which is here indicated. The patient ought also to be able to sleep six or seven hours at a time, and to lie in bed all night. If these results follow, the ultimate cure of the disease may be looked for; but it may at the same time be taken for granted that the asthmatic can never with impunity eat and drink as other people. It is only by the exercise of such self-denial as is implied in the carrying out of such instructions that the patient has it in his own power to live a life of comparative ease and comfort. Many such patients who are not possessed of such resolution, self-denial, and strength of mind, will say such dieting does not suit their constitution, and that consequently they cannot or will not persevere; but no trial of the remedy can be considered sufficient which does not embrace a period of at least six months, the physician taking care to ascertain—(1.) The weight of the patient, his age and height, before commencing any treatment; (2.) The state of his excreta, and the amount of the urinary elements—especially the urea, the uric acid, and the like; (3.) During the course of the treatment, at stated times, such physiological information ought to be regularly obtained.

It is confessedly difficult to persuade many people to live so abstemiously, for many cannot control their appetite; or they believe that in so limiting themselves in regard to diet they will injure their constitution.

The abnormal ravenousness of appetite it is therefore necessary to subdue by sedatives other than opiates. Asthmatics are generally dyspeptics; and as a result of the experience of Dr. H. Salter, the most simple rule regarding the diet is:—Let no food be taken after such a time in the day as will allow digestion being completed or the stomach empty before going to bed. The time when the last solid food should be taken will depend upon what the bedtime is. If ten, or half-past, then three or four should be the dinner hour, after which no more solid food should be taken.

Dr. Salter's dietary in cases of asthma would be sometimes as follows:—*Breakfast.*—A breakfast cup of bread and milk, an egg, or a mutton chop, or some cold chicken or game. Tea is better than coffee, and milk and water better than either. *Dinner* (not before two or four o'clock).—Mutton ought to be the staple diet; beef or lamb rarely, pork or veal never. Succulent vegetable or potato may be eaten, and a little farinaceous pudding or stewed fruit, or fruit out of a tart, should conclude the dinner. Water is the best fluid to drink, and there should be no cheese and no dessert.

The quantity of food eaten should be small, and therefore highly nutritious, extremely digestible, and of the simplest and plainest kind.

Open air exercise must be freely taken, but not within three hours after eating animal food, and the exercise must be always short of fatigue. The greatest punctuality is necessary to be attended to as regards the taking of food as well as medicine; and the bowels should be caused to act immediately after breakfast, either naturally or by means of an enema. In truth, success greatly depends on the regularity with which all the functions of the body can be performed; and care should be taken to rest the body and mind at least for one hour after food. Smoking stramonium, the inhalation of chloroform, and the like, although they appear to soothe and mitigate the paroxysm in some cases, yet they do not appear to *shorten* the attack in any.

The fumes of *stramonium* are to be collected in an inverted glass bowl with a narrow mouth. The bowl being charged to its full, is placed under the mouth of the patient, who is directed to inhale, to the fullest extent in his power, the smoke which has been collected in the bowl. Or the stramonium may be smoked as tobacco is smoked, then puff the smoke into a tumbler, and inhale it cold into the lungs (BULLAR). Or it might be smoked as an Eastern smokes the Oriental hookah, in which the smoke is purified by being passed through water. Savory and Moore prepare cigars and cigarettes of stramonium. The fumes of brown paper saturated with a solution of nitrate of potash sometimes also relieve the spasms.

Indian hemp, in doses of from two to four grains of the extract, or thirty minims of the tincture, will often relieve the spasm for the time being, but may fail ever after (See Watson, *Lectures on the Practice of Physic*, vol. ii.)

SECTION. V.—DISEASES OF THE LUNGS.

PNEUMONIA.

LATIN EQ., *Peripneumonia;* FRENCH EQ., *Pneumonie;* GERMAN EQ., *Lungenentzündung*—Syn., *Pneumonie;* ITALIAN EQ., *Pneumonitide.*

Definition.—*Inflammation of the lungs, which, in its acute sthenic form, uncomplicated by constitutional or specific diseases, runs a definite course, expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day, in proportion to the severity of the disease and the textures implicated, and while the local productive results of inflammation in the form of the lung-lesion are yet intense, but which are eventually removed.*

The natural course of pneumonia is materially modified by constitutional or specific diseases, especially if any organ, such as the kidney, the heart, or the liver, is involved; or it may be modified by the secondary contamination of the blood by absorption of lung-exudation in the latter stages of the disease, tending to inflammation of the other lung, to pleurisy, to pericarditis, or to blood-coagula in the cavities of the heart or great vessels.

Pathology.—In the continental cities of Europe about *eight per cent.* of all the deaths are caused by pneumonia; and while about *three per cent.* of all diseases are due to the same cause, about *two per cent.* of all cases of disease in hospital are referable to pneumonia. It is a disease of very general and universal prevalence, and is subject to more or less well-defined periodic fluctuations, and sometimes appears as if it were epidemic; while its prevalence has been observed to be very much coincident with that of *typhus* fever (NIEMEYER, *New Syden. Society Year-Book*, 1862).

The only variety recognised by the College of Physicians is (*a*) lobular; and the term "*secondary*" has been applied to pneumonia when it occurs as a complication of some other disease.

On the other hand, Niemeyer regards inflammation of the lungs as of three kinds, namely :—

(1.) *Croupous pneumonia*, in which the air-cells are involved in a morbid process identical with that which attacks the mucous membrane of the larynx in tracheal and laryngeal croup (*acute pneumonia*).

(2.) *Catarrhal pneumonia*—a process intimately related to that already described as *acute catarrhal bronchitis;* and when inflammation affects the smaller tubes (*capillary bronchitis*) the disease can with difficulty be separated clinically from catarrhal pneumonia, which produces an augmented secretion and active generation of pus corpuscles, but in which no coagulable exudation is formed.

In these forms (1.) and (2.), the products of inflammation are thrown out upon the free surface of the mucous membrane of the air-cells, the tissue of the lung itself suffering no essential lesion.

(3.) *Interstitial pneumonia*, when the inflammation involves the walls of the air-vesicles and the interlobular connective tissue, which from its chronic course is sometimes termed *chronic pneumonia.*

Under these three heads it is proposed to consider pneumonia.

(1.) *Croupous or Acute Pneumonia.*

Dr. Clymer states that pneumonia was very common during the American War, in both the United States and Confederate armies, and that the mortality rate was high.

In the United States, for the two years ending June 30, 1862 and 1863, there were 31,527 cases and 7,091 deaths out of a total of 8,098 deaths from all the inflammatory diseases of the respiratory organs, giving a ratio of deaths to cases of 1 in 6·8 in the Atlantic region, and 1 in 3·8 in the central region (*Circular, No.* 6, *Surgeon General's Office, War Dep.*, 1865).

In the Confederate army, during a period of nineteen months (January, 1862, to July, 1863, inclusive), $17\frac{6}{10}$ths per cent. of the mean strength, on an average, had pneumonia; and it gave 2·7 per cent. of all cases of disease and wounds entered upon the field reports; and 3·15 per cent. of all the cases of disease and wounds entered upon the hospital reports. In a period of fifteen months (January, 1862, to March, 1863, inclusive), there were in certain general hospitals in the State of Virginia, 4,774 cases of pneumonia reported, and 1,261 deaths, a mortality of a fraction more than one-fourth the cases, or more exactly 22·86 per cent., or 1 death in 3·78 cases. During four months, September, 1862, January, February, and March, 1863, 1,527 cases of pneumonia were entered upon the registers of the general hospitals in and about Richmond, Virginia, with 405 deaths. For this period the ratio of deaths from pneumonia to the entire number of deaths from all causes was 19·22 per cent.; whilst, on the other hand, the cases of pneumonia during these four months was only 3·54 per cent. of the entire number of cases of all diseases treated. For four months, April, May, June, and July, 1863, there were 108,165 cases in all the general hospitals of Virginia, with 2,705 deaths, the ratio of deaths from all causes to the entire number of cases treated being 2·5 per cent., or 1 death in 39·98 cases. 21·29 per cent. of the deaths from all causes were from pneumonia, whilst it made only 2·16 per cent. of the entire number of cases of all diseases. 24·14 per cent. of the cases of pneumonia ended fatally, or 1 death in 4·05 cases. In the General Hospital at Charlottesville, Virginia, nearly one-third, or, more exactly, 31·9 per cent. of the cases of pneumonia were fatal, or 1 death in every 3·12 cases; the ratio of deaths from pneumonia to the entire number of deaths from all causes, 23·84 per cent., or 1 death from pneumonia in 4·17 deaths from all causes, including pneumonia. On the other hand, the cases of pneumonia were less than one-twenty third of the total number of cases (4·32 per cent.), or, more plainly, 1 case of pneumonia in 23·13 cases of all diseases, pneumonia included. In General Hospital, No. 1 (Confederate), Savannah, Georgia, during a period of twenty-five months (December, 1861, to December, 1863, inclusive), more than one-third of the entire deaths from all diseases were caused by pneumonia, whilst the cases of pneumonia to all diseases was in the ratio of 1 to 19·32; a little over one-third, or 1 in 3·18, or 31·35 per cent. of all the cases of pneumonia were fatal (J. JONES, *U. S. Sanitary Commission Med. Memoirs*, 1868).

From January 1 to May 1, 1864, 784 cases of pneumonia, happening amongst the U.S. coloured troops, were treated in the hospital attached to Benton Barracks, Mo., of which 156 died. Besides the above there were 675 cases of measles, of which 130 died, death being caused mainly by pneumonia (IRA RUSSELL, *U. S. S. Com. Med. Mem.*).

In the British army, during the Crimean War, the deaths from pneumonia to the number of cases of the disease is reported at 1 to 3·6.

Dr. Clymer concludes from the foregoing statistics that the death-rate from pneumonia is much higher in armies than in civil life. Many of the reported cases of pneumonia, in both the United States and Confederate armies, he observes, were intercurrent affections, particularly in measles, chronic camp diarrhœa, and malarial toxæmia, and this measurably explains the great mortality, without reference to the methods of treatment. Dr. Clymer is satisfied, from personal observation, that many cases of capillary bronchitis were reported as pneumonia. The prevailing type of the disease in both armies was adynamic.

The phenomena which are appreciable during the natural course of a disease which is uncomplicated and unmodified by any other disease or by treatment, afford the best means of studying its pathology. To Dr. Parkes, the Emeritus Professor of Clinical Medicine in University College, science is indebted for the records of such a case (*Med. Times and Gazette*, Feb. 25, 1860). The case was one of acute sthenic pneumonia, which occurred in a well-built temperate man of about ten stones weight, and which Dr. Parkes conscientiously believed could be left without any treatment (beyond the application of a few leeches) to the unassisted processes of nature.

TABLE OF THE TEMPERATURE, PULSE, AND RESPIRATIONS.

FROM HOURLY OBSERVATIONS DURING THE DAY. MEAN NUMBER OF DAILY OBSERVATIONS,—14.

Date.	Day of Disease. (The disease commenced at 4 p.m.)	Temperature in Axilla. Hourly Observations during the Day.					Pulse. Hourly Observations during the Day.					Respirations hourly observed. Mean No.
		Mean.	Max.	Min.	Hours of Max.	Hours of Min.	Mean.	Max.	Min.	Hours of Max.	Hours of Min.	
Jan. 7.	Part of Third and part of fourth day.	103·6° Fahr.	104·2°	102·6°	7 P.M.	9 A.M.	108	120	100	1 A.M.	2, 5, 10, and 11 P.M.	36
,, 8.	Part of fourth and fifth days.	103·9°	104·2°	103·6°	5, 7, and 8 P.M.	11 A.M.	107	116	98	7 P.M.	11 A.M.	36
,, 9.	Part of fifth and sixth days.	103·4°	104·2°	103·0°	1 P.M.	8 and 10 P.M.	106	112	100	4 and 6 P.M.	10 and 12 P.M.	43
,, 10.	Part of sixth and seventh days.	100·6°	101·4°	99·4°	9 P.M. 1 P.M.	11 P.M.	93	112	82	5 P.M.	10 P.M.	38
,, 11.	Part of seventh and eighth days,	98·6°	90°	98°	3, 5, and 6 P.M.	11 A.M.	76	84	72	2 P.M.	4 P.M.	30
,, 12.	Part of eighth and ninth days.	98·5°	98·8°	98·4°	1 P.M.	10 A.M., 2 and 4 P.M.	76	84	70	1 P.M.	9 A.M.	31

The characteristic phenomena of pneumonia in the majority of cases are evolved in a regular and consecutive manner; and they may be considered under the three following heads:—

1. *The Course of the Pyrexia as Measured especially by the Thermometer and the Pulse.*—In the case which Dr. Parkes records, the commencement of the pneumonia is dated from the *shivering;* and the patient was admitted to hospital on the *third day* of the disease, which was completed at four P.M. of Jan. 7, 1860. He was then intensely febrile, with flushed cheeks, constant cough, viscid bloody pneumonic expectoration, hurried breathing, with crepitation and bronchial respiration over the posterior base of the left lung. The conjunctivæ were a little yellow. Thermometrical observations in the axilla were taken hourly during the day—from nine in the morning till eleven and twelve at night—by Mr. Ringer and Mr. Miller; and the preceding Table gives the general results—records of the pulse and respiration being also given, for the purpose of comparison with the temperature, and with each other.

During the *third* and *fourth days* of the disease the temperature was uniformly high, the difference between the maximum and minimum being only 1·6° Fahr. on the *third*, and 0·6° Fahr. on the *fourth day.* The *fourth day* was the most febrile, both as to mean temperature and as to the constancy of its height. The *fifth day* was scarcely less febrile, the mean temperature being only 0·2° Fahr. below the *fourth day.*

Towards the end of the *fifth day*, however, and during the commencement of the *sixth day* (from four to twelve P.M., 9th January), the thermometer decidedly fell slightly; then, during the early part of the *sixth day* of the disease (night of January 9), it made a great descent from 104·2° to 101° Fahr.; and after this time it never rose above 101·4°. During the whole of the after part of the *sixth day* and the first part of the *seventh*, the temperature continued to fall, and at 11 A.M., January 11—the nineteenth hour of the *seventh day* of the disease —it reached 98° Fahr. In thirty-six hours it had fallen from 103·2° to 98°, or no less than 5°. Afterwards it oscillated for two days between 98° and 98·8°, but never rose above this latter point. The fever, in fact, had ended by crisis (a sudden and rapid defervescence), which is usually accompanied by a strong action of some eliminating organ, such as the skin, the kidneys, or the bowels; and in the case recorded by Dr. Parkes there were both profuse sweating and considerable urinary excretion; but whether more at the period of rapid fall than before is uncertain.

The pulse ranged from 120 to 100 during the first three days, and then fell to 90, 80, and 70. On comparing carefully the hourly variations of the pulse and temperature, it is quite clear that there is a connection between them, so that either simultaneously, or often a little before or after, a fall or rise in the thermometer occurred, with a fall or rise in the number of the pulse. Not unfrequently the alteration in the pulse occurred before the change in the thermometer; and sometimes the pulse rose, though never greatly, when the temperature was falling. When the thermometer oscillated and finally fell, the pulse fell at the same time, and very uniformly. The respirations averaged 38 in the minute during the first four days, and 35 afterwards. They did not fall nearly so much as the *temperature and the* *pulse;* and were not nearly so good an indication of the course of the pyrexia. As the mean of the thermometer was not above 104°, as the mean of the pulse was not 120, and as the mean of the respiration was not 40, the case must be considered a slight one, according to the rule laid down by Wunderlich—namely, that the intensity of cases of pneumonia is to be judged of by the concurrence of these phenomena, so as to call all cases slight that fall *below,* and all cases severe that are *above* those averages in which *the temperature records* 104°, *the pulse more than* 120, *and the respirations more than* 40 *in the minute during the height of the disease.* It is only by a rigorous application of some such rule to the cases of pneumonia which are grouped together to furnish statistical data, as a guide to treatment, that anything like trustworthy results can ever be obtained. Such results have not yet been so obtained; and it is scarcely to be expected that the task will be accomplished in our day.

2. *The Course of the Local Lung-Symptoms.* When the patient was admitted, there was considerable crepitation, and some bronchial respiration in the lower lobe of the left lung. The hepatization increased, and was considerable on the fifth and sixth days, and its greatest amount was either at the period of the *defervescence* or was subsequent to it—the number of respirations being even greater after the temperature and pulse had commenced to fall than before, so that they appeared rather to run *parallel with the amount of the hepatization than with the general fever.* The sputa were most bloody during the *third* and *fourth days* of the disease—were less florid and more rusty on the *fifth* and *sixth*—after which they became less viscid and free from blood or hæmatine. The pain in the side disappeared on the *fourth* or *fifth day.* After the *seventh* or *eighth day* the bronchial respiration began to lessen, and had ceased by about the *twelfth* to the *sixteenth day.* Harsh respiration, some *redux* crepitation, and a little sonorous *râle* were left for some days more.

3. *The Condition of other Organs.*—Having twice had rheumatic fever (eleven and two years before the pneumonia), the heart of the patient in one or other of the attacks had been slightly damaged, and he had slight aortic obstruction, with mitral regurgitant disease, but without any marked alteration in the size of the heart's cavities. There was supplementary breathing in the right lung, and about the angle of the right scapula was some slight suspicious bronchial respiration, as if some consolidation were there; but if so, it disappeared early.

The liver was not enlarged nor tender, but the conjunctivæ were very slightly yellow; and the yellowness disappeared at convalescence. There was thirst, loss of appetite, and dry furred tongue.

The action of the eliminating organs was as follows:—(*a.*) The skin acted profusely throughout, both before and at the period of defervescence; (*b.*) The bowels were rather confined, but the motions were said to be natural; (*c.*) The amount of excretion from the lungs was undetermined; (*d.*) The excretion from the kidneys was not determined till during the *fifth* and *sixth days* of the disease, the last day of the intense pyrexia. On this day there was a little albumen (one-sixth of the height of the urine in a test tube). There were no chlorides, and on the following day the albumen had disappeared. The other urinary ingredients were not determined; but there were copious deposits of lithates on the *fifth, sixth, seventh, eighth,* and *ninth days* of the disease, and probably afterwards, of which no note was made. On part of the *seventh* and *eighth day, eight grammes of chloride of sodium* were taken by the mouth, and on the *eighth, ninth,* and *tenth days,* as on previous days, there was no chlorine in the urine. But on the *eleventh day* of the disease the chlorine began to re-appear, was in some quantity on the *fourteenth day,* and on the *twenty-first* and *twenty-second days* of the disease it was so abundant as to yield seventeen to twenty-two grammes. The urates throughout were abundant, but their exact amount was not determined.

The records of this case of pneumonia have been thus fully given, in the hope that it may serve as a model for the investigation and recording of similar cases. Till such inquiries are fully carried out by many observers, working independently and uniformly, the statistics of pneumonia will get more confused than they now are, and will therefore become even of still less value.

The following is a tabular statement of the results:—

TABLE.

Date.	Day of Disease.	Condition of Patient.	Amount of Urine in cub. cents. in twenty-four hours. (*b.*)	Urea in grammes in twenty four hours. (*a.*)	Chloride of Sodium in twenty-four hours, in grammes. (*a.*)	One kilogramme of body-weight excreted in twenty-four hours of urea, in grammes.
Jan. 10.	Part of sixth and seventh days.	First day of defervescence, temp. 100·6° Fahr.	980	85·56 (= 1,321 grains).	—	1·345
„ 11.	Part of seventh and eighth.	Temp. 98·6° Fahr. Complete defervescence.	—	—	—	—
„ 12.	Part of eighth and ninth.	Complete defervescence. Absorption of exudation.	865	87·38 (*b.*) (= 1,349 grains).	—	1·373
„ 13.	Part of ninth and tenth.	do.	865	87·38 (= 1,349 grains).	—	1·373
„ 14.	Part of tenth and eleventh.	do.	—	—	Some amount unknown. 0·040 grms. in 10 c. c.	—
„ 17.	Part of thirteenth and fourteenth.	Complete convalescence. Exudation gone.	1300	35·1 (= 542 grains).	5·2 (= 80 grains).	0·551
„ 24.	Part of twentieth and twenty-first.	do.	1297	30·2 (= 466 grains).	17·18 (= 265 grains).	0·474
„ 25.	Part of twenty-first and twenty-second.	do.	1760	44·0 (= 679 grains).	21·95 (= 339 grains).	0·691
„ 27.	Part of twenty-third & twenty-fourth.	do.	1910	40·1 (= 619 grains).	14·32 (= 221 grains).	0·630
Mean excretion of urea by one kilogramme of body-weight on the seventh, ninth, and tenth days during resolution, ...						1·363
Ditto ditto during the fourteenth, twenty-first, twenty-second, and twenty-fourth days' complete convalescence, ...						0·586
Mean physiological excretion in men aged twenty-two, ...						0·500

(*a.*) The urea and chloride of sodium were determined according to the method of Liebig, by Dr. Parkes' excellent Clinical Clerk, Mr. Smith. The chloride of sodium was not got rid of before testing for urea, but the usual correction was made. The amounts are given both in grammes and grains.

(*b.*) The urine of two days added together, and the total halved.

It is now beginning to be appreciated by thoughtful physicians that the so-called physical signs are inferior in value to those afforded by accurate observation of the correlations of temperature, pulse, condition of the urine, and tendency to crisis; and these observations, so ably and clearly recorded by Dr. Parkes, are in unison with many similar records by Wunderlich, Metzger, Barthez, Bergeron, and others. They show that the crisis may occur on any day of the disease, but the usual dates are the *fourth,* or *sixth,* or *seventh day.*

A *practical summary* of these results may be embodied under the following heads:—

The Urine in Pneumonia.—(A.) *As to normal products:*—(1.) *The amount of water* is lessened by a third or a half, (600 to 800 c. c.) This is most marked during the early days, and is no doubt due to retention of water in the febrile body. During the stage of resolution, the amount of water increases.

(2.) *The urea* is greatly increased during the height of the disease—an increase not due to any other cause than the metamorphosis of tissue during the pyrexia; and in equal periods of time it is more abundant during the day than during the night; and it is in larger quantity *before* than during resolution. Dr. Parkes, however, has found it to be very great during resolution; and the general fact must be admitted that *the greatest amount of urea coincides with the highest temperature, and that it arises from general increased metamorphosis.*

(3.) *The specific gravity* is high (1025–1035), partly from deficiency of water, and partly from excess of urea; and as the chloride of sodium is absent, the specific gravity measures pretty accurately the amount of urea.

(4.) *The uric acid* is increased, and its free excretion is a favourable sign.

(5.) *The urine-pigment* is increased two or three fold, or altered; and it tints the urates, when they fall, brown, red, or carmine; generally the colour of the urine is of a deep yellow-red hue, or flame colour.

6. *The chloride of sodium* is diminished, or is entirely absent during the existence of the fever and the occurrence of the lung-lesion—*i. e.,* during the early period, or at the commencement of hepatization. Even when given by the mouth at this period it will not pass off as in health, but is retained, and will not appear in the urine (in Dr. Parkes' case, for more than forty-eight hours). It re-appears during, or rather after resolution, but it does not always re-appear directly resolution commences. It may still be retained for some days—as many as *eight* or *ten*—after which it is poured out in such quantities as sometimes to raise the specific gravity of the urine. This especially occurs in cases where there is little expectoration and no purging, although the water of the urine is increasing and the urea is diminishing. *Fifteen* to *twenty-five grammes* may be thus passed in the urine during twenty-four hours; and the period of its increase is some time after the excretion of urea has reached its acme and is declining. There is thus ample evidence that the chlorine has been retained, and not excreted in excess through the skin; and even when *hydrochloric acid* is given to the patient, no chlorine appears in the urine. Beale's observations also prove that the lung-exudation is very rich in chlorides, which are often largely excreted in the sputa. The retention of the chlorides thus seems to be inti-

mately connected with the development of the lung-lesion or exudation; and their excretion is increased during the absorption and disappearance of the exudation. Dr. Clymer remarks that these variations are not constant, and that in other diseases a similar variation in the amount of alkaline chlorides is noticed. He considers the presence or absence of *chloride of sodium* in the urine as probably more intimately related to the amount and kind of food taken than to the several stages of this disorder. In three cases of pneumonia in old persons, two of which ended fatally, he relates, on the authority of Bergeron, that chlorides were present in the urine. In a case of pleuro-pneumonia in an adult, which had lasted already sixteen days, and where all signs of lung-solidification had ceased, but some pleural effusion remained, Bergeron could obtain no *chloride of sodium* in the urine; on the twenty-first day there was a slight precipitate; on the twenty-third and twenty-fourth days there was no trace of it; on the twenty-fifth and following days it re-appeared and was abundant.

In two cases of intermittent fever, noted by Dr. Clymer, in which an analysis of the urine was made by J. Moss, the following were the amounts of *chloride of sodium*:—*

		First Case. Grammes.		Second Case. Grammes.
During the febrile paroxysm,	...	5·5	...	0·8
During the period of apyrexia,	...	6·4	...	7·6

In two successive paroxysms of tertian ague, Ranke found the amount of *chloride of sodium* in the urine to be—

		First Case. Grammes.		Second Case. Grammes.
During the period of Apyrexia,	...	0·44	...	1·29
During the period of Paroxysm,	...	4·18	...	3·10

On the authority of Wachsmuth, Dr. Clymer records, in a case of acute pneumonia, the following analysis, showing the daily amount of *urea* and *chloride of sodium*. Convalescence began on the twelfth day, and up to that time the diet was rigid.

Day of Disease.	Daily Amount of *Urea*.†	Daily Amount of *Chloride of Sodium.*
2	13·39	5·88
3	36·28	3·79
4	22·08	4·18
5	31·27	0·47
6	17·36	0·62
7	32·37	0·38
8	23·72	0·38
9	20·15	
10	15·67	Traces.
11	18·95	
12–14 Convalescence Food.	12·51	2·69
15–16	25·70	13·73

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* Average amount excreted in twenty-four hours in health, about 11·5 grammes = 177 grains.

† Mean daily amount excreted in health (between twenty and forty years) about 33·18 grammes = 512·4 grains.

(7.) *The sulphuric acid* is increased by about a third.

(8.) *The phosphoric acid* and the free acidity are lessened.

(B.) *As to abnormal products in the urine.*—They are chiefly as follow:—

(1.) *Bile-acids.*

(2.) Bile-pigment, with or without jaundice.

(3.) Albumen is present in a large proportion of cases. The period of its occurrence is variable; but both Weller and Parkes agree that it is most common at the commencement of and before resolution—*i. e.*, at the height of the disease. It has been supposed that some of the absorbed exudation is got rid of in this way (J. W. BEGBIE); but in the cases observed by Dr. Parkes, the albumen in the urine always appeared *before* resolution, when the lungs were yet commencing to be consolidated; and in one case there were, at intervals of from *six* to *eight* weeks, three recurrent attacks of acute pneumonic consolidation, affecting different parts of the two lungs, and at the height of each attack albuminuria occurred; while in the intervals, and subsequently, the urine was quite free. Dr. Parkes, therefore, does not ascribe the appearance of albumen in the urine in cases of pneumonia to the absorbed exudation passing out by the urine in this form, but to the implication of the kidneys in the general congestion and exudation, the most marked local seat of which was in the lungs.

Of five cases in which albuminuria occurred during the height of the disease, no fewer than three died; while among seven *non-albuminous* cases observed by Dr. Parkes, only one died. While the mortality of all the cases recorded by him was *four* in *thirteen*, or 30·7 *per cent.*, that of the cases with albuminous urine was 50 *per cent.*, and with non-albuminous urine it was 14 *per cent.; "therefore albuminuria occurring before resolution appears to be an unfavourable sign;* and the very frequent occurrence of albuminuria," continues Dr. Parkes, "in sthenic pneumonia is one of the most interesting facts in its clinical history. In no disease seen in this country, except, perhaps, in *maculated typhus*, is albuminuria so common as in *pneumonia*. *Bronchitis* of the large tubes, chronic phthisis, and emphysema present a most marked contrast to pneumonia in this respect." It is not invariably connected with *extent* of local disease; cases of enormous consolidation, and with great dyspnœa, have not necessarily albuminous urine.

In some cases, instead of being increased, the urinary solids are lessened, even in intensely febrile cases, with a heightened temperature of *five or six degrees*. Such is a result of retention, and not of diminished excretion (see vol. i., p. 56, *et seq.*); and in such cases some one or other of the following events happen:—(1.) At a later period of the disease a large amount of some ingredient may be poured out by the urine, although the febrile symptoms have almost or entirely gone—*e. g.*, uric acid—the crisis by the urine of the older physicians; (2.) Towards convalescence spontaneous diarrhœa may come on; (3.) The recoveries are not so rapid as in cases in which the urinary excretion is large. The amount of the urine, indeed, is a good guide for prognosis. As in *typhoid fever* and other acute affections, it is probable that the products of metamorphosis are retained, and poison the blood.

During convalescence the urine generally augments considerably in amount, and (cases of previous retention of excreta being excluded) the *urea*, the *uric* and *sulphuric* acids diminish for a short period below their healthy range; the *chloride of sodium* increases, and an extraordinary amount is sometimes passed; but if the quantity got rid of by *sputa* or by *diarrhœa* has been large, the amount of *chloride of sodium* excreted by the urine will be proportionally less (Parkes *On the Urine*, pp. 270–279).

(2.) **Records of Temperature in (Acute) Croupous Pneumonia.**—The commencement of primary croupous (acute) pneumonia is marked by a rigor, in almost all cases, which lasts from half an hour to several hours before giving place to a sensation of heat, but nevertheless body temperature is appreciably elevated during this algide stage; and except in *ague* and *pyæmia*, rigors of equal violence to those in pneumonia are not met with in any other disease. In ague and pyæmia, however, the rigors are repeated; in pneumonia the rigor is not repeated; and hence it marks the commencement whence the duration of the disease is counted. In children, convulsions may take the place of rigor. A knowledge of the range of body-heat in cases of pneumonia indicates (Wunderlich):—

(1.) The degree of the affection and its danger.

(2.) Improvement or relapses.

(3.) The completion of the disease-process.

(4.) The certainty of convalescence and recovery.

(5.) The continuance of unresolved or lurking lesions, and the supervention of sequelæ.

Four hours after the initial rigor the temperature has been noted as high as 102·5° Fahr. in the axilla; and, eight hours later, *i.e.* twelve hours after the commencement or invasion, it had risen to 104° Fahr. (Ziemssen). Nine hours after the onset, Thomas of Leipsic found the temperature 104·8° Fahr.; in another case, twenty-three hours after the rigor, 105° Fahr.; in a third, on the morning of the second day, 106° Fahr. In eight other cases, where the body-heat was taken on the evening of the second day, it ranged from 102·2° to 104·4° Fahr.

Several observations carefully made at Netley have shown an elevation of temperature in the axilla of the affected side greater than that of the opposite side.

The elevation of temperature rises on the first day even to 103° or 105° Fahr. (rarely higher), and is accompanied by acceleration of pulse, and by increase of thirst. The face is flushed, pain is felt in the back and loins, with soreness of limbs and joints. The fever is never a continued one, but is remittent or sub-remittent—*i.e.*, the daily fluctuations in its exacerbations and remissions may be considerable, varying from 75° Fahr. to 1·80° Fahr.; or may be so slight as not to exceed from 0·4° Fahr. to 0·5° Fahr. (Thomas, of Leipsic). The temperature is lower during the early morning hours; and the exacerbation for the day begins in the course of the forenoon, attaining its height usually in the afternoon, when in bad cases it may rise to 105·8° Fahr., or even to 107·7° Fahr. In most cases a day or two before the crisis the remission increases. The progressive increase of temperature rarely continues beyond the second day; for generally about that time, or about forty-eight hours after the initial rigor, the maximum of temperature is reached.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF *CROUPOUS* (*ACUTE*) *PNEUMONIA*, DATING FROM THE FIRST EVENING OF THE ATTACK. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) OBSERVATIONS, (Wunderlich, fig. 51, Table VI.)

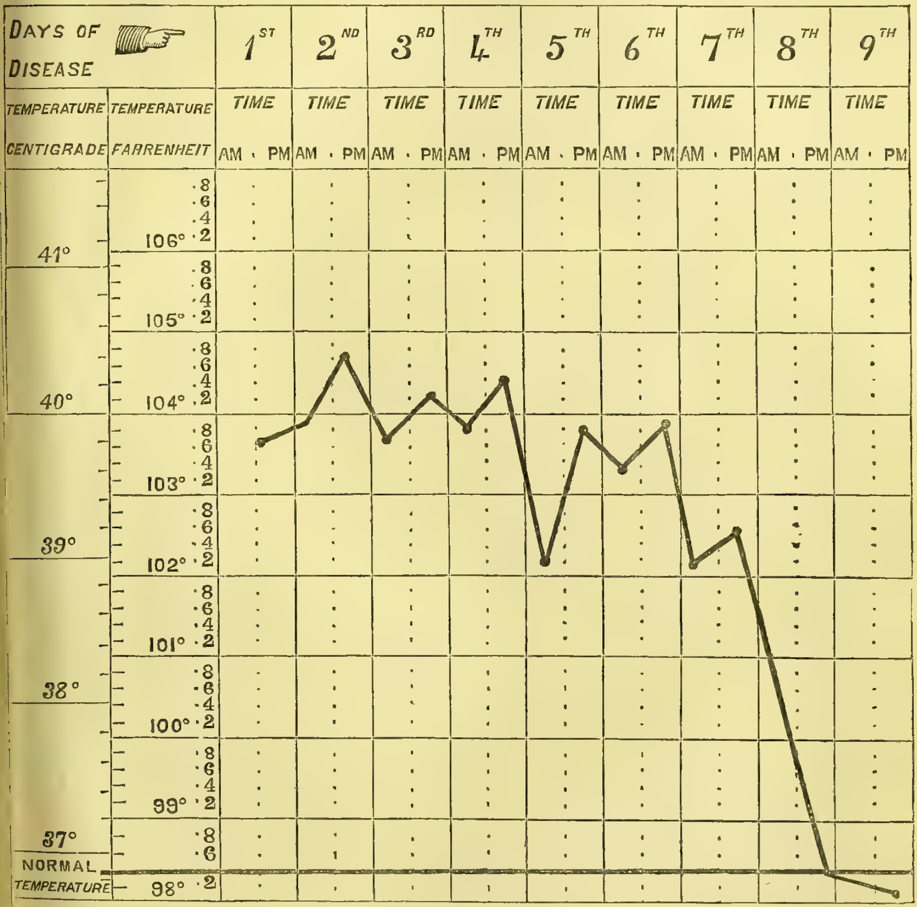

Fig. 51.

The fever of pneumonia has also many peculiarities, as pointed out by Wunderlich, namely:—

(1.) The occurrence of isolated *brusque* elevations and intercurrent falls of temperature, the latter of which are in direct contrast to the elevations.

The continuous high temperature lasts as long as the process set up in the lungs progresses; which is seldom less than three days, and seldom longer than seven.

Defervescence in the majority of cases begins late in the evening, and takes place rapidly.

(3.) **The Co-relation of the Pulse and Temperature in Pneumonia.**—In a pneumonia of average severity the pulse usually ranges between 90 and 120 beats in a minute; but in severe cases, when the temperature is very high, it may attain a frequency of 130, or even 150, or more. At the outset it is generally large and full; but as the disease progresses, it becomes small and soft, from depression of the heart's action consequent on the blood-heat (NIEMEYER).

TABLE OF CO-RELATION OF PULSE AND TEMPERATURE USUAL IN PNEUMONIA (Waters).

Day.	Temp. Fahr.	Pulse.	Day.	Temp. Fahr	Pulse.
1	102·8°	123	6	102·8°	122
2	102·3°	120	7	100°	114
3	103·6°	122	8	99°	94
4	104°	126	9	Normal	98
5	103°	122			

(4.) **The Natural History of Pneumonia** would be incomplete without some attempt to expound the causes of this tendency to a spontaneous favourable termination of uncomplicated cases, and their determinate duration. This cannot be better expressed than in the words of Dr. Parkes. First of all he considers the following question:—

"What is the exact connection between the lung symptoms and the general pyrexia? The course of the two is certainly not exactly parallel. The complete consolidation seems to be posterior, in point of time, to the height of the pyrexia. It has even been supposed by Wunderlich that the exudation into the lungs coincides with the end of the pyrexia—that is to say, that the defervescence commences when the lungs become completely hepatized. I have not been able to satisfy myself rigorously on this point. If it could be satisfactorily made out, it would certainly imply that the exudation into the air-cells relieved or cured the fever—in other words, that the lung-disease is not a primary, but a secondary condition, and that it succeeds to and brings to an end, by purifying the blood, a condition of general pyrexia, arising from blood-disease. Without believing that this relation is quite determined (if it were determined, the case would be settled), there is no doubt that the fever ends spontaneously, or very greatly lessens at the time when the inflammation of the lung is very great.

"There are at present two views, more or less clearly defined, which aim at explaining these phenomena, and which may be thus expressed—

"1. According to the first of these, there is a blood disease of some sort, of a nature not thoroughly known, but which consists, in part, in an augmentation of the fibrin, as in acute rheumatism. Increasing up to a certain point, and giving rise to the slight malaise which precedes all cases of pneumonia, it is at last brought to a head by some exposure, by a dietetic error, or by reaching a point at which the functions of the blood are seriously interfered with. Then ensues high general fever, from implication of the nervous system, and at the same time some organ or other is, on account of special affinity for the morbid blood, or from previous damage to its structure, specially irritated. In pneumonia the lung is the seat of election, and there is rapid hyperæmia and transudation of fluid into the air-cells. By this transudation the morbid blood is purified. The process is analogous to that of gout, in which a diseased blood gives rise to a local disease by the deposition of urate of soda in and about joints. When the localization and consequent purification is finished, then the fever ends. There remains the lung-exudation, which gradually softens down, is partly expectorated, partly absorbed; and, in the process of absorption, it may produce again secondary contamination of the blood, and certain affections of other organs, which constitute those secondary affections which sometimes complicate the after-course of pneumonia.

"The weak points in this hypothesis are the want of definite indications of the blood-disease, and of its mode of production. There is some evidence on these points, but it is certainly not very great.

"Its strong points are the explanation it gives of the previous malaise; of the sudden outburst of fever, when the diseased blood implicates at last the nervous system; of the singular and rapid termination of the pyrexia at a time when the lung-lesion is yet intense; and of the enormous elimination of urea during the very first days before the lung-exudation has softened down.

"2. The second hypothesis is the exact contrary of the former. The lung affection is supposed to be the primary lesion: it is a local inflammation produced by the (still obscure) causes of local inflammation, running the ordinary course of such inflammations, and giving rise to violent symptomatic pyrexia. The undoubted increase of fibrin in the blood is supposed to be not primary but secondary, to be caused by and to augment with the inflammation, and to be at its height with it. Virchow has stated that this fibrin is nothing more than the albuminous substance absorbed by the lymphatics of the inflamed and hepatized lung, and poured into the blood. The fever is believed to be entirely symptomatic of the local disease, and to be commensurate with its intensity and extent. The arguments for this view seem to be, that the lung-symptoms are remarkably early in manifestation, though they may not be very intense. Pain in the side and cough are very soon present, and sometimes occur even before the shivering and headache. The pyrexia, although great in the early days, is perhaps not greater than might have been produced by the condition of the lung; and as to the termination of the fever, this may be supposed to occur because the really true febrile stage of pneumonia is not the period of complete exudation, but the preceding period of intense hyperæmia. To say that the pyrexia is gone when the lung-lesion is yet most intense, may be an incorrect expression of the fact: the consolidation may possibly, indeed, be most intense, but this may be merely the natural termination of that enormous hyperæmia and blockage of vessels from local changes of nutrition, which is in reality the essential disease.

"The difference between these two hypotheses would be this: the fever ends spontaneously, first, because the blood is purified, or, secondly, because the local disease ends spontaneously—*i. e.*, the active febrile-making local disease. This last assumption, however, is decidedly a very bold and hazardous one.

"Between these two views it is not very easy, nor perhaps is it desirable, yet to choose, for the blood has not yet been sufficiently examined. The only blood-disease which has yet been indicated by the supporters of the first view, as anterior to pneumonia, is hyperinosis (excess of fibrin); and as hyperinosis occurs in acute rheumatism without pneumonia, it is evident that there must be some other cause, either in the blood or in the local structure of the lung, which locates the disease in that part.

"That hyperinosis is really anterior in pneumonia as in rheumatism, must, in spite of the opinion of Virchow, be considered likely, from the experiments, among others, of Professor Naumann, of Bonn: that it is not the only condition in either of these cases will be generally admitted. But what other blood affection is there? None has yet been indicated, to my knowledge, in the acute sthenic pneumonia of young persons without gouty or renal disease. But there is one point on which I have been trying to collect evidence for some years, but at present without sufficient success. It is well known how frequently the liver is affected in pneumonia, so that some amount of jaundice is not at all uncommon, and sometimes bile-pigment appears in the pneumonic sputa. I have also found in some cases evidence of liver affection for some time before the lung disease, especially the so-called torpor with deficient biliary flow.

"Is there, then, any condition of the liver which adds something to the blood which ought not to be there? Taurin has been found in the healthy tissue of the lung; but in the hepatized lung it seems, from Verdeil's observations on his pneumic acid (taurin), to be in excess. Is it some compound of this sort which, in combination with the hyperinosis, determines the localization of the blood-disease, or produces by its irritation the inflammation of the lung? I know no facts whatever which can lead to a decision; but it is to be hoped that some competent person will soon undertake a more complete analysis of blood in the very first days of pneumonia than has yet been made.

"But, whatever be the facts as to the order of things in pneumonia, whether the lung affection is a mere localization of the blood-disease, or whether the undoubted blood-disease of the developed stage is merely produced by absorption from the inflamed lung, it is certain that the usual course of pneumonia is such as we witnessed in this untreated case, viz.,—(1.) There is an early period of intense fever, ceasing, if no complication be present, of itself at a tolerably determinate time; (2.) There is a later period of lung hepatization, which softens down during a period of moderate fever, and is expectorated or absorbed. There are therefore two periods in pneumonia, and both have their dangers. The intense early fever may kill by its intensity; the exudation may kill, subsequently, by apnœa, or may contaminate the blood, during softening, to such an extent as to lead to renewal or increase of the fever and inflammation of other parts, or to coagulation of the blood in the heart or great vessels. Each period has its own dangers, and must have its own treatment."

Morbid Anatomy.—Following Andral and Laennec, all subsequent writers on pneumonia are persuaded that a knowledge of the anatomical characters of the lung in this disease is essential to a perfect appreciation of the symptoms, physical signs, prognosis, and treatment of the disease.

In all forms of diffuse inflammation of the substance of the lungs the vesicular pulmonary tissue is more loaded with dark venous blood than usual, and its texture is more easily broken down than in health; air, however, still penetrates the air-cells, and consequently the lung still crepitates, swims in water, and, if washed, the colour is nearly restored; but it is doughy, pits on pressure, and is red or livid, and heavier than the normal lung.

This is the *first stage of pneumonia—namely, that of simple engorgement.* This state of inflammation may terminate by resolution, or it may pass to more complicated and dangerous lesions. Dr. Stokes is of opinion, however, that a *stage of irritation* has previously existed, so that the *first* stage described by Laennec would be the *second* described by Dr. Stokes, who has repeatedly seen a condition of the lung, regarded by him as the first or earliest stage of pneumonia, in which the lung-tissue is drier than usual, and of a bright vermilion colour from intense arterial injection; but there is no engorgement (*On Diseases of the Chest,* p. 310). Skoda and Fuller are opposed to this conjectural view.

When effusion of serum succeeds, the lung is in the same gorged state, but, in addition, it is loaded with watery fluid, so that on cutting into it the serous fluid mixed with blood streams from it as from a sponge. When its action is listened to during life, a lung in this condition no longer crepitates, and its bulk is enlarged; for it may be seen after death to have taken the impression of the ribs, and it does not collapse when the chest is opened. The lung is now technically said to be in a state of *red hepatization,* or, as Andral has termed it, *red softening;* for, although firm, its texture has lost its natural toughness, cohesion, and resistant power.

This is the so-called *second stage of pneumonia.* The diseased part readily breaks up by a thrust of the finger into its substance. This state has many degrees, the air-cells being either wholly or partially impermeable.

Up to this point the description of the morbid anatomy is common to the extreme stage of bronchial catarrh, capillary bronchitis, and the commencement of acute or croupous pneumonia.

In some instances the productive effects of inflammation (commonly called exudation) are very large in quantity, mixed with blood, and the more fluid portion can be readily separated, or, as it were, pressed out of the lung. In the other extreme of this form of inflammation the exudation forms an integral part of the lung, which then becomes so solid that, if cut, it represents with much accuracy a portion of the liver or spleen. In this state it contains at the diseased part little or no air, does not crepitate nor float in water; it cannot be injected, is of a deep venous colour, while its texture is easily broken down and penetrated by the finger. The lung is still enlarged or swollen, and does not collapse when the chest is opened. It varies in colour from a reddish brown or deep dull red to a violet hue, and is generally darker in the aged than in the adult.

Much speculation has been entertained with respect to the more particular seat of pneumonia; some contending that the inflammation affects the connective-tissue of the lung, others the air-cells, and others both. It is quite certain, however, that the minute bronchial tubes are not affected in slight pneumonia; for in such cases their divided extremities stand out in the midst of the inflamed part like so many white points. When the lung is more acutely inflamed, the bronchial tubes are red, and evidently greatly inflamed (*Broncho-pneumonia*). There can be no doubt, however, from what we now know of the nature of the inflammatory process, and also from direct observation upon the tissue of inflamed lungs, that the minute elements of the vesicular and connecting tissue composing the parenchyma of the lung are from the first directly altered in their vital properties, and in the form of interstitial or chronic pneumonia the whole parenchyma is extensively implicated, (vol. i., p. 24, *et seq.*)

But the most commonly acute form of pneumonia in adults is that which suddenly attacks an extensive portion of the lung, usually commencing at the root of the lung, from which it spreads first to the lower lobe, and passes thence upwards—so that the entire lung participates in the process—extending sometimes into the other lung, and so constitutes a double pneumonia.

When it reaches the second stage, which has been described, the air has disappeared from the air-vesicles, which are filled by small firm plugs of coagulable fibrine (hence *croupous pneumonia*), coloured red by blood, and which extend into the extremities of the bronchi. This new material renders the lung heavy, so that it sinks in water, does not crepitate, is dense to the touch, but is at the same time tender and friable in texture. When cut in sections, the cut surfaces appear granular, in consequence of the fibrinous plugs swelling out the air-vesicles, to the walls of which these plugs firmly adhere. This firmness of section, rigidity, friability, and granular aspect, with redness and condensation, has obtained the name of *hepatization of lung* for this morbid state.

The microscope shows that a very active formation of new cells takes place in the air-cavities, which, no doubt, spring from the epithelium of these air-cavities; and when resolution sets in, fatty disintegration precedes that sanitary process, as already described at p. 46, vol. i., under "Compound granular corpuscle."

When the pneumonia passes into the third stage—namely, that of purulent infiltration, cell-formation especially—pus becomes prominent, so that suppuration may follow this form of pneumonia, and the pus effused may be either infiltrated or limited, as if contained in an abscess. Infiltration is by far the most common; and although this form of lesion may occur suddenly, as a result of serous infiltration in unhealthy persons, or during the progress of general diseases, yet in the belief of most pathologists it more generally follows the *red hepatization.* In this latter case the pulmonary tissue, red, dense, compact, and impermeable to air, passes to a grey colour, and hence it is termed *grey hepatization* or *grey softening*, and is regarded as the *third stage of the disease,* following Andral and Laennec. The structure in other respects of either form of hepatization appears to be the same; for if the lung be examined with a microscope, the same granulations (only they are white or grey, instead of red) are found. There are instances, however, in which these granulations are wanting, and then a grey smooth surface only is observed. Hence the *granular* and *non-granular* forms of hepatization described by some authors. In aged persons the granulations of a pneumonic lung are much larger than in the adult, depending, no doubt, on the increased size of the air-cells in persons of advanced life (MM. Hourmann, Dechambre, and Dr. Maclachlan). Hepatization in old people, with granulation, is nearly four times more common than non-granular or planiform hepatization (Maclachlan, l. c., p. 279).

In the *grey*, as in the *red hepatization*, the pulmonary tissue is easily torn, and the quantity of pus infiltrated is sometimes so great that, on cutting into the lung, that fluid readily flows from it: at other times the pus will not flow on a simple incision, but exudes by compression. The lung is solid and impermeable to air. It sinks in water, and has ceased to crepitate. The interspersion of red and dark points gives a granite-like appearance to the section of a lung which is in a state of grey hepatization. The lung is then more friable than in red hepatization. The finger sinks into the grey portions of the lung upon the least pressure, and when the grey texture is squeezed it breaks down into a pulp.

(b.) *Catarrhal Pneumonia.*

It is always preceded by catarrhal bronchitis, and may be in some cases a form of capillary bronchitis, extending to the air-cells of the lungs; hence, many of its features are similar and undistinguishable from capillary bronchitis (q. v., p. 732). It is often a complication of measles and of hooping-cough.

It is the form to which the name or variety of lobular pneumonia has been applied, because it is limited to lobules, and as such it is disseminated and insulated in contra-distinction to the acute or croupous form which affects whole lobules.

The lesion is recognized by the scattered condensed foci of inflamed tissue which pervade the lungs; chiefly abounding towards the periphery, where they are more or less wedge-shaped. On section, these condensed portions have a smooth homogeneous appearance, without the granular characteristics of acute or *croupous* pneumonia. These condensed parts are impervious to air; and a dilated bronchole filled with tenacious secretion may be seen in the centre of these spots. These condensed foci go through the same stages of pneumonia as have been already described.

The kind and manner of the cough, and the character of the fever, are the phenomena which indicate catarrhal pneumonia. The cough is painful; and the temperature of the body always becomes elevated, as when catarrhal pneumonia supervenes upon catarrhal bronchitis.

In simple capillary bronchitis the temperature rarely exceeds 102° Fahr. (Ziemssen); but when catarrhal pneumonia supervenes, it may soon reach 104° or 105° Fahr., with frequent pulse and flushed face. Collapse of the lung extends in proportion to the foci of tissue involved, and progress to a fatal end may thus be more or less rapid. When resolution sets in, it commences gradually and progresses slowly but steadily, with a range of 2° to 3° Fahr., after about the 6th or 7th day, as in the accompanying diagram.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF *CATARRHAL PNEUMONIA*. THE RECORDS INDICATE MORNING (A.M.) AND EVENING (P.M.) TEMPERATURE, (Wunderlich, fig. 59, Table VI.)

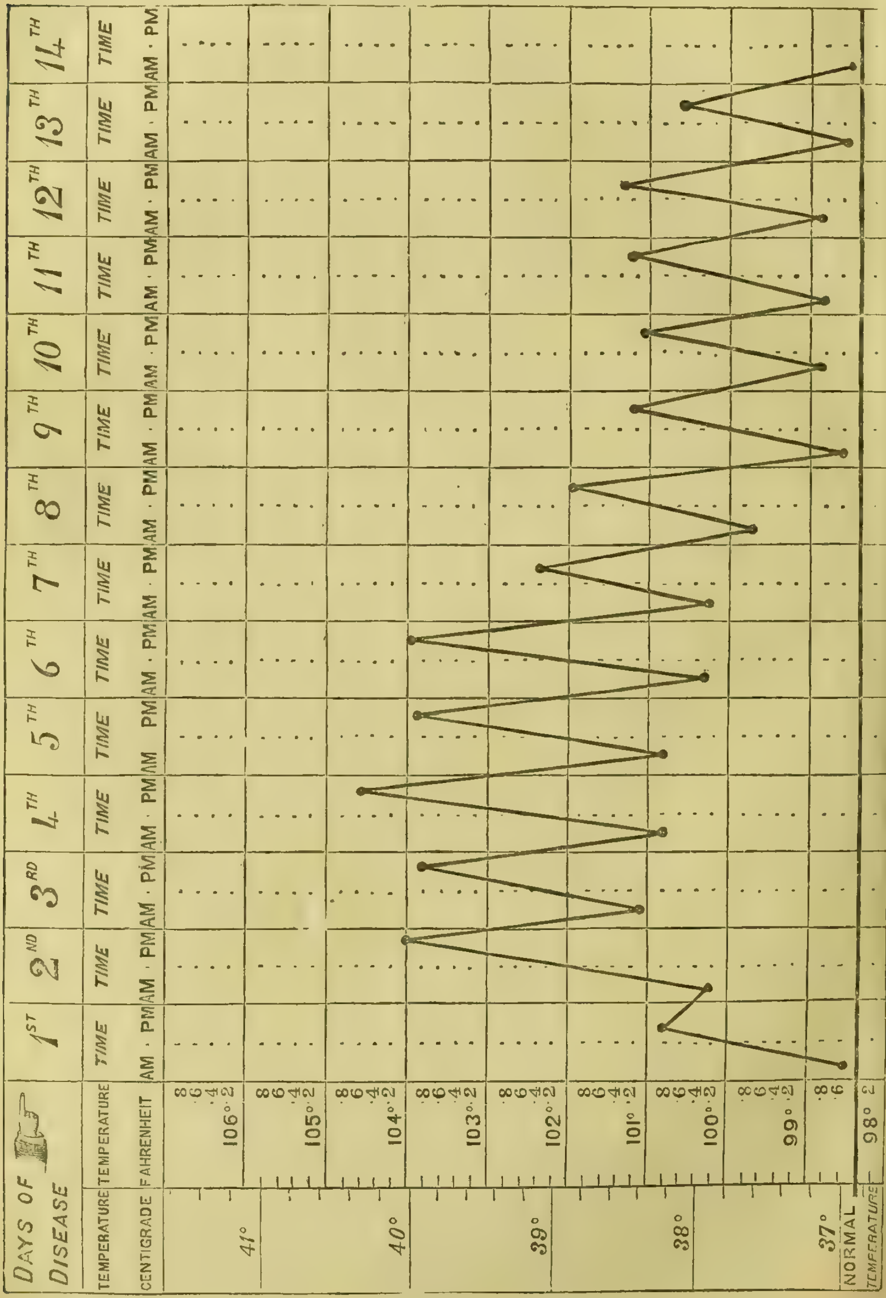

Fig. 52.

(c.) *Chronic or Interstitial Pneumonia.*

The little connective tissue which prevades the lung is here the principal texture involved, so that a hyperplasia or growth of its substance takes place, resulting (1.) in considerable augmentation of the substance of the lung, with a diminution of the air spaces, and (2.) subsequent contraction of the new material. Interstitial pneumonia scarcely ever occurs as an independent lesion, but is often the result of prolonged irritation, as from the inhalation of iron or steel filings, fine dust of coal or other substances, which establish first a bronchitis, followed by the induration peculiar to this chronic form of pneumonia. Before contraction of the new material commences, the lung substance is solidified and void of air, hyperæmic and red, or of a pale bluish-grey colour. After the new tissue of chronic pneumonia has contracted, various forms may be assumed by the lesion, such as bands or condensed masses of tissue running through the pulmonary substance, sometimes containing much pigment and grating-like cartilage under the knife. It is the material which forms the walls of old cavities, and of abscesses of the lung. It is usually an affection of middle and advanced life, and is most frequently met with among spirit-drinkers. It has sometimes been described in England and America under the name of "*fibroid degeneration of the lung.*" It has been regarded—(1.) As a variety of phthisis (BAYLEY); (2.) As a result of unresolved pneumonia (ADDISON); (3.) As a special form (interstitial) of pneumonia (ROKITANSKY, NIEMEYER); (4.) As a peculiar growth or hypertrophy (WILKS); (5.) As a pulmonary cirrhosis (FELTZ); (6.) As fibroid degeneration of the lung (CLARK, CLYMER, SULTAN). In general both lungs are affected; and if only one, generally it is the left that suffers, commencing in the upper lobes. It occurs as a nodular growth, or as a diffused fibrous-tissue development. Cavities are apt to form, while constant cough, with expectoration and gradual waste, brings the disease under *one form* of pulmonary phthisis to be afterwards considered; the progress of which is very slow, extending over several months, and even years.

The weight of pneumonic lungs is generally in proportion to the extent of the pulmonary tissue involved. Grisolle has recorded the weight of a hepatized lung as high as 5½ lbs. In eighteen pneumonic lungs examined by Dr. R. Cressen (quoted by Dr. Clymer), there was an average excess of 2 lbs. over the weight of the opposite sound lung—one weighed 5 lbs. 11 oz., the weight of the opposite sound lung being 1 lb. 8 oz.—the weight of the new material being about 4 lb. 3 oz.

The right lung is more frequently attacked first than the left, in the proportion of thirty-two to eighteen out of fifty cases noted by Dr. Russell; and, according to Dr. Clymer, pneumonia of the right lung is about twice as frequent as on that of the left, and thus holds good at all ages. The inferior lobe is oftener affected than the superior, in the proportion of four to three. Pneumonia of the apex is two and a half times more common in the right than in the left (GRISOLLE). Double primary pneumonia is very rare.

(d.) *General Symptoms of Pneumonia.*

Pneumonia is generally preceded by some antecedent fever, by shivering, more or less violent, and often by bronchitis. In a few cases, however, consolidation may be complete ere any primary affection is observed. In others, failure of the appetite, general weakness, and wandering pains in the limbs and chest, precede any definite attack by several days.

The disease being set up, the patient is restless and uneasy; his respiration difficult and hurried—from 30 to 50 in a minute—according to the amount of lung whose function is suspended; his cough frequent, and his expectoration streaked with blood; but notwithstanding this symptom he seldom, unless the pleura is affected, suffers pain, which consequently increases the danger. The aged, however, seldom complain of difficulty of breathing during the progress of the disease, whatever may be the frequency of the respiration, so that it is incumbent to count the movements of the chest in old people, to avoid all sources of error (HOURMANN, DECHAMBRE, and MACLACHLAN). Obscure and latent forms of attack, in the experience of these eminent physicians, are frequently met with in aged subjects labouring under chronic disease of the brain, heart, or some other internal organ. The pulse is full and frequent—from 100 to 120; the countenance livid; the nostrils dilated; the tongue and lips more or less livid, the former of which is coated with a white or yellow mucus. The patient inclines to lie on his back, supported by pillows. If he recovers, these symptoms are gradually mitigated; but should his case tend to a fatal end, the tongue becomes brown and typhoid, his pulse more rapid, profuse sweats break out all over his body, and at length his mind wanders, and he dies by *coma* or *apnœa*. There are many instances, however, where the course is widely different, and in which the patient, though evidently distressed by impeded respiration, has yet moments of cheerfulness, gets up, and may even walk about. But he suddenly dies, seized with a severe paroxysm of dyspnœa or of coughing, followed by collapse of the remaining healthy part of the lung.

In typical cases of acute or croupous pneumonia certain groups of symptoms are more or less constantly combined,—namely, accelerated respiration, with more or less difficulty of breathing, and rapidity of pulse. These are so correlated that the severity of the case is best measured by observing the correlation of these symptoms. The cough is at first dry, but soon there appears a scanty, semi-fluid, grey, frothy, mucous expectoration, which becomes more and more viscid, and which, in the first stage of the disease, remains of a catarrhal yellow or white appearance. Afterwards, during the second stage, it becomes still more tenacious, reddish, or rust-coloured; and the heat of skin increases, with thirst and marked prostration, and a pulse increasing in rapidity.

Such are the general symptoms of pneumonia; and, except by their different degrees of intensity, it is difficult, if not impossible, to distinguish the different stages of inflammation from each other, without the aids of percussion and auscultation. The general symptoms of serous pneumonia, however, are the most marked; the uneasiness being greater, the respiration louder and more difficult, the countenance more livid and swollen, the cough more harassing, the expectoration more abundant, and the attempt to lie down impossible. A gangrenous state of the lung is determined chiefly by the intolerable fetor of the breath.

The leading symptoms of the several forms of pneumonia which have been recognized may be stated as follows:—

(1.) *Symptoms of Acute or Croupous Pneumonia.*—In about a fourth of all the cases, the usual initial symptoms of a threatening illness prevail, and the actual invasion is generally marked by a rigor, the nature of which has been already described; and in old people it is invariable. Sharp lancinating pain at or near the nipple of the affected side is an early and most common symptom (DURAND-FARDEL, BERGERON, CLYMER). Some describe the seat of pain as at the point where the inflamed lung comes in contact with the thorax; but opinions are expressed of the most opposite kind with regard to pain in pneumonia. Simultaneously with the rigors, shortness of breath sets in, and hurried respiration is an early and constant symptom—varying from 30 to 60 in a minute (which latter number is rarely exceeded), in proportion to the amount of lung disabled. In children the breathing attains the greatest frequency.

Cough, hardly ever absent, and often present from the very beginning —is short, dry, ringing, and harsh; and at an early period the peculiar, viscid adhesive sputum begins to be ejected. It almost always contains blood, from the rupture of capillaries and extravasation of their contents. So viscid are the sputa, that the containing vessel may be everted without the sputa falling out; being highly charged with albumen and mucin, and fibrinous casts of the minuter bronchi may be found in the expectoration (NIEMEYER).

Headache usually continues throughout the attack, frontal, acute, lancinating, or constrictive. It is generally combined with sleeplessness, and sometimes slight delirium. It is at its worst about the fourth day, and generally begins to subside as the fever subsides, about the seventh day.

The duration of a case of pneumonia varies greatly. Laennec conceived that the diffuse inflammation lasted seven or eight days. We have seen that in simple uncomplicated cases the crisis is reached about the fourth or sixth days (WUNDERLICH, PARKES, METZGER). Andral records the duration of the disease in the adult at eleven days; Chomel and Laennec at from seven to twenty days; Bouillaud from eight to twelve days; Hourmann and Dechambre fourteen days. The mean duration in the aged, of 109 carefully recorded cases by Hourmann and Dechambre, was found to be nine days and seven-tenths; and Dr. Maclachlan agrees with them in considering the progress of pneumonia to be more rapid in the aged than in the adult. More generally, however, taking all its forms, cases of pneumonia terminate between the seventh and the twentieth day. Defervescence may begin between the third and fifth day, and from that to the seventh or ninth; but the belief in certain critical days is getting less and less.

Physical signs are to be appreciated by *inspection*, *palpation*, *mensura-*

tion, percussion, and *auscultation. The physical signs of pneumonia,* in the adult and in the aged, are so dependent on the morbid changes which have been described in the lung in the various stages of the disease, that a description of them is best given in correlation with the morbid anatomy of the disease. In doing so, the descriptions given by Dr. Fuller in relation to adults, and by Dr. Maclachlan in relation to the aged, will be mainly adhered to.

On striking the chest of a person in health it returns a certain hollow resonant sound, demonstrating it to be partly filled with air. Also, if we place the ear to the chest we hear certain sounds, on inspiration and on expiration, which are termed the respiratory or vesicular murmur (as described at p. 507, *ante*). In pneumonia these natural sounds are altered, the sound on percussion being rendered duller than natural, while the bronchial respiration undergoes still more remarkable alterations; and these modifications enable us, by percussion and auscultation, to determine the nature and seat of the disease. During the *first stage*, inspection of the chest shows that the costal movements are not materially diminished, unless the motions of respiration are restrained by pain; and it is only by percussion and auscultation combined that the existence of pneumonia can be determined.

By *percussion* a peculiar euphonic resonance may be emitted by that portion of the chest at which pneumonia is beginning, which ceases after the commencement of exudation, to be replaced by dulness more or less marked according to the extent of the lung involved (FULLER). During the period of invasion in the aged, the chest continues to sound clear over the seat of the impending inflammation; and as congestion increases, the lung becoming more dense and less permeable, the sound emitted on percussion may be somewhat duller; but in general there is no appreciable alteration till the *second stage* has set in (MACLACHLAN).

The signs elicited by *percussion* depend on the consolidation of the tissues, the amount and position of the consolidated part in relation to the surface of the chest, and the amount of intervening healthy lung. If the consolidation is near the surface the sound is decidedly dull, and the sense of resistance to the extremities of the percussing fingers is considerable in proportion to the amount of the consolidation. For the chest to emit this unequivocal dull sound of hepatization in middle age the disease must be extensive, and without intervening healthy lung; but in some cases, if the hepatization be central, the air in the more superficial portions of healthy lung often prevents a dull sound from being returned on percussion. In such cases percussion elicits a clear but shallow resonance. A slight diminution in the resonance of the chest, immediately over the affected portion of the lung, is usually all that percussion elicits in old age, in the second stage of pneumonia. By auscultation during the second stage, tubular breathing is found to be a most constant accompaniment alike in the adult and in old age. The respiration is of a hollow character, diffused throughout the hepatized portion—tubular towards the centre, but harsh and blowing towards the periphery of the affected part. Where the tubular breathing prevails, râles and rhonchi are absent; while vocal resonance is intense, usually of a metallic ringing character. This is most marked in aged persons. The *ægophony* in them is more decided, the shrill, acute, tremulous voice of old age being more favourable to *ægophony* than to *bronchophony.* When fibrinous exudation is great and complete, blocking up vesicles and tubes completely, absolute dullness on percussion, with little or no vocal resonance, can alone be heard; and the heart's sounds, greatly intensified, are then frequently transmitted through the consolidated lung.

Auscultation during the early period of engorgement, before exudation has taken place, finds the breathing weak in the affected parts, and exaggerated in their immediate vicinity. Dr. Clymer's observations, carefully made through a series of years, incline him to believe that both weak and harsh respiration may be heard at the very beginning of pneumonic engorgement, according as the bronchial or vesicular element of the breath-sound predominates. If the morbid process is limited to the air sacs, the increased vascularity of the pulmonary plexus, and probable dryness with congestion of the membrane, will impair their function, and the soft, breezy, vesicular sound will be feebler, while the natural sound in the minute bronchial tubes, more or less marked in healthy respiration, will become more audible, without absolute increased intensity; and those may alone reach the ear. Substantial testimony in favour of this view is given by Drs. A. Flint and J. R. Leaming, of New York. In the aged, tubular breathing is sometimes audible at the root of the lung at a very early period, if the respiration is weak. But as soon as fluid exudes into the affected parts, the respiratory sounds are obscured or replaced by the small crepitation characteristic of pneumonia. This sound, called crepitation, is made up "of a multitude of minute crackles, which occur in a volley towards the end of inspiration, and are unaffected by coughing or expectoration" (FULLER). It is the true crepitating râle of Laennec, and has been variously compared "to the crackling of salt thrown upon the fire," or to "the sound produced by rubbing a lock of hair between the finger and thumb close to one's own ear" (WILLIAMS). It usually, but not invariably, accompanies the accession of pneumonia; and it is very seldom present in advanced life. In subjects beyond the age of fifty, and particularly in still more advanced age—as between sixty and eighty—the bubbles composing the pneumonic crepitation are very generally larger, more hurried, and less numerous. The râle essentially resembles the subcrepitation of capillary bronchitis, and is very often speedily marked by copious accumulation in the larger bronchi (MACLACHLAN, GRISOLLE, CAZNEUVE, HOURMANN, DECHAMBRE). When the air-cells are completely filled by the exudation, and the minute bronchial ramifications obstructed, crepitation ceases; and when capillary bronchitis commences, small bubbling râles are heard accompanying both expiration and inspiration. When acute pneumonia arises in connection with *acute rheumatism,* Dr. Fuller has often observed that crepitation never occurs. This, he believes, is attributable to the occurrence of the exudation into the interlobular connective tissue, and consequent exclusion of the air-cells (l. c., p. 221). Vocal resonance is generally intensified. The return of the respiratory murmur, and the gradual disappearance of crepitation, indicate the resolution of the disease—a process which is generally more tedious and less perfect in the aged than in the adult—often followed in elderly persons by symptoms of continued irritation of the lung or bronchi. On the other hand, in proportion as crepitation masks the respiratory murmur, and replaces the sound of pulmonary expansion, the supervention of consolidation may be anticipated.

Beyond the limits of dulness and consolidation, the extension of fine crepitation denotes the extension of the process of *hepatization;* but when resolution has commenced, crepitation again returns to the part which was dull and consolidated before; or moderate sized râles are heard, denoting the passage of air to and fro amongst the fluid which occupies the air-cells and smaller bronchial ramifications; and very soon the percussion note assumes its normal character.

During the *second stage,* inspection of the thorax shows the costal movements diminished in the affected side, and they may be somewhat increased on the unaffected side; but the movement of elevation is less affected than that of expansion.

By *palpation* we learn that *vocal fremitus* is usually above the average: and Dr. Fuller has verified the observation by *post-mortem* results, which showed that when the bronchi were plugged with exudation vocal fremitus was entirely abrogated. The *fremitus* produced by a deep-toned powerful voice is seldom observed in the aged, and even *bronchophony* is occasionally absent (Maclachlan).

The passage from the first to the second stage of pneumonia is generally extremely rapid in old age, sometimes within a few hours (Maclachlan). In the *third stage* of the disease the physical signs do not differ from those already described; and neither in the adult nor in the aged are any certain phenomena appreciable by which the passage from the second to the third stage can be determined. A persistence of the sounds already described in place of those denoting resolution, and especially when the expression of the phenomena diminishes with increasing prostration of the patient and diminished energy of voice and respiration, betokens suppuration of the lung. It is by the succession of the physical signs in the adult and in the aged; by the occurrence of a rhonchus composed of large humid bubbles, without crepitation, in a portion or portions of lung previously consolidated; by the coincidence of this state with increased exhaustion; by a peculiar dusky cachectic expression of the patient; by feebleness of the pulse; and by typhoid symptoms and bloody sero-purulent sputa—that no room is left for doubting the accession of suppuration of the affected part (Maclachlan).

Fine crepitation of the lung cannot always be regarded as pathognomonic of acute pneumonia. In some instances it is absent throughout the attack, alike in the adult and in the aged. "It is only," as Dr. Fuller justly observes, "by the concurrence of different signs, that it is possible to arrive at a trustworthy conclusion. Fine crepitation occurring coincidently with intense heat of skin and rusty coloured expectoration, warrants the strongest suspicion of pneumonia, and justifies the adoption of active treatment; and if it be accompanied by marked alteration in the ratio of the pulse and respiration, speedily followed by dullness on percussion and tubular breathing, the existence of acute pneumonia cannot be doubted. But crepitation, however fine, if not attended by an alteration in the ratio naturally subsisting between the pulse and the respiration [and I would add, the temperature], and not speedily followed by tubular breathing, cannot be relied upon as indicative of pneumonia. If it occurs without these symptoms, it is commonly indicative of capillary bronchitis with scanty secretion; whereas, if under the same conditions, it is accompanied by dullness on percussion, it is probably due to rapid œdema, or else to congestion of the lungs, connected with some febrile hæmic disorder, cardiac disease, or the deposit of tubercle" (l. c., p. 229).

(2.) *Symptoms of Catarrhal Pneumonia.*—The disease generally supervenes on bronchitis, and is marked by a sudden and great rise of body-heat; cough is painful; and the disease may run an acute course or pass on to excessive emaciation and wasting.

(3.) *The Symptoms of Interstitial Pneumonia* are so obscure that the special lesion cannot be recognized with certainty. It is apt to follow an acute bronchitis which has become chronic, a pleurisy, or the tardy resolution of acute or croupous pneumonia, as induration to some portion of affected part. The contraction of the indurated part is then generally rendered conspicuous by the sinking in of the thoracic wall, or flattening of the chest over the site of lesion, and different degrees of pulmonary condensation complete the physical signs. Constant cough, expectoration, and breathlessness are complained of, and there is great loss of flesh at first, which continues to progress, although more slowly than at first. Profuse hæmoptysis is sometimes an early symptom. The expectoration is that of chronic bronchitis, sometimes streaked with blood. There may be concomitant symptoms of hyperplasia of the connective tissue in other organs, such as *granular kidney, fibroid degeneration of the heart,* or *pylorus* or *cirrhotic liver.*

Causes.—Sudden chilling, when the body is overheated, and the influence of season of the year in causing, are not to be set aside. The largest number of cases of pneumonia happen in February, March, April, and May, during protracted winters and prevalence of north-east winds; but opinions are divided as to the influence of absolute cold in producing pneumonia. It is often associated with an acute dyscrasia, such as *measles* and *typhus fever;* and as such the lesion has obtained the name of "*secondary pneumonia.*" Pneumonia is rare before five years of age.

Diagnosis.—Pneumonia is distinguished from phthisis by the previous good health of the patient, and by the more acute nature of the disease; and, in some degree, by a difference of its seat, the lower lobes being more particularly affected in inflammation, the upper lobes in phthsis. The two diseases, however, it should be remembered, are often combined. In determining distinctly the further diagnosis of pneumonia, from the phenomena of condensation and respiratory murmurs, it is necessary to bear in mind the various conditions which may produce condensation of the lung either in the child or in the adult. The observations of Laennec, Legendre, and Baillie, Jörg, Fuchs, Barthez, Rilliet, West, and Gairdner, have especially elucidated this subject, and shown its importance as an element to be attended to in diagnosis. The following is a short statement of the conditions which lead to consolidation of the vesicular tissue of the lung:—(1.) A partial unexpanded state of the lung in a new-born child, termed *atelectasis* (JÖRG). (2.) Consolidation connected with the accumulation of mucus in the bronchi and air-cells—*bronchitis* in the infant, followed by what has been called *lobular pneumonia* of children, the *état fœtal* of the French (FUCHS, WEST, ZENKER). (3.) Collapse of the pulmonary air-cells, causing *lobular* or *more diffuse* forms of pulmonary condensation in adults, as well as in children, due to bronchial obstruction (GAIRDNER). (4.) It is probable that the *hypostatic pneumonia* described by Piorry, and the *péripneumonia des agonisans* of Laennec, and *some* of the so-called *latent pneumonias*, are forms of condensation due to *pulmonary collapse*, combined with serous effusion or vascular congestion. (5.) The consolidation of inflammatory lymph in or about the pulmonary vesicles, so that the vesicular tissue of the lung is imbedded in solid material, "as the stones of a wall are in mortar." (6.) Condensation of the lung from the pressure of pleuritic effusion. (7.) Condensation of the lung from extravasation of blood (apoplexy of the lung), or from tubercular or cancerous deposits; or enlarged bronchial lymphatic glands, which are arranged along the sides of the air-tubes in their passage through the substance of the lungs.

Seeing, therefore, that consolidation may result under such a variety of conditions, the truth of the statement so well expressed by Dr. Stokes must at once appear in the strongest possible light—namely, "That in the cases we are every day called to treat, the value of physical signs must be tested by the history and symptoms (records of temperature and the like), and these in their turn must be corrected by the physical signs." In truth, no disease shows more forcibly than cases of pneumonia do, and especially as regards diagnosis and treatment, that *every individual case of disease requires to be made a special study as regards its individual history, progress, combination, and sequence of symptoms.* In the aged, pneumonia is extremely dangerous, and often difficult to detect.

Prognosis.—Between the ages of fifteen and forty, acute, single, or croupous pneumonia, uncomplicated, is not a dangerous disease. Recovery is the rule, and it is often rapid. Secondary pneumonia, on the other hand, or pneumonia as an intercurrent affection to any of the general, and especially exanthematous disease, is a dangerous lesion, and one apt to be fatal. Pregnancy also is an unfavourable condition; especially during the first six months. After fifty years of age the danger increases. Between forty and fifty years of age about one in five die; after sixty years of age one in two die. Double pneumonia is very dangerous. An elevation of temperature above 106° Fahr., with an increase in the frequency of the pulse over 120 beats per minute, renders the prognosis bad. The disease is attended with extreme danger to aged persons and to drunkards. In advanced life the mortality is between sixty and seventy *per cent.* (NIEMEYER). Absence of all sputa is an unfavourable sign; and so also is the appearance of a very dark, brownish red, commonly called *prune juice* expectoration. It signifies, says Niemeyer, a poor state of nutrition and fragility of the pulmonary capillaries—a general cachectic condition of the individual. Very copious liquid œdematous sputa are also significant of evil. Scanty expectoration during resolution is not specially significant of any danger if the dulness continues to disappear; but if expectoration fails, while gurgling sounds are heard with each respiration in the chest, palsy of the bronchial tubes, and *œdema* of the lungs are indicated, and approaching death.

Treatment.—One of the most discordant topics in the Science of Medicine seems still to be with some—the treatment of pneumonia. At one time, especially about fifty years ago, large bleedings were the rule, and they appear to have been well borne. But, Laennec and Louis seem to have been early impressed, from their experience, that large bleedings were by no means an eminently successful practice, and that in some cases they were absolutely injurious. The same difference of opinion was held with respect to large doses of the *tartrate of antimony.* These discrepancies are now to be explained by the circumstance that pneumonia, like other inflammations, not only varies in type and severity in different cases, but that " extremes in practice," whatever they may be, when adopted as a *rule absolute,* applied to all cases, can never give satisfactory results. (See also p. 761, *ante.*)

The ancients bled in pneumonia, and sometimes to deliquium; and Galen appears to have adopted this practice. This was also the practice of Sydenham; and Laennec says it was common in France at the beginning of the last century to take twenty-four, thirty, and thirty-six ounces of blood at one bleeding. This practice was also followed throughout Europe at that time.

Now, however, it would be an unwarrantable error to make excessive bleeding the basis of remedial measures in all cases (see vol. i., p. 292, *et seq*). At the same time there can be no doubt that we have it in our power materially to modify the course and to shorten the duration of pneumonia by the judicious employment of blood-letting, leeches, tartar emetic, certain salines, and opium (PARKES).

The correlation of the body-temperature, the number of the pulse, and the respirations *per minute,* furnish the best guide as to the course to be pursued in the treatment of pneumonia. If the pneumonia is not intercurrent to some other disease, but happens in a person otherwise healthy; and if the mean body-temperature remains below 104° Fahr., and the pulse does not exceed 120 beats in the minute; and if the respirations never exceed forty in the same time, the case must be regarded as a typically favourable case, which will certainly begin to get well when the cycle of the pneumonic process is complete, *i.e.,* in from three, four, seven or eight, to twelve days without any special medicinal treatment; but with the careful management implied by absolute rest in the horizontal position, and a strictly antiphlogistic diet. The natural history of an uncomplicated pneumonia shows that it has a very definite cyclical course; and that if left to itself in a vigorous patient, uncomplicated by any other ailment, and if it be of the typical intensity as here indicated, it almost always ends in recovery. The expectant mode of treatment of the Vienna School confirms this view, as the statistics collected by Balfour, Magendie, Skoda, Niemeyer, Schmidt, and Legendre fully prove.

Bourgeois (d'Estampes) abstained also from all *active* treatment of his cases of pneumonia during a period of twenty-five years. The result of his experience is that, on an average of all his cases, a decided tendency to amelioration of all the symptoms was apparent by the eighth day. The sputa became less viscid, the breathing easier, the pain in the chest subsided, restlessness or wakefulness lessened or had disappeared, and a desire for food was experienced. By the ninth or tenth day convalescence had fairly commenced (*Union Medicale*, Jan. 1850, vol. i). The results obtained by the *Homœopaths* have also shown this to be the case. Unless warranted, therefore, by such special indications as will be pointed out, active interference has an unfavourable effect upon the course of pneumonia (NIEMEYER); and when treated by blood-letting as a rule, it more often terminates fatally than where no venesection has been practised (DIETT). Blood-letting is no specific. It does not cut short a pneumonia. In epidemic pneumonia, says Laennec, it is hardly possible to bleed the patient without rendering him worse. In the year 1814 pneumonia was very common among the conscripts of France, yet there were few indications for bleeding, and those that were bled were rendered much worse.

With respect, therefore, to bleeding in cases of pneumonia, much must be left to the discretion of the practitioner. That there are cases in which the patient can only be saved by general blood-letting, everybody must admit; while, on the contrary, when pneumonia is epidemic, the quantity of blood drawn must be greatly limited and the case well watched. The ancients held that bleeding should not be practised after the fifth day, as it prevented *concoction*. In other words, it tended to change the type of the disease in its natural tendency to a cure, so that the processes tending to the resolution of the inflammation by the natural *cell-therapeutics* of the part were altered for the worse. The cases of Louis appear to establish the propriety of bleeding early as a general rule, if blood-letting is to be used at all; for he says those bled in the first four days of the affection are cured four or five days sooner than those who are bled later in the disease. The experience of Dr. Alison is similar. In short, as a general rule, the earlier the inflammatory state is detected (if possible before the third day—ALISON) the more likely will bleeding be followed by well-marked beneficial results—the disease will be sooner cured, and the convalescence more rapid and perfect.

From what has been said at p. 291, vol. i., under the management of inflammation, and adopting the indications furnished by Niemeyer, blood-letting should only be had recourse to when the following special indications for it arise:—

(1.) When the pneumonia attacks a vigorous and hitherto healthy person, is of recent occurrence [not exceeding fifty or sixty hours after the rigor], the temperature being at or exceeding 105° Fahr., and the frequency of the pulse as much as 120 per minute.

The object of the venesection here is to reduce temperature, lessen the frequency of the pulse, and so lessen the danger from the violence of the fever.

(2.) When collateral œdema in the portions of the lung unaffected by pneumonia is causing danger to life, the presence of the blood is reduced by bleeding, and farther transudation of serum into the air-vesicles is prevented. When the great frequency of respiration is due to the pneumonic process alone—not to fever or pain—and as soon as a serous foamy expectoration appears, together with a respiration of forty or fifty breaths a minute at the commencement of pneumonia; and when the rattles in the chest do not cease for a time, after the patient has coughed as well as he can, copious blood-letting is indicated to reduce the mass of blood, and to moderate collateral pressure.

(3.) When there are symptoms of cerebral pressure indicated by stupor or transient paralysis.

Antimony is another remedy which has gone through several extremes of administration also. Rasori, in modern times, introduced the practice of giving large doses of *tartar emetic* in the treatment of pneumonia, and Laennec was so dissatisfied with his own results of bleeding that he adopted Rasori's method. He claimed great success at first, which, however, appears to have been of short continuance; for M. Lagarde afterwards published an account of 16 cases treated by Laennec by this method, of whom 5 died. Lecoultreaux has given a list of 30 cases likewise treated by Laennec, and of whom 12 died. Neither have other physicians in other years been more fortunate with this extreme (or Rasorian) method of giving antimony. Louis treated 15 cases according to this method, and 6 died; Chomel, 140 cases, and 40 died; and Gueneau de Mussy treated 90 cases, of whom 38 died. Andral has treated a considerable number of cases of pneumonia by tartar emetic, in quantities varying from six to thirty-two grains in the twenty-four hours; and although it "may therefore be given with impunity in much larger doses, yet," he says, "I have not seen pneumonia ameliorated by such large doses of this medicine; for neither has it appeared to do good when borne by the stomach, nor when it has excited distressing nausea and vomiting." Few, therefore, if any, now believe in the usefulness of such large doses of antimony; and the use of tartar emetic, formerly so common, has of late years fallen into discredit.

The Rasorian method is decidedly contra-indicated in the pneumonia of old people; but when the remedy is cautiously administered as a sedative, or diaphoretic and expectorant, in doses of *the eighth* or *twelfth of a grain* every *two* or *three* hours, and in doses of a *sixth* or a *quarter of a grain*, in the more sthenic attacks, it is a most valuable remedy (MACLACHLAN). It is contra-indicated in gastric or gastro-enteric irritation. There are cases where the remedy produces severe depression and nausea—cases in which the stomach will retain *no* food, scarcely even water when given alone. It is under such circumstances that Dr. Anstie, with justice, advocates the administration of alcohol in stimulant doses often repeated. He cites one case of double pneumonia in a man aged twenty-four, who, beginning with twelve ounces of brandy *per diem*, ultimately took twenty-four ounces in divided doses every half hour, and for *ten* days lived on *nothing else*, and a little water. In one month he was able to resume his work. A child fourteen months old, under similar morbid conditions, was similarly treated with port wine and water. Taking about six ounces of wine a-day, in spoonful doses, with water, the child subsisted on the wine and water *alone* for twelve days. At the end of that time, the stomach still refusing food, a little cod-liver oil was substituted for the wine, and in ten days more the appetite for ordinary food began to show itself, and the child made a good recovery (*Stimulants and Narcotics,* by Dr. Anstie, p. 446).

In the statistics which bear upon the treatment of pneumonia little or no reliance can be placed as a guide to practice. The results revealed are so variable and contradictory as to deprive them of the slightest claim to authority. For example:—*Without depletion,* Dr. Bennett's statistics show a mortality of 1 in 21·4; Dietl's, 1 in 13; the homeopathic treatment (mainly expectant), 1 in 6; Vienna, 1856, 1 in 4. *With antimony and bleeding,* Grisolle lost 1 in 8; Dr. Bell, Glasgow, 1 in 17·7; Trousseau, 1 in 26; Wossildo, 9 in 76. *Treatment by the inhalation of chloroform,* Baumgartner, 1 in 10; Varrentrapp, 1 in 23.

Former statistics in the Royal Infirmary of Edinburgh are said to show a mortality of 1 in 3 to 1 in 6, when blood-letting was freely practised.

These results are clearly too variable a guide to any sound basis of practice. No satisfactory conclusion can be drawn from cases massed together. Each case must be studied by itself, as well as each epidemic, (see also p. 761, *et seq.*)

The numerous qualifying conditions connected with age, season, climate, epidemic and endemic influences, earliness of treatment, stage, extent, and complications, all tend to modify the line of treatment required. Dr. Lawson, Professor of Medicine in Cincinnati College, has well expressed these conditions, in a general sense, under the designation of "*the individuality*" of each case.

> "Indeed," he remarks, "so great are the differences in constitutions, that no two examples will exhibit the same characteristics throughout, nor will they admit of precisely the same method of treatment. It is a due appreciation of these more minor shades of differences, as well as the broad distinctions observed in the varying *forms* of the disease, that denotes the truly skilful physician, and which enables him to meet the emergencies of each case, instead of relying on conclusions drawn from groups of cases. It has been remarked that English physicians are apt to think more of some other case they have seen than the one under treatment; while the French think more of the disease than of the patient. Hence English physicians *individualize* the disease; the French physicians *generalize* the patient.
>
> "The true course has been indicated by Hufeland—namely, *to generalize the disease and individualize the patient.* The tendency of statistics is undoubtedly to rob each case of its individuality. Thus, one group of cases will all be bled, another will receive tartar emetic, a third will be left to the chances of Nature. In the first group some are bled who ought to have been stimulated; in the second group *tartar emetic* is administered when *bleeding* would have been preferable; in the third group some are permitted to die from over-action. Thus the individuality of a case is ignored, and the physician prescribes for a mere *name.*"

A rational treatment is one which must secure to each case its own individuality and consideration. And as the shades of differences and the corresponding modifications of treatment cannot be expressed in *groups*, statistics in this sense become simply an impossibility. For example, bleeding, antimony, mercury, and blisters may be demanded in one case; quinine, opium, and wine in the next; a third may require but little interference beyond a well-regulated diet, with moderate stimulants and absolute rest.

The treatment of pneumonia, therefore, demands not a single, but many agents.

It is the proper *combination* of remedies, not a single agent or mode of practice, which must be sought for in its treatment (LAWSON); but now we know that the so-called "rational" or "restorative" mode of treatment is the one to be adopted; and it only remains to add the more matured opinions entertained as to the line of treatment to be adopted with pneumonia as it now occurs.

In cases of sthenic pneumonia characterized by intense heat, above 104° Fahr., dryness of the skin, a full resistant pulse of 120, rusty coloured expectoration, *great oppression of the breathing*, and to the extent of beyond forty respirations per minute, blood-letting, had recourse to at the beginning of the attack, in the stage of congestion before the *fastigium* of the pyrexia (not later than forty or fifty hours after the initial rigor), not only affords immediate relief to the breathing, but appears to remove the extreme tension of the vascular system and to promote secretion. The most suitable time is that during the evening exacerbation (HUSS). The indications for blood-letting, as already given, should be decidedly marked before it is undertaken; and the amount of blood to be lost must vary in each case, according to the oppression of breathing, the type of the disorder, and the constitution of the patient, as well as to whether or not there is any prevalent epidemic tendency associated with the pneumonia. It is seldom necessary to draw more than from *ten* to *sixteen* ounces; and *eight* or *ten* ounces will usually suffice. After bleeding, the pain in many instances ceases, expectoration takes place more easily, and alters in character; the skin becomes moister, and evidence is afforded of the action of remedies which before proved inoperative (FULLER).

In very many cases it is now either unncessary or inexpedient to let blood—inexpedient chiefly because of the constitution of the patient being shattered by excesses, constitutional disease, anxiety, and mental distress; and excessive blood-letting, under whatever circumstances practised, impairs the strength, leads to great impoverishment of the blood, arrests the actions on which the absorption of exudation-matter depends, exposes the patient to risk, and induces a tardy convalescence.

Bleeding, therefore, ought certainly not to be employed after extensive consolidation of the lung has taken place.

A combination of *antimony* and *calomel* is believed to have saved a much larger number of cases than *antimony* alone; a quarter of a grain to a grain of the *tartrate of antimony*, combined with one grain of *calomel*, given every four or every six hours, according to the severity of the disease, is the treatment in some cases to be adopted. Previous to its use the bowels should be well cleared out; and after the mercurial effects are indicated by the condition of the gums, the further administration of the remedy should cease.

In cases of simple serous pneumonia even simpler remedies are sufficient. Two grains of *ipecacuanha* given every four or six hours have frequently been followed by the recovery of the patient. The cases in which *tartar emetic*, in small doses ($\frac{1}{8}$th of a grain), fails to exercise a curative action are those in which hepatization proceeds with extreme rapidity; in which crepitation either does not exist at all, or is of very short duration, giving place after a few hours to intense tubular breathing; and those which are marked by extreme depression almost from the first.

Salines and *stimulants*, with *calomel* and *opium*, if necessary, are found to be the most efficient means of treatment in such cases. The efficacy of *mercury*, in the experience of Dr. Fuller, is most conspicuous in those cases of pneumonia in which *tartar emetic* is of least avail; in other words, in those instances in which the productive results of inflammation are the greatest, in cases in which crepitation does not exist at all, or is replaced in a few hours by intense tubular breathing (*On the Chest*, p. 238). In these, *calomel* and *opium*, in combination with *salines* and small doses of *tartar emetic*, will often produce very beneficial results.

Of many other proposed remedies it may be necessary to notice the influence of the following, namely:—

Aconite, carbonate of ammonia, digitalis, cold water compresses, linseed poultices, turpentine, opium, hydrochlorate of ammonia, iodide of potassium, and quinine.

(a.) *Aconite* is to be given at the very outset or first stage of pneumonia, from iii.♏ to v.♏ of the tincture of B. Ph. every four hours (WILKS); or from ii.♏ to iii.♏ every three hours (REITH). Dr. Ringer recommends half a drop to one drop of the tincture in a tea-spoonful of water every ten or fifteen minutes for two hours, and afterwards to be continued every hour. In such modes and doses of administration it has been useful in *febrile catarrh* and *catarrhal croup*, and may be useful also in some of the phases of pneumonia, where it may be indicated at the outset, especially to subdue increased action of the heart and circulation, and reduce the abnormally high temperature. Care must be taken that the remedy is not combined with alkalies. Dr. Reith speaks very highly of the good effects of *aconite* at the outset of the congestive stage of pneumonia (*Ed. Med. Journal*, Oct., 1868, p. 905).

(b.) *Carbonate of ammonia.*—Dr. Waters, in his work on *Diseases of the Chest*, speaks highly of a combination of *ammonia* and *chloroform* in the treatment of pneumonia, where antimony in small doses is not indicated. The following prescription, to be taken every three or four hours, combines the beneficial properties of both stimulants:—

℞. *Ammon. Carb.*, gr. iv.; *Spt. Chloroformi*, ♏xx.; *Aq. Camph.*, ℥x.; *misce.*

Dr. R. C. Styles, of New York, has been accustomed to give the *carbonate of ammonia* in *one drachm doses* in solution every two or three hours; and by a series of carefully conducted experiments he concludes that it is converted into the hydro-chlorate of ammonia in the stomach, and no longer acts as a stimulant (*Med. Record*, Oct. 1., 1866.*)

*** Quoted from American Edition of this Work.**

If such a statement is correct, why not give the *muriate of ammonia* at once, in place of throwing the burden upon the stomach of the patient of transforming the *carbonate* into the *muriate?* There may come a time when the stomach and its surroundings may not be equal to this feat. The local action of *carbonate of ammonia*, in doses above ten or twelve grains (and these repeated), is that of an irritant and corrosive. In doses of about thirty grains it occasions vomiting, and in large doses it excites pain, inflammation, and all the consequences of an irritant poison. Its remote action is equivalent to that of *caustic ammonia* (PEREIRA). From three to ten grains are the limits of the dose of *carbonate of ammonia* in Great Britain. But American stomachs may be stronger than ours; or the *carbonate of ammonia* used in America may not be so strong or so pure as it is in this country.

(c.) *Digitalis* has of late years been employed in the treatment of pneumonia by Oppolzer, Traube, Schneider, Niemeyer, and others, as one of the best agents for the relief of the febrile symptoms. It lowers the temperature and diminishes the frequency of the pulse, without exercising so weakening and depressing an effect upon the system as bleeding. A pulse of 100 to 120 in frequency is an indication for its employment—a less frequent pulse does not require it. It is usual to give it in the form of *infusion*, combined with the neutral salts of *nitrate of potass* and *soda*.

(d.) *Opium* may generally relieve the pain of the side, and diminish the distressing cough of pneumonia; and is of use in warding off the delirium which is apt to supervene on pneumonia. It ought to be given when there is want of sleep, restlessness, and staring of the eyes, with slight tremors of the hands. Opium, combined with stimulants and nourishment, may avert the attack (WATERS). It may be used hypodermically.

(e.) *Hydrochlorate of ammonia* is of use in congestion of the lungs, in the advanced stages of *pneumonia*, and in the cough of old age. The following prescription is often productive of good results:—

℞ *Ammon. hydrochlor.*, ʒi.; *Ext. Glycyrrh.*, ʒi.; *Spt. Æther Sulph.*, *f* ʒii.; *Aquæ ad f* ℥iv.; *misce*. A table-spoonful to be given for a dose every two or three hours (WARING).

For the past twenty years Dr. Clymer has substituted *muriate of ammonia* in place of calomel in the second stage of pneumonia, giving it in *one* or *two* grain doses every hour or two.

(f.) *Iodide of potassium* in the advanced stages of pneumonia appears to promote the absorption of effusions and indurations.

(g.) *Quinine* is of use chiefly in the advanced stages of pneumonia when the patient is old, the constitution debilitated, and typhoid symptoms prevail. It is best given in combination with *sulphuric acid*, the dose being *five grains* every third hour. The pulse ought to become slower and steadier under its use, and the respiration freer. According to the experience of Niemeyer, quinine should be given in doses of *two grains* every two hours, or in two or three *ten grain* doses within a few hours, whenever danger threatens from *excessive elevation of the temperature of the body*.

(h.) *Veratria*, in the hands of Vogt and Biermer, has proved of service in reducing pulse and lowering temperature. To obtain its full effect, $\frac{1}{20}$th of a grain of the active principle *veratrine*, or $\frac{1}{6}$th of a grain of the resin of *veratri veride*, should be given in the form of a pill. When the body-temperature runs high, short of the necesssity of blood-letting, the reported results of Vogt and Biermer warrant more extended trials with care and watchfulness, as it tends to induce great prostration, collapse, vomiting, and diarrhœa (Waring, Niemeyer).

(i.) *Alkalies.*—Dr. John Popham, Physician to the Cork North Infirmary since 1865, has treated twenty-eight cases of pneumonia by alkalies. They all recovered. In some the signs of engorgement only existed at the time of admission; in others, hepatization was found. The reliable signs of pneumonia were present in all—*e. g.*, localized dulness, crepitation, tubular breathing, rusty sputa (in many), and labial herpes in about half. Six were cases of double pneumonia. *Bicarbonate* of potash was given, largely diluted in mucilaginous liquids, five grains up to thirty grains in each dose,—four, six, or even eight doses being given in the twenty-four hours. An adult took to the extent of two to three drachms of the salt per day. The evidence of its good effects appeared first in altering the character of the expectoration on the second or third day of its use. The viscid sputa became resolved; the fine bubbles became course and large; the rubiginous colour of the expectoration was changed to white; its tenacity of adhesion was lessened, so that it was brought up easily; and the cough, instead of continuing dry, harsh, and irritative, became moist, soft, and expulsive. Thus the alkali acted as a sedative, allaying the cough and abating the congestive state on which it depends. The white pasty fur over the tongue dissolved away in an increased flow of saliva. The urine became alkaline, and the physical signs of pneumonia became resolved. "Flying blisters," applied for four or six hours, so as to redden the skin (not for suppuration), is a valuable auxiliary. Suppuration from blistering is exhaustive and prejudicial (*Brit. Med. Jour.*, Dec. 28, 1867, p. 586).

(k.) *Stimulants.*—The treatment by repeated stimulants alone is but the opposite extreme of practice to excessive blood-letting, and will no doubt soon find its level and appropriate place as a valuable aid to other remedies, especially in patients whose constitutions are depressed under the influence of life imposed upon them by *the "great town system."*

When pneumonia tends to death by exhaustion, as the disease advances; when exudation has been excessive, the fever prolonged, or the constitution debilitated before the attack, such a state of complete adynamia requires the use of stimulants, in the form of large doses of *camphor, mint, benzoic acid,* alcoholic drinks; and in addition, concentrated soups and milk are called for in small quantities, given often. In old persons such remedies as stimulants, generous diet, preparations of quinine and iron are indicated from the very commencement. Alcoholic stimulants are rarely required in young persons or in previously healthy adults.

Some local applications, more recently recommended, also require special notice, namely:—

(l.) *Cold Applications.*—Of these Niemeyer has made extensive use in the treatment of pneumonia, and strongly recommends the practice. In all cases he covers the chest of the patient, and the affected side in particular, with cloths that have been dipped in cold water and well wrung out. Compresses thus made must be repeated every *five* minutes.

The procedure is unpleasant in almost all cases; yet even after a few hours the patients experience a material relief. The pain, dyspnœa, and frequency of the pulse are reduced, and the body temperature may go down an entire degree. Smoler, in the Hospital of Prague (where every pneumonia is treated with such cold compresses), confirms the experience of Niemeyer, and both acknowledge the treatment to be not merely palliative, but as tending to shorten the duration of the illness, and hastening convalescence. Few cases delay to improve beyond the seventh day, many improve on the fifth, and a very large number as early as the third day. The remedy is justly held in repute for *meningitis* and *peritonitis;* and hence it may fairly claim to have a similar good effect in pneumonia.

(m.) *Linseed Meal Poultices.*—On the other hand, heat combined with moisture, as by this poultice, made as light and soft as possible, and continued for a long time, made sufficiently large as thoroughly to enclose the affected side, and changed as often as it gets unpleasant, not only affords present relief, but seems to exercise a favourable influence on the course of the disease. The skin may be at the same time stimulated by mustard, or terebinthine applications added to the poultice.

(n.) *Turpentine.*—Hot turpentine fomentations may be applied over the affected side also with advantage.

But, as a rule, under certain conditions, any or all of these remedies may be dispensed with. The natural course of the disease is to go on, and that rapidly, to a spontaneous favourable termination, by a process of resolution through which the solidified lung-tissue regains its natural and healthy state, independently of remedies, so that time is an important element in the cure; but as particular phases in the course of the pneumonic cycle may call for special treatment, so much the more successful will the management of the case be if the indications for interference are properly understood and acted upon. Those remedies that have now been specially noticed may each, if judiciously used, be of service in safely and surely controlling the circulation when the indications for treatment are short of those mentioned as requiring the agency of blood-letting. Their effects must be watched so that they do not lower the vital powers nor disturb the digestive functions. The indications for nourishment, properly adjusted to the circumstances and conditions of each case, and each stage of the case, are of paramount importance from the very first; and the happy issue of a case of pneumonia depends quite as much upon early and properly feeding the patient as a case of typhus or enteric fever does. A milk diet should be given from the commencement, to be followed by beef-tea and meat-stock soups as the disease advances and the pulse softens. Stimulants in any form cannot be regarded as the equivalent of food (Clymer).

ABSCESS OF THE LUNG.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abcès;* GERMAN EQ., *Abscess;* ITALIAN EQ., *Ascesso.*

Definition.—*A circumscribed collection of pus in the lung in the form of an abscess.*

Pathology.—Although pus is more commonly diffused through the pulmonary parenchyma, yet sometimes it is collected into an abscess. In the infancy of pathology physicians regarded phlegmonous abscess of the lung as a common and ordinary occurrence, but it is extremely rare; and Laennec, when he published the first edition of his work, had only met with six cases, notwithstanding his extensive research; and in the practice of other physicians phlegmonous abscess of the lung is equally uncommon. Louis and Andral have each seen it *once;* Trousseau has seen it *twice;* Chomel has seen it *three* times during a period of twenty-five years; Flint, out of 133 cases, met with it *four* times; Chambers, at St. George's Hospital, out of 600 cases, found it in *three;* and in Vienna, out of 750 cases treated in the hospital there, in 1847 to 1850, pulmonary abscess was observed only in a single case (FLINT). Abscess of the lung (although termed phlegmonous, to distinguish it from tubercular abscess) generally exists without any great intensity of inflammation, or other considerable alteration of its tissue.

They vary in number and in size. If single, they are generally about the size of a hen's egg; if multiple, they vary in size from that of a filbert, or even less, to a walnut. The cavity is often irregular and divided by bands of tissue. They increase in size from continual melting down of the surrounding inflamed tissue, and several may coalesce, so that finally a very large abscess may take up nearly the whole lung. It opens into the bronchi, or into the pleural sac, when the lesion is at once fatal.

In the experience of Dr. Maclachlan, however, the rarified condition of the lung in the aged "seems to favour the formation of small abscesses, which are occasionally seen interspersed through the red and gray consolidated tissues, as if certain air-cells had broken down and coalesced during the plastic or suppurative process, and formed so many sacs for the reception of the effused or secreted matter (l. c., p. 279).

Pneumonia sometimes, though rarely, terminates in abscess.

Symptoms.—The physical symptoms previously to the bursting of the abscess are those of hepatization; but supposing the abscess to have burst into the bronchial tubes, the pus of course escapes, and a cavity filled with air is left, communicating with the bronchial tubes, and this new state of parts gives rise to a new series of phenomena. The air, for instance, having penetrated into the cavity, the part which returned a dull sound while the abscess was yet unbroken, will now return a sharper and clearer sound on percussion than natural, denoting a larger admission of air than takes place in health. Again, on auscultating the chest, we find some changes have taken place both in respiration and in the transmission of the voice. If the cavity, for example, be large, and the opening small, the natural respiratory murmur at that part will be superseded by a sound resembling a person blowing into a jug, and from this circumstance termed by Laennec, " râle amphorique," or " bottle sound." Again, if the cavity be large and its walls dense, and the abscess still contains some pus, we hear a sound as if a drop of water had fallen into a pool; and this sound is so sharp and metallic that it has still preserved the designation, originally given to it by Laennec, of *metallic tinkling*. It is usually supposed that this sound is produced by a globule of pus or fluid dropping from above into the fluid below; but some are inclined to believe that it is owing to the bursting of a bubble of air mixed with the pus of the abscess. If, on the other hand, the abscess be large, and contains some pus, on the patient coughing we actually hear a *splashing sound* of the pus against the walls of the abscess, especially if the chest of the patient be quickly moved to and fro (succussion).

In aged persons, pneumonic abscesses are generally numerous, of limited extent, and interspersed through the lung. Physical diagnosis of them is therefore scarcely possible; but if the abscess be large, the sudden evacuation of purulent sputa, with gurgling and cavernous respiration, denotes the circumscribed suppuration of an abscess cavity.

Another circumstance revealed by auscultation, in the event of an abscess, is *pectoriloquy*. This physical sign is heard when, the stethoscope being applied to the chest and the patient desired to talk, we hear his voice as if he were speaking directly at the end of the stethoscope, the sound passing to the ear as through an ear-trumpet.

Pectoriloquy, however, does not take place in all cases of abscess of the lung: its occurrence may be considered the exception rather than the rule in this disease. The cause of this is, that many conditions are necessary to its existence; first, that the lung must be condensed so as to have some conducting power, or the voice will be destroyed, as in health, before it reaches the aperture communicating with the abscess. Again, it is necessary that the patient should have a sufficient quantity of voice to produce strong vibration; but this is often wanting. Another condition is, that the bronchial opening of the abscess be not too large; for in that case the vibrating force is diminished. It is likewise injurious to the effect that there should be not more than one opening into the abscess; for in that case not only is the vibrating force diminished, but the counter motions of sound destroy all vibration. It is plain, also, that the walls of the abscess must have a certain density, or their flaccidity will act as a damper and destroy all vibration. Many conditions, therefore, are necessary to pectoriloquy; and we cannot feel surprised that one or more of these signs of an abscess cavity may be wanting.

Besides, on opening into the bronchial tube, the abscess may at the same time open into the cavity of the chest, and this new pathological state gives rise to the sound of a *metallic tinkling* in the chest infinitely more powerful than that caused by a simple pulmonary abscess. Indeed, the intensity and sharpness of the sound quite equals that returned by a copper vessel when struck with a slight force; for the intercostal muscles brace the walls of the chest like a drum, so that they become an excellent conductor of sound. The immediate cause of the sound is supposed to be exactly the same as when it results from an abscess; that is, either a drop of fluid falling into the pus below, or the extrication of a bubble of air from the gravitated pus. The chest in these cases always returns a remarkably clear sound on percussion beyond the precincts of the fluid.

Pyæmic Inflammation and Abscess. — In some classes of cases, as in the *typhus* and *enteric fever*, pneumonia occurs as a local lesion, and is sometimes named "typhoid pneumonia;" so also in another class of cases, characterized by *blood-poisoning* or by *embolism*, the lung disease is a secondary result, and is said to be due to pyæmia. While in yet another class of cases, as in those where some morbid condition of the constitution prevails—as in the alcoholism of drunkards—a primary pneumonia in them may give rise to blood-poisoning and the phenomena of pyæmia, by the sanious decay of the exudation and its re-absorption during resolution, or its passage to the left side of the heart.

The pneumonia of such cases may be either single or double—that is, it may attack one or both lungs at the same time. Of the part of the lung attacked, inflammation of the inferior lobe is most frequent; next, of the superior lobe; while rarely the whole lung is inflamed.

Bronchitis may also take place in such cases without pneumonia; but in many cases pneumonia follows as a consequence. Pneumonia also may take place without bronchitis, but in general bronchitis accompanies it. Pneumonia also may take place without pleuritis, but it generally happens that the pleura is more or less affected; and it is so especially in the pneumonia of pyæmia. In old persons primary pneumonia is mostly lobar, and the disease should be particularly looked for at the inferior and posterior parts of the chest. It runs its course very rapidly to purulent infiltration, and the *prodromata* are not distinctly marked. Parotitis is a frequent complication, and of bad omen, and the pneumonia may be marked by cerebral phenomena.

GANGRENE OF THE LUNG.

Latin Eq., *Gangræna;* French Eq., *Gangrène;* German Eq., *Brand*—Syn., *Gangrän;* Italian Eq., *Gangrena.*

Definition.—*Disintegration and breaking up into fragments of the filamentous tissue of the lung. Every simple element of the tissue is so changed that neither blood-fibres nor epithelium can be recognized in a sound state. The texture becomes broken up, and shreds of fibrous tissue may here and there be distinguished; but the whole mass becomes converted into a heap of amorphous granular matter, of a yellowish-brown or black colour, mingled with drops of oil. The tissue becomes soft and flaccid, in some parts perfectly liquescent, and generally emits a fetid smell.*

Pathology.—The comparative rarity, the almost invariable fatality, of pulmonary gangrene, obtains for it a melancholy interest whenever it occurs; and since Laennec directed attention to its peculiar characters, many pathologists have contributed to the records of this disease. But notwithstanding the researches of Andral (1822), of Lorimer, of Schroeder Van der Kolk (1826), of Bright (1827), of Cruveilhier (1833), of Guislain (1836), of Craigie (1841), of Ernest Boudet (1843), of Silfverberg (1856), of Walshe (1860), of Fuller (1862), gangrene of the lung is still a condition at all times difficult of diagnosis in the first instance; and its existence has often been unknown until the disgusting *post-mortem* appearances proclaimed the condition which general symptoms during life had failed to disclose, and which in some cases hardly even suggested a suspicion of the real nature of the affection. In gangrene of the lung the dead part putrifies and undergoes chemical decomposition, a condition favoured by contact with the air it meets with in the lungs.

Symptoms.—The diagnostic symptoms of gangrene of the lungs which have been most frequently recorded are derived from the expression of the countenance becoming small, pinched, contracted, haggard, ghastly, miserable, and death-like; eyes sunk and void of lustre; patient squeamish and languid, with occasional vomiting, and a feeling of indifference to all external objects,—some or all of which symptoms may or may not be associated with an intolerable fetor of the breath, and which, when it is present with these symptoms, may be considered conclusive of the existence of gangrene. Not one of all these symptoms, however, may manifest themselves, and yet gangrene of the lung may exist. From the recorded cases, therefore, as well as from those which I have myself seen, it may be of some use to classify the conditions of disease in which pulmonary gangrene has occurred.

The termination of acute sthenic pneumonia, in an otherwise healthy person, by gangrene of the lung, is by all authors considered as one of the least frequent terminations of that disease. Morgagni only records one instance; and Laennec is reported to have seen only six or eight during the whole course of his practice. But there can be no doubt that pneumonia does sometimes terminate in gangrene of the lung, as an accidental or occasional complication, as has been clearly shown by clinical and *post-mortem* evidence. The insidious and often sudden mode of its attack, the sudden, remarkable, and generally fatal collapse which supervenes, show, at the same time, that no local condition is suspected or considered sufficient to account for the presence of gangrene. The extent to which the death and destruction of lung-tissue takes place also varies much. Sometimes nearly the whole of a lung passes at once into a gangrenous mass. In other cases only a small portion in the centre of an exudation will die; but in every case there is the formation of a slough, its liquefaction, and the formation of a cavity. Sometimes a line of separation between dead and living parts is attempted to be formed; while in other cases no evidence exists of any limit to the extent of the destruction; and this latter diffuse form of gangrene I have generally found associated with cases in which the blood was greatly changed, as in typhus fever, or in which the nervous functions were impaired, as in maniacs, or in the paralysis of the insane. Of fifteen cases recorded by Guislain, all were maniacs. Of three cases recorded by Van der Kolk, one was a maniac, and two laboured under some peculiar nervous condition of the body. Of six cases recorded by Dr. Craigie, two were mentally deranged, having lesions in the brain; two suffered from *Bright's disease* and *mercurialism;* one had *variola;* and one had *typhus.* Of fifteen cases in the Royal Infirmary of Edinburgh when I was a student there, four had *typhus;* three had fever, the type of which is not specified; four suffered from ill-health and *mercurialism;* one suffered from a blow; and one had a lesion in the brain.

These cases may be classified as follows:—

Nineteen cases were associated with, and doubtless influenced by, lesions in the great nervous centres; and under this class are included the insane.

Nineteen cases were associated with, and doubtless influenced by, such morbid conditions of the blood as exist in *typhus fever, variola, tuberculosis, Bright's disease, mercurialism.*

One case was associated with and influenced by arterial obstruction, causing pressure upon the entire mass of the affected parts and total absence of circulation in them (Aitken, *Edin. Med. and Surg. Journal,* No. 178, 1848).

The frequency with which lesions of the lung co-exist with and succeed to lesion of the brain renders it apparent that the condition of the lung is materially affected by the influence of the nervous centres. In the cases recorded by Andral and Bright, diseases of the lung, frequently going on to gangrene, were often the immediate cause of death in persons suffering from cerebral disease; and it was also observed that the tendency of inflammation of the lung to pass into gangrene was promoted by the existence of disease of the brain. Cruveilhier also directed attention to the frequency of gangrene of the lung in epileptic cases; and the insane generally are prone to the disease when the bodily health has suffered, or where, as in maniacs, a greatly depressed state of the animal functions succeeds to inordinate nervous excitement.

The following table, modified from that by Dr. Walshe, describes the conditions under which gangrene of the lung occurs:—

I. Pulmonary diseases.	*Pneumonia,* acute and chronic; tuberculization; cancer; hæmorrhage; hydatids (Walshe); apoplectic foci in the lung; acute and chronic dilatation of the bronchial tubes; pleuritis; bronchitis (Silfverberg).
II. Other thoracic diseases.	*Cardiac:* acute endocarditis of the right side of the heart. *Mediastinal:* tumor. *Aortic:* aneurism,
III. Diseases in which the blood is materially changed.	*Animal venoms:* stings of certain insects. *Morbid poisons:* Glanders; exanthemata; typhoid fever; Bright's disease; alcoholism. Scurvy; purpura; septicæmia; pyæmia. *Other poisonous agents:* poisonous gases; mercurialism.
IV. Chronic abdominal disease (Silfverberg).	
V. Perverted innervation.	Epilepsy; insanity; organic cerebral disease.
VI. Traumatic.	

The disease may occur at any age; but it appears to be more common in children than in adults; and more so in adults than in persons much advanced in life.

The symptoms vary according as the lesion is *diffused* or *circumscribed:* —(1.) In the diffused form the symptoms and progress of the case are extremely rapid. Utter prostration of strength; oppressed breathing; a small, weak, and frequent pulse; pallor and anxiety of countenance; and all the general appearances of intense adynamia, are those which characterize the disease. There is also frequent but feeble cough, with profuse, frothy, and diffluent expectoration, of a peculiar greenish colour and intensely fetid gangrenous odour. But the power to expectorate is soon lost; and ere long the vital powers seem utterly oppressed or exhausted; the pulse fails, the features collapse, and the patient rapidly sinks—death generally occurring from apnœa. (2.) In the *circumscribed* forms of gangrene the train of symptoms varies greatly at different periods of the disease. At first, signs of pulmonary congestion are coupled with an amount of prostration quite out of proportion with the apparent extent of local lesion—a prostration which is indeed sometimes the only prominent feature in the case. After a time expectoration commences; at first, muco-purulent, rarely bloody in adults, frequently so in infants and children (if they expectorate at all), and generally emitting a disagreeable odour the moment a communication is established between the bronchial tubes and the dead tissue. The sputa then lose their muco-purulent character, and become extremely liquid, sero-purulent, and of a dirty greenish, yellowish-brown, or ash-grey colour. They exhale an odour distinctly gangrenous, while the breath acquires the same offensive putrid smell, resembling somewhat that of wet mortar, or its fetor is one *sui generis.* The pulse becomes feeble and rapid, with every evidence of great and increasing prostration. The patient passes rapidly into a state of collapse, and sinks in a few hours or days, without the occurrence of any other change, unless profuse hæmorrhage occurs, which terminates in death (WALSHE, FULLER).

Treatment.—The chief reliance is to be placed in all forms of stimulants, combined with such tonics as *bark* or *quinine* in full and repeated doses. Dr. Walshe recommends that repeated doses of an ounce of *yeast* are deserving of investigation, as well as the influence of *chlorate of potass.* He is disposed to put more confidence in them than in *ammonia,* the beneficial effects of which he has never seen demonstrated. Dr. Fuller has not found *ammonia* so useful as the *mineral* acids. Inhalation of the vapour of turpentine poured upon boiling water, as recommended by Skoda, is reported to exercise a distinctly remedial power. The *mineral acids,* especially *nitro-muriatic,* combined with *quinine,* are the main remedies in the chronic state of this disease. A generous diet, easily digestible, with as much malt liquor as can be taken, are recommended as most suitable for such cases.

PASSIVE CONGESTION OF THE LUNGS.

LATIN EQ., *Congestio passiva;* FRENCH EQ., *Congestion passive;* GERMAN EQ., *Passive congestion;* ITALIAN EQ., *Congestione passiva.*

Definition.—*Over-fulness of blood in the capillaries of the lung, resulting in the discharge of blood by expectoration, or hæmoptysis.*

Pathology, (see p. 70, vol. i.)—*Hæmoptyses* are hæmorrhages occurring through the air-passages. The amount expectorated may be small—mere specks or streaks on the sputa; at other times a few ounces, or even pounds, causing great alarm to the patient or his friends, and sometimes ending in suffocation. Its occurrence is always suggestive of organic disease of the chest, such as engorgement of the pulmonary vessels, giving way of pulmonary tissue, indicating disease of the heart, or pulmonary phthisis, or pneumonia, or thoracic aneurism, or cancer of the lung; and consequently *hæmoptyses* have attracted the attention of medical men ever since medicine as a science began to attract attention.

Cases are also recorded of its so-called idiopathic occurrence, as from variations (sudden) of atmospheric pressure, ascending high mountains, or descending in diving-bells, violent straining efforts, or from plethora; but in such cases, according to the experience of Drs. Fuller, Walshe, and others, "there is usually some latent mischief in the chest—some local cause of pulmonary congestion—some mechanical interference with the capillary circulation through the lungs." Spitting of blood may even precede for years the fatal development of pulmonary phthisis, with which it seems to be most prevalently connected; and hæmoptysis is often hereditary.

The source of the hæmorrhages, however—*i.e.*, the absolute demonstration of the vessel or vessels whence the blood has come—has almost always escaped observation; and one of the best accounts of investigations in this direction has been given by Dr. Vald. Rasmussen, of Copenhagen (already referred to), which has been translated in the *Edinburgh Medical Journal* for October, 1868, by Dr. William D. Moore.

Early medical literature is rich in reports of profuse hæmorrhages; and in many the quantity of blood said to have been brought up borders on the incredible—*e.g.*, thirty pounds in three hours (RHODIUS, quoted by Rasmussen).

Rare and imperfect information exists as to the source of the hæmoptyses, as indicated by Dr. Rasmussen. *Varices* are spoken of by Gilibert as a cause of hæmoptysis and phthisis; Morgagni speaks of tubercles, with dilations of the surrounding vessels, in persons dying of hæmoptysis. De Haen saw an aneurism open into a cavity. Portal records the observation of ruptured vessels from the lymphatic glands opening into the bronchi; and J. Frank found that in injection of the pulmonary artery in a young man, who had died of hæmoptysis, the injected material passed into a cavity. Generally the earliest writers looked for the cause of pulmonary hæmorrhages in rupture of large vessels; in the present century, especially on the authority of Laennec, modern pathologists, on the other hand, assumed the existence of an exudation of blood through the bronchial membrane—an hypothesis now rendered not improbable by the experiments of Cohnheim. But the amount of blood in such instances could not be otherwise than extremely small, even although the more modern name of "*capillary hæmorrhage*" takes the place of "*exudation of blood.*"

Some observations on the morbid anatomy of the lung led to the rejection of the belief that large vessels were the source of the hæmoptyses, and especially the observation that the vessels in the condensed tissue surrounding the cavity are obliterated.

Baillie called attention to this condition, and Laennec demonstrated the fact by injections,—discovering, at the same time, that the *trabeculæ*, which so frequently cross cavities in the lungs, enclose obliterated blood-vessels, which, in rare cases, and usually only partially, remain open. The injections, also, of Schroeder Van der Kolk showed that the closing of the vessels takes place from the smaller, and gradually extends to the larger branches.

Speculations regarding the source of *hæmoptyses* embrace the following beliefs:—

(*a.*) Rupture of pulmonary arteries, or branches of pulmonary veins; or bursting of an aortic aneurism into the pulmonary passages; or exudation of blood through mucous membrane of the bronchi; or violent hæmorrhages from pulmonary apoplexy (LAENNEC).

(*b.*) Hæmorrhage from bronchial membrane without co-existent tubercle; from pulmonary apoplexy; from the eroded blood-vessel in a trabecula of a cavity (ANDRAL).

(*c.*) Hæmorrhage due to the altered collateral circulation surrounding tubercle growths, so that vessels remaining distended readily burst, while others are compressed by condensed portions (ENGELSTEDT).

(*d.*) Hæmorrhage due to molecular rupture of the capillaries of the parenchyma (WALSHE).

(*e.*) Hæmorrhages from branches of the pulmonary artery situated in the condensed walls of cavities, especially in dilated bronchial cavities, either through a slit or an aneurismal dilatation (ROKITANSKY).

(*f.*) Hæmorrhages due to ruptures of small capillary vessels in the walls of recent cavities from little fungus excrescences on them (HERARD and CORNIL).

(*g.*) Hæmorrhages from rupture of capillaries, either from over-distension or morbid delicacy of their walls—a result of perverted nutrition (NIEMEYER).

The positive testimony of Dr. Rasmussen as to the anatomical source of *hæmoptyses* is based upon the careful *post-mortem* examination of cases in which the patients have died during violent hæmoptysis, and is as follows. The source is embraced in one or other of the following classes of lesions, namely:—

I. Cases where the hæmorrhage proceeds from rupture of a vessel running in the wall of a cavity—"*hæmorrhages from a cavity.*" These are due partly to ruptures of small sac-like aneurisms developed on branches of the pulmonary artery running in the walls of cavities; partly to small dilatations (*ectasias*) of similar vessels with operculated rupture. An account of these aneurisms has been already given (see p. 649, *ante*).

Ectasias are small aneurismal dilatations of vessels running in the walls of cavities. They occur under two forms:—

(*a.*) As cords of different lengths on the inside of the cavity, which, on being slit up, exhibit a slight dilatation of their calibre—towards the cavity—with corresponding thickness of the walls. Such *ectasias* are rare in relation to hæmorrhages.

(*b.*) A form in which the vessel comes into limited contact with the wall of the cavity. It is represented by a rather oblong prominence, sometimes as large as a bean or a pea, and partly due to dilatation of the calibre of the vessel, and partly due to thickening of its wall.

The perforation into this vessel takes place by a V-shaped slit, which constitutes a valve-like or lid-shaped flap. The rupture occurs generally in the boundary between the vessel and the wall of the cavity, and the apex of the lid lies always in the direction of the current of the blood. The walls of aneurisms proper are always thin at the seat of perforation; but at the seat of the lid-shaped rupture the wall of the vessel is often very thick. As a rule, however, the V-shaped lid forms a tolerably thin, easily moveable flap of a yellow colour.

Apart from the question of tubercular deposit in the lungs, Rasmussen comes to the conclusion that "*every cavity in the lungs whose walls are formed by condensed pulmonary tissue, containing non-obliterated blood-vessels, may be the seat of aneurisms or aneurismatic dilatations with consecutive ruptures.*" Aneurisms may occur in the largest as well as in the smallest cavities; hence they are often easily overlooked.

Profuse and fatal hæmoptysis almost always proceeds from cavities; and the occurrence of hæmorrhage is quite independent of the size of the cavity.

A very important question is raised by Dr. Rasmussen, namely,—What is the probable source of pulmonary hæmoptyses which do not cause death, but after which patients may live for many years? He believes such hæmoptyses may be due to the rupture of very minute aneurisms, which stops from the fortunate formation of a thrombus in the ruptured vessel, or by pressure of inspissated bronchial secretion.

Parenchymatous pulmonary hæmorrhage may also no doubt be due to the breaking up of the texture of the lung after consolidation, the capillary pulmonary vessels being perforated by lesions affecting their coats; but positive data are wanting in demonstration. Certain it is that there is a great predisposition to hæmoptyses in persons suffering from various forms of pulmonary consumption. But, on the other hand, hæmoptyses are not to be regarded as anything like a sure indication of incipient or even of established wasting lesions of the lungs.

II. Another source of *hæmoptyses* has been traced to systemic aneurisms, such as of the aorta, bursting into a bronchus or into pulmonary tissue. Such hæmoptysis has already been noticed as an early symptom of thoracic aneurism, either as evidence of actual rupture or as producing by its pressure such disturbances of the circulation in the lungs as leads to hæmoptysis. Thus, Mr. Liston, the celebrated English surgeon, during the space of eight months before his death, frequently brought up small quantities of blood, under the influence of such disturbance by pressure of the circulation in the lungs.

Hæmoptyses may also result, not only immediately after penetrating gun-shot wounds of the chest, in proportion to the greater or less degree of direct violence to the lung-tissue, but they may occur long after the external wound caused by a gun-shot penetrating the lungs has healed up. Such may be called secondary hæmorrhages of the lung after gun-shot injury; but they are not mentioned by military surgeons. Of such cases Rasmussen records several; and the probability is that some part of the course of the wound in the lung remains unhealed (perhaps from the presence of a foreign body, such as a piece of dress or bit of bone), being thus converted into a fistula or small cavity, and so becomes liable to the lesions already described, as the usual source of *hæmoptyses.*

Symptoms.—Hæmoptysis may take place suddenly, or be preceded for two or three days by a sense of heat or a feeling of weight at the chest; or the patient may suffer pain between the back and shoulders, or may labour under dyspnœa, palpitation, cough, or coldness of the extremities. At length a fit of coughing, or a tickling of the throat, is followed by the appearance of expectoration of blood. Laennec says he has seen as much as ten pints of blood thrown up in forty-eight hours, and as much as thirty pints in a fortnight. The effort of coughing also often causes vomiting, so that the blood discharged is frequently mixed with alimentary matters.

If the quantity thrown up be inconsiderable, the patient's health is in no degree affected; but if it be large, its effects are strongly marked; for the patient feels oppressed at the præcordia, breathes with difficulty, and with a gurgling sound, caused by the air passing through the viscid blood retained in the bronchi. This is shortly followed by increasing weakness, even to complete prostration. In still more severe cases, as the blood flows the patient turns pale, his countenance becomes anxious and strongly expressive of terror; or he falls into a complete syncope, which sometimes has a curative effect. In a very few instances the effusion is so sudden and so considerable that the patient dies suffocated.

Although bronchial hæmorrhage may be considerable, it often diminishes so rapidly that at the end of some hours only a few rare isolated sputa are spat up, and at considerable intervals. Usually, however, the hæmoptysis recurs after a greater or less length of time, but not perhaps to the extent of the primary attack. It is then remittent, each attack being ushered in by a violent fit of coughing. After the patient has lain for a time in a state of depression, a re-action takes place. In sthenic persons the appetite becomes increased, they enjoy everything they are allowed to eat, and after some slight febrile action they rapidly recover. In other cases anæmia is apt to set in from the repeated losses of blood, so much so that a small recurring hæmoptysis may prove fatal, even though the blood lost be inconsiderable.

Clinically we are not yet able to distinguish an hæmoptysis due to rupture of a vessel in the wall of a cavity from an hæmoptysis occurring during the course of pulmonary phthisis, which may possibly have another cause than those specially demonstrated by Rasmussen. In the fatal cases the pulse becomes rapid, the tongue brown and dry, and the patient sinks.

Hæmoptyses due to *aneurism*, or to *ectasia* of a branch of the pulmonary artery, usually occurs suddenly without either the patient or the physician having the slightest suspicion of its impending supervention. During a fit of coughing or violent bodily effort, blood rushes from the nose and mouth, and the patient may die asphyxiated in the course of a few minutes.

Diagnosis.—The only disease which it is important to distinguish from *hæmoptysis* is *hæmatemesis*, and the diagnosis between them is difficult, because while the contents of the stomach are always rejected in *hæmatemesis*, they are frequently rejected in hæmoptysis also. The stethoscope, however, greatly assists in determining the seat of the disease; and again, blood is generally found in the stools in cases of *hæmatemesis*, while it is for the most part wanting in *hæmoptysis.*

Prognosis.—The prognosis is always unfavourable eventually, so far as freedom from organic disease is concerned, and danger is more or less directly imminent in proportion to the amount of blood lost, and the frequency of its recurrence.

Treatment.—The medicines most useful in *hæmoptysis* are the *bitartrate of potash, in doses of a drachm*, repeated every four or six hours, and to each dose of which may be added a quarter to half a grain of *opium.* The *mineral acids,* as the *infusion of roses* with diluted *sulphuric acid,* in doses of from three to five drops, combined with *opium* or *morphia,* every four or six hours; larger doses of the *dilute sulphuric acid* have often been tried, but are apt to be either rejected or to act injuriously on the coats of the stomach. Many practitioners use one to three grain doses of the *acetate of lead* every four or six hours, with *half a grain of opium* to each dose, or combined with *dilute acetic acid* and *laudanum* (A. T. Thompson); and, according to Andral, when the system has long been under the influence of lead, the red globules suffer a great diminution; but, nevertheless, this is certainly a less efficacious medicine than either of the preceding ones. The *nitrate of potash* has been much used in France, but Gendrin has not found it efficient, or not more so than any other diuretic. The *muriate of soda*, in doses of half a drachm to a drachm, is in estimation with some practitioners on the Continent.

In *atonic hæmoptysis, ergot* is said to be of service, especially in the following combination:—

℞. *Ext. Ergotæ Liq.*, ʒii.; *Tinct. Digitalis*, ʒii.; *Acidi. Gallici.*, ʒi.; *Magnes Sulph.*, ʒvi.; *Acid. Sulph. Dil.*, ʒi.; *Infus. Rosæ Acid.* ad ℥viii.; *misce.* A sixth part of this mixture to be taken every three hours till hæmorrhage ceases (Dobell, Warring-Curran). Wunderlich also recommends the *secale cornutum* in doses of from five to ten grains, to be pushed until a numb sensation is experienced in the fingers and toes.

When hæmoptysis is connected with amenorrhœa, preparations of iron often succeed when the above remedies have failed. Two grains of the *sulphate of iron*, with one drachm doses of the *sulphate of magnesia*, three times a day, often restore the menstrual secretion and cure the hæmoptysis. Indeed, it is in this form of amenorrhœa that iron is most successful.

When hæmoptysis depends on disease of the heart, cupping from the chest, or moderate bleeding from the arm, is often efficacious, combined with the use of the *bitartrate of potash* or the mineral acids, to which should be added five to ten minims of the *tincture of digitalis.* It is in many cases proper to add half a drachm to a drachm of the *spirit of nitrous ether* to each dose, to give steadiness to the irregular, turbulent, or rolling action of the heart.

Dr. Fuller's experience leads him to testify most strongly in favour of repeated dry cupping, aided by the application of ice down the spine, and by the internal administration of *full* doses of *digitalis* (half a drachm of the tincture; or a grain and a half of the powder). It is chiefly as an adjunct to other means, when hæmoptysis is attended with much vascular excitement, that digitalis is of service.

If these remedies fail, full and frequent doses of *gallic acid*, or *lead and opium*, may be given, if the circulation is much accelerated, and of *spirits of turpentine*, in half drachm doses, if the bleeding is unattended with vascular excitement. The *gallic acid* is more adapted for chronic cases, and should be given every hour in *eight* or *ten grain* doses, until the hæmorrhage is subdued, or till a dark-green colour in the sputa indicates its action on the system (*op. cit.*, p. 265). Its efficacy appears to be increased by combination with *sulphuric acid*, and may be conjoined with *opium*, *digitalis*, and such-like remedies.

The following formula, by Dr. L. Earle, is a useful one:—

℞. *Acid. Gallic.*, gr. xxx.; *Acid. Sulph. Dil.*, ʒi.; *Liquor, Opii. Sedat.*, ♏ xxx.; *Infus. Rosar. Co.*, *f*℥vi.; *misce.* Two table-spoonfuls for a dose every three or four hours.

Absolute bodily and mental rest must be insisted on during convalescence.

Dietetic and General Treatment.—The patient should be placed in bed, with his head and shoulders raised; the window should be partly open so as to keep the room cool. It has been recommended that the air respired should pass through a respirator containing ice; and it is common to place a bowl of ice immediately before the patient, so that he may suck small portions as often as he can. Some practitioners have recommended ice to the chest; but this often causes great anxiety and constriction of the chest, and is of doubtful efficacy.

The bed-clothes should be light. The diet should be slops, and these slops cold; and if cooled to a low temperature by ice, so much the better.

PULMONARY EXTRAVASATION—*Syn.*, PULMONARY APOPLEXY.

LATIN EQ., *Hæmorrhagia pulmonalis*—Idem valet, *Appolexia pulmonalis;* FRENCH EQ., *Apoplexia pulmonaire*—Syn., *Pneumo-hæmorrhagie;* GERMAN EQ., *Blutaustritt in die Lunge*—Syn., *Apoplexie der Lunge;* ITALIAN EQ., *Stravaso polmonale*—Sin., *Apoplessia polmonale.*

Definition.—*An extravasation of blood by capillary hæmorrhage into the air-cells, terminal bronchi, and interstices of elastic tissue, by which the air-cells of the lung are entwined.*

Pathology.—In pulmonary apoplexy, when the effusion is trifling, and the patient survives for some time, an induration at one or more points of the lung, and exactly circumscribed, is found, caused by an incorporation of the infiltrated blood with its tissue. The extravasation is confined to a minute and sharply defined section of the lung, often bounded by the limits of a single lobule. The blood is extravasated partly within the cavity of the vesicles and terminal bronchi, and partly in the interstices of the lung; and it generally proceeds from the capillaries pertaining to a single twig of the pulmonary artery. These indurations may be black, brown, or red; and if scraped with the scalpel half-coagulated blood escapes, while the surrounding tissues are healthy, or only more or less congested.

The lesion has more recently received the name of "*hæmorrhagic infarction.*" Such infarctions occur either in the interior of the lung when they are large, or towards the periphery when they are generally small in size and of the shape of the superficial lobule. The blood is generally coagulated, the liquid part being absorbed. The masses of extravasated blood vary in size from that of a hazel nut to that of a hen's egg, of a blackish-red or dark chocolate colour, inelastic, and void of air. On handling the lungs, the extravasations may be felt as hard knobs or nodular masses in their substance. On section, they show a surface which is irregular in outline, coarse, and granulated, but sharply defined. When the infarction has existed for some time, it looks more pale and yellow than when recent, the colouring matter of the blood having become decomposed and absorbed, and at last the only trace of its existence may be a localized black induration of the lung.

If the patient perfectly recovers, no trace of disease is to be found after death, the effused blood being absorbed, and the seat of apoplectic effusion, according to Laennec, is marked only by a linear cicatrix, denoting an antecedent rupture of the cells of the lung. In graver cases, and when life is quickly extinguished, the blood effused into the lung is in considerable quantity, half coagulated, and the pulmonary tissue so broken down that it is impossible to demonstrate its structure or to assign the limits of the extravasation. In the worst cases, the lung ruptures, and the effused blood escapes into the cavity of the chest. The bronchi, in most cases, also, are more or less loaded with blood.

Latour appears to have been the first to describe this disease (*Hist. Philosop. et Med. des Hæmorrhagies,* t. i. et ii., p. 220, Orleans, 1815). He gave it the name of *apoplexie pulmonaire,* and the term was adopted by Laennec.

Some modern views regarding pulmonary extravasation are embraced in the doctrines of *thrombosis* and *embolism,* which have been already stated. When the branch of the pulmonary artery is examined, within whose range a pulmonary infarction has formed, a clot is usually found by which its calibre has been more or less obstructed, but it is always difficult to demonstrate this in small vessels. Such clots, from which the infarction arises, are now believed to be *embolic*—*i. e.,* they come from some region of the body; being detached in whole, or bit by bit, from the place where they formed, and so swept into the current of the blood, they become impacted into some branch of the pulmonary artery, too narrow to admit of further passage (VIRCHOW, NIEMEYER).

Thus, as already shown, arise the so-called metastatic infarctions of the lungs from disintegrating *thrombi* found in peripheral veins (see also vol. i., p. 73).

Remote Cause.—The worst cases are generally seen to be connected with extensive disease of the heart, especially cases of disease of the mitral valve, in which clots (embolic) are found in the arteries leading to the infarctions (ROKITANSKY, GERHARDT, NIEMEYER). But, according to these observers, the *emboli* which block the artery in disease of the heart do not come from the greater circulation, like the emboli of metastatic infarction, but they come from the right side of the heart, and especially from the right auricle, in which clots usually exist, firmly entangled in the *trabeculæ* formed there as a result of the slowness of circulation. It is the particles of such clots which obstruct the pulmonary artery. The *debris* from these cardiac clots is also generally much greater than from the aortic or systemic circulation, and the resulting infarction from *embolism* is also much more extensive than those from *metastatic* sources. The infarctions of heart disease are usually also found at or towards the roots of the lungs, while the *infarctions* of metastasis are generally found near the periphery, and generally involve the pleura in a local pleuritis. They are often also mixed together in heart disease. Why the obstruction of a branch of the pulmonary artery by *embolism* should produce capillary hæmorrhage within a certain area of the obstructed vessel has received several theories towards elucidation by Rokitansky, Virchow, Ludwig, and Niemeyer; but their explanations are not quite clear (see Niemeyer, p. 156, vol. i.)

Symptoms.—The symptoms of pulmonary apoplexy have various degrees. The effusion may be slight and the patient recover; or it may be extensive, and the patient survive some days; or it may be so sudden and considerable as to cause the immediate death of the patient.

The *first degree* of pulmonary apoplexy may be determined during life. If in a case of chronic heart disease, and especially of the valves, there occurs a sudden difficulty of breathing which may threaten suffocation, some expectoration of blood, some mucous rhonchus, and cough, and a total inability for a time to lie down, the formation of one or more hæmorrhagic infarctions may be inferred. On percussion of the chest, also, that portion which corresponds to the seat of the disease returns a dull sound—a sign of circumscribed condensation of the lung, not unfrequently followed by signs of pneumonia or of pleurisy. Gendrin is of opinion that blood cannot be effused without causing inflammation; and if the patient recovers, pneumonia of little intensity always follows.

In pulmonary apoplexy of the *second degree*, the symptoms which have been described exist, but in greater severity, so that the patient is more oppressed in his breathing; he is obliged to be supported by pillows, and his head often falls forward, while his face is purple, and his pulse small and frequent; yet, however formidable these symptoms are, life is still capable of co-existing with them for some time.

In the *third degree* of pulmonary apoplexy the patient appears to be almost instantaneously destroyed.

There are signs of cardiac thrombosis of the right side of the heart, which are tharacteristic of hæmorrhagic infarction. These are stated by Niemeyer to be "sudden irregularity of the pulse, a sudden widening of cardiac dulness, sudden cessation of cardiac murmurs previously heard;" and he confirms them, as thus stated by Gerhardt, from his own experience.

Diagnosis.—Apoplexy of the lung may be distinguished from hæmoptysis by the dulness on percussion, by the tubular breathing, and by the subsequent fever and pneumonia.

Prognosis.—Pulmonary apoplexy is always of grave prognosis; but should the patient survive the attack for a few days, and the effusion be inconsiderable, and the subsequent inflammation slight, he may recover.

Treatment.—The treatment must of course depend on the disease, symptomatic of the conditions already referred to. When the diagnosis can be relied on, bleeding must not be thought of, as the oppression and difficulty of breathing might be apt to suggest. Stimulation must be resorted to in the first instance; after which the *mineral acids* or *super-acid salts* appear to offer the most chances of recovery. If the appoplexy be secondary, and depends on disease of the heart, *digitalis*, and perhaps some slight narcotic, may tranquillize the excitement of the organ.

The *dietetic* and *general treatment* are the same as have been directed for hæmoptysis.

EMPHYSEMA.

Latin Eq., *Emphysema;* French Eq., *Emphyseme;* German Eq., *Emphysem;* Italian Eq., *Enfisema.*

Definition.—(1.) Vesicular Emphysema—*Relative increase of air in the several air-cells of the lungs, causing a misshapen enlargement of them, by dilatation and blending of several into one or many great cysts, attended with gradual effacement of the functional blood-vessels distributed over their walls; anæmia of the lung in the affected parts, tending to dilatation of the right side of the heart, with anasarca.* (2.) Interlobular Emphysema—*Air infiltrating the meshes of the sub-pleural and interstitial connective tissue of the lungs.*

Pathology.—Some confusion has arisen from the use of the term "*Emphysema*" (which means the presence of air in the connective tissue) to designate dilatation of the air-cells of the lungs. Here the air is where it ought to be; but the air-cells are too large and misshapen, and contain too much air; and being limited in this way to the vesicles of the lung, this form of disease was named by Laennec "*vesicular emphysema.*" In 1698 the disease was identified and well described by Sir John Floyer as existing in broken-winded horses; and Dr. Baillie, in subsequently describing enlargement of the air-cells of the lungs, refers to Sir John Floyer's description as applicable to the lungs of the human subject.

The dilated air-vesicles vary from the size of millet seeds to that of Barcelona nuts, or even larger; but when they form a great expansion, it is probable that many air-vesicles are dilated into one common cavity by rupture of the partitions which separate them from each other. The dilated vesicles may be seen clearly through the pulmonary pleura: they also protrude from the surface of the lung. The emphysematous parts are pale, and sometimes quite white; the tissue is drier than normal; it cannot be easily emptied of air, resembling the lungs of a reptile rather than those of a human being. It possesses fewer capillary vessels; and they become obliterated by distension of the air-cells. The lung is therefore anæmic, and contains less moisture than the normal lung. It is so dry and light that it floats much higher in the water than a healthy lung.

Causes.—Several theories have been put forward to explain the mechanism which produces emphysema. Dr. Elliotson considers a want of due expansion of the lungs as the most common cause of emphysema. "Whatever prevents any one part of the lungs from expanding when the thorax expands—whether it be a material obstruction of the bronchial ramifications or a compression of them, or whatever else, it will occasion those parts which remain dilatable to keep dilated in a correspondingly increased degree, in order to fill up the vacuum which the expansion of the chest occasions. When we inspire, we dilate the chest, and the air rushes down the trachea, and the lungs follow the dilated portions. If there be any part [of the lung] that will not dilate, then other parts are over-dilated to fill up the vacuum; and in that way those parts which we distend are *over*-distended, in order to compensate for the want of distension in other parts; and when once *over*-distended they are often unable to recover themselves, just as is the case in other parts of the body—the urinary bladder, for example. I presume it is on this account that dilatation of the air-cells is so common in persons labouring under chronic bronchitis, especially where the membrane is most thickened, and where the secretion, if there be any, is tough and adherent, so as to produce obstruction" (*Practice of Medicine*, p. 851). Sir Thomas Watson follows Laennec in believing that the dilatation in the outset is mainly due to the imprisonment of air within the cells under the influence of disease, such as imperfectly obstructed tubes, so that air enters the vesicles more readily than it can escape from them. More and more air then accumulates, and is incarcerated in certain air-cells, which, yielding to the distending force, lose their elasticity and become permanently large.

But, to confirm this view, it ought to be shown that the dilated air-cells are those belonging to the tubes in which the obstruction exists; and Dr. Elliotson is of opinion that they are not those in which there is obstruction. Dr. Gairdner, indeed, has very ably demonstrated this in connection with the occurrence of emphysema in bronchitis (see page 728, *ante*). The disease forms one of the most serious complications of *bronchitis;* and the tendency to vesicular emphysema appears to be hereditary—60·4 *per cent.* acknowledging hereditary transmission (FULLER).

Symptoms.—In typical cases of emphysema the patient is short-winded and distressed by a constant sense of fulness and oppression at the chest, and generally seeks advice after suffering and discomfort have become too great for him to bear any longer.

The difficulty of breathing is often aggravated by spasm, as in *asthma;* and emphysema is a frequent consequence of that disease—the one re-acting on the other, so that the phenomena of each are mutually aggravated.

The physical signs are, incompleteness of the act of expiration, the thorax remaining prominent and round over the emphysematous lung. In spare persons the clavicles are not well defined. Percussion, over the bulging parts especially, yields a peculiarly clear and resonant sound; and although there is thus shown to be abundance of air underneath the part which yields such a sound, yet the *vesicular murmur* of breathing is extremely indistinct, showing the air is not in motion there. It is shut up in the enlarged air-cells (WATSON).

The disease tends to impede the circulation through the lungs, and so to produce hypertrophy, with dilatation of the right side of the heart, nervous congestion of the head and face, attacks of palpitation, paroxysms of cough and dyspnœa, œdema of the feet and legs, general anasarca; and dropsical effusion is a frequent termination of the disease.

Treatment.—Apart from the management of the bronchial congestion, on the principles already given under *bronchitis,* little can be done for the special treatment of *emphysema.* If bronchial spasm prevail, Hoffman's anodyne may give relief. It is the *spiritus ætheris* of the British Pharmacopœia, of which *thirty* to *sixty minims* may be prescribed in *camphor* water, or in *spiritus ammoniæ aromaticus,* or in *volatile tincture of valerian;* or it may be combined with stimulant doses (*one grain*) of opium; or with *twenty minims* of the *æthereal tincture of lobelia, belladonna, conium;* or the *æthereal tincture of Indian hemp and hydrocyanic acid* may each in turn be found of service (FULLER).

Dry cupping between the shoulders often relieves passive pulmonary congestion; and if an attack is imminent, an emetic, or unloading the bowels by a dose of the *compound jalap powder,* may prevent its accession or moderate the paroxysm.

SYPHILITIC DEPOSIT IN THE LUNGS.

LATIN EQ., *Deposita ex syphilide;* FRENCH EQ., *Dépôt syphilitique;* GERMAN EQ., *Syphilitische ablagerung;* ITALIAN EQ., *Deposito sifilitico.*

Definition.—*The development of gummatous nodules in the substance of the lungs after syphilis, with or without bronchitis or miliary tubercle.*

Pathology.—Morton, Sauvages, Portal, Morgagni, and more recently Graves, Stokes, Ricord, M'Carthy, Walshe, Wilkes, Virchow, and Munk, have all described, with greater or less uniformity and distinctness, pulmonic lesions in cases of syphilis.

The following are the kinds of lesions which predominate:—

1. *Evidences of Inflammation of the Mucous Membrane of the Bronchial Tubes.*—In such cases bronchial irritation, with fever in many cases, precedes the skin lesions of syphilis, and may disappear wholly or partially when the skin lesions are established. On the other hand, if the syphilitic

eruption suddenly disappears, bronchitis may ensue. Walshe records well-marked instances of this; and it is a circumstance to be looked for amongst soldiers especially, who, having recovered from a primary syphilitic sore, are apt to be exposed to the risk of bronchitis when mounting guard soon after being discharged to duty. Secondary symptoms and pulmonic lesions are then apt to date their commencement; and all the general symptoms of phthisis may supervene, and yet no tubercle in the lung may be developed; but chronic bronchitis remains persistent. On this point Dr. Walshe observes that in the persistence of the general symptoms there "is assuredly enough to create a strong suspicion of the existence of tubercle in the lungs, taken in conjunction with the indubitable tendency of syphilis, *plus* mercury, to induce the outbreak of pulmonary phthisis. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms: the patient with syphilitic bronchitis has neither consolidation signs nor, *à fortiori*, the evidences of excavation. But there is a curious source of difficulty which sometimes starts up in these cases and renders doubt imperative—the infra-clavicular ribs and clavicle thicken from periostitis, and produce dulness under percussion, which cannot with positiveness be distinguished from that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis" (*Diseases of the Lungs*, p. 233).

2. *The Occurrence of Gummatous Nodules in the Pulmonary Substance.*—These are, in the first instance, of the same histological constitution as the well-known node of the shin, or the subcutaneous product described by Ricord, Bärensprung, Virchow, and M'Carthy. They form especially towards the periphery and bases of the lungs. In the former site they resemble nodules of lobular pneumonia. They may soften and be eliminated much in the manner of tubercle, although they may have at first a consistence like scirrhus. It is concerning those which soften in this way that Ricord gives the warning "not to confound suppuration of a few syphilitic nodules of the lungs with phthisis." Regarding those gummatous nodules Dr. Walshe observes:—"I can find no positive answer to the query, Do these gummata ever form independently of other tertiary evidences of syphilis in the bones and cellular tissue? If they do, their diagnosis must be infinitely difficult—difficult, indeed, under all circumstances; for the physical signs can be none other than those of solidification, followed by softening and excavation, while the local and general symptoms closely simulate those of phthisis" (*Diseases of the Lungs*, p. 431).

3. *The Occurrence of Gummatous Nodules in various Stages of Growth and Degeneration, associated with the Miliary Deposit of Tubercle.*—In such cases the history of events in the illnesses of the patient may be found to correspond more or less closely with the appearances seen in the lungs—appearances which distinctly indicate the formation of lesions commenced at different dates—appearances which denote the occurrence of lesions in crops, or as a succession of events which may be illustrated by the history of the symptoms during life.

The minute structure of these *gummatous nodules* has been closely examined by many observers. They consist of a growth of elements which leads to the development of an elastic tumor composed of a well-defined tissue, and the elements of which are extremely minute. The tumor takes origin from the connective tissue, or the analogues of such; and hence the universality of the site of syphilitic lesions. When these are sufficiently large to attract attention—as in the form of a node on the shin-bone, or on some part of the true skin—they are small, solid, pale knots, like a hard kernel, about the size of a pea. They are generally first seen on some part of the true skin or subcutaneous or submucous tissue; and when the tissue in which they happen to grow is sufficiently lax, they grow to a considerable size, and convey to the touch a sensation as if they were filled with gum. Repeated examinations of this growth show that in its gelatinous or soft state it arises from a proliferation of nuclei amongst the elements of the connective tissue, not unlike the formation of granulations in a wound. The component cell-elements appear as round, oval, or oat-shaped particles imbedded in a matrix of fine connective tissue of a granular character, and tending to fibrillation. The cell-elements are a little larger than blood-globules, and are distinctly granular in their interior when mature. In the growing part of the node, and immediately in its vicinity, where growth is abnormally active, the minute cell-elements are seen to be developed in groups within the elongated and enlarged corpuscles of the connective tissue. In form, therefore, the node or gummatous nodule resembles a tubercule; and by fatty degeneration or tuberculization may not be capable eventually of being distinguished from tubercular deposit. How then are we to recognize the specific nature of such gummatous nodules? There is nothing in them so specifically and anatomically distinct that, apart from their history, they can be recognized. The history of the syphilitic case during life is the great guide. The nodes on the shin-bone or clavicles have long been recognized as the product of syphilis. It may almost be said that they have been seen to grow under the eyes of the patient and the observer; and their anatomical characters are found to be such as compose the gummatous nodules just described. In a case of inveterate syphilis, therefore, whose history is fully known, in whom the node on the shin or other bones is characteristic, and has been seen to grow, and in whom also we find similar nodules in the lungs, or in the liver, or in the testicles—symmetrically growing in these latter organs, and consisting of minute cell-elements exactly the same as the node on the shin—it is impossible to overlook the fact, or not to be impressed with the belief, that all of these lesions acknowledge one and the same cause of development—namely, the syphilitic poison—of which they are the expression. The progress of the node is also characteristic and suggestive. Growths of a similar form which result from idiopathic inflammation generally proceed to the formation of an abscess, or to the hypertrophy of fibrous tissue. Abscesses are recognized by their pus; fibrous tumors or hypertrophies, by the fibre-elements which compose them.

Growths of a form similar to the node, which result from cancer, are in general to be recognized by the juice expressed from them. In the gummatous nodule we have no juice, and the cell-elements seen in cancer are generally so diversified in their form and mode of growth as not to be easily mistaken. The gummatous nodule is uniform as to the size and form of its cell-elements, and forms a growth less highly supplied with blood-vessels than a cancer. Cancers tend to infiltrate and involve neighbouring textures; the gummatous nodule remains isolated and distinct.

By way of elimination, therefore, and by duly observing the history of the case, we are generally able to recognize the nature of such growths, and to assign to them their proper place in pathology.

The gummatous nodule has now been recognized and described in almost all the solid viscera of the body. Symmetrical development is a most constant characteristic. If a node grows on one shin, it is probably also to be found advancing on the other; if found in one testicle, it is extremely probable that it will be seen in the same relative spot in the other. Numerous examples of this symmetrical development may be seen preserved in the Pathological Museum of the Army Medical Department at Netley. During the growth of the nodule, proliferation advances slowly, and a gluey-like material forms, which constitutes the inner cell-material of the nodule. If near the surface, such a nodule is apt to melt down, soften, open, and ulcerate; and such a result seems to be associated with other evidence of active constitutional disease, such as exists with a predisposition to tubercle, or with its actual existence. The tumor, however, continues gelatinous and coherent if it is enclosed in a dense part, or is deeply seated, as in gummata of the periosteum, scalp, brain, liver, testicle, lungs, and heart, if constitutional disease remains latent or inactive. Fatty degeneration may also eventually occur in the gummatous nodule, and eventually lead to its absorption; or its absorption takes place as a natural process of cure, the changes of which are not exactly known. We know only that the node on the shin-bone not seldom disappears from view, and does not return.

For reasons already stated, pulmonary phthisis must be regarded in many cases as the product of syphilis; and I would fully endorse the statement of Dr. Balfour, from what I have seen in the *post-mortem* rooms, when he says that a great cause of pulmonary disease among the Guards is the amount of syphilis which prevails amongst the men, which he has not the least doubt is a very fertile cause of its being called into active operation. The influence of syphilis on the health of the soldier is indeed powerful for evil throughout the whole army.

Treatment.—The treatment of such pulmonary lesions, when their nature is clearly established, must be guided by the rules already laid down for the treatment of syphilitic disease at p. 895, vol. i.

PULMONARY PHTHISIS.

LATIN EQ., *Phthisis;* FRENCH EQ., *Tuberculeux;* GERMAN EQ., *Phthisis*—SYN., *Schwindsucht;* ITALIAN EQ., *Tise cronica polmonale.*

Definition.—*Lesions commencing with lobular induration of the lungs, which end in disintegration of the new material and of the textures involved* *in the induration with the formation of caverns, and with or without the deposit or growth of miliary tubercles.*

Pathology and Morbid Anatomy.—The rapid advance of more accurate knowledge along the very tortuous course pursued by Medical Science has not been more marked in any direction than in demonstrating the nature of the lesions which lead to destruction of the lung, and the relation of the formation of miliary tubercles in them, to the processes of disintegration of pneumonic products.

While the observations and writings of eminent Continental pathologists and physicians on the Continent have done much of late to rectify erroneous views regarding the subject of pulmonary phthisis, it is now beginning to be acknowledged that the late Dr. Addison, of Guy's Hospital, did more than any one else in England to advance the doctrine of pneumonic phthisis. His labours had remained entirely unknown on the Continent, and having been mainly brought to light again by the New Sydenham Society, they show that already, when Laennec's teaching had just commenced to dominate over the pathology of lung diseases, an independent observer arrived at and firmly held the opinion which during the past five years has been established by the observations of Reichardt, Dr. Andrew Clark, Virchow, and Niemeyer, that "*inflammation constitutes the great instrument of destruction in every form of phthisis;*" while, on the other hand, the formation of miliary tubercles in the lungs, and their relation to cheese-like products and to phthisis, have been placed in a new light by the practical experiments and clear expositions of Drs. Villemin, Burdon-Sanderson, Wilson Fox, A. Clark, Colin, Chauveau, Cohnheim, Buhl, Waldenburg, and Gasquet.

The term "phthisis," as implying a condition of lung ready to disintegrate and to have cavities form in its substance, must no longer be regarded as having always a base of tubercle formation as its starting point; and the doctrine that pulmonary phthisis is a constitutional disease—in the sense of Laennec—the result of a specific morbid product, arising from some peculiar diathesis or constitutional fault, and caused solely by the deposition of this substance, can no longer hold its own.

Next to Dr. Addison, no one has done more to spread sounder views as to the nature of pulmonary phthisis in this country than Dr. Andrew Clark, of the London Hospital; and the following provisional classification of pulmonary lesions by him will show the wide view he originally took of its pathology:—

PROVISIONAL ARRANGEMENT OF THE VARIETIES OF PULMONARY PHTHISIS (DR. ANDREW CLARK).

Name.	Chief Anatomical Characters.
1. Tubercular, granular, or specific phthisis.	The true grey granulation. Pigmentary tubercle. Fibrous tubercle. Cellular tubercle?
2. Scrofulous or epithelial phthisis.	Primitive yellow tubercle; accumulation, cheesy degeneration, and disintegration of epithelium-like cells.
3. Catarrhal or bronchial phthisis.	Ulceration of bronchi, with adjacent fibroid and cellular deposits, and cheesy degeneration of the same.

PROVISIONAL ARRANGEMENT OF THE VARIETIES OF PULMONARY PHTHISIS (DR. ANDREW CLARK)—*Continued.*

Name.	Chief Anatomical Characters.
4. Pneumonic phthisis.	Disintegration of recent or old deposits occurring in vesicular, lobular, or lobar pneumonia, primary or secondary, common or scrofulous.
5. Fibrous phthisis (cirrhosis, chronic, or interstitial pneumonia).	Fibroid deposits, with cheesy degeneration of imprisoned portions of lung, due to—(*a.*) Mechanical irritation (as in grinders, masons, miners, &c.); (*b.*) Rheumatic inflammation of interlobular tissue; (*c.*) Chronic pleurisy; (*d.*) Constitutional states, as in granular kidney and liver.
6. Lardaceous phthisis.	Circumscribed or diffuse cellular formations composed of lardaceous material.
7. Syphilitic phthisis.	Cheesy disintegration of nodules of nucleo-fibrous tissue, and diffuse infiltrations of the same.
8. Hæmorrhagic phthisis.	Cheesy degeneration and disintegration of nodules of extravasated blood.
9. Embolic phthisis, including pyæmic deposits and suppurations.	Cheesy degeneration and disintegration of grey or yellow deposits, arising, directly or indirectly, from pulmonary emboli coming from the liver, lymphatics, or veins.

The nomenclature of the College of Physicians regards the subject of phthisis from two points of view, namely—(1.) As allied to scrofula—a general constitutional disease, with or without tubercle; and (2.) As destructive disintegration of the pulmonary tissue, having its source in *acute* or in *chronic* pneumonia.

The present position of our knowledge regarding the pathology of pulmonary phthisis will be stated here mainly from the writings of Addison, Niemeyer, Virchow, Burdon-Sanderson, and Gasquet; and it will already appear, from the definition, that miliary tubercle, as a cause of phthisis, plays a very insignificant part in the process; while many lesions tending to induration of the lungs, in the course of their subsequent disintegration, give rise to phenomena which have been confounded under the common name of "tubercular phthisis."

It has now been shown that in a large number of cases in which death results from pulmonary phthisis tubercles are not present in the lungs (ADDISON, NIEMEYER).

In the aged, it has also been shown by Dr. Maclachlan (*Diseases of Advanced Life*, p. 333), that "consumption may exist independent of tubercular development, and that tubercles are not the essential anatomical character of senile phthisis; and that the most extensive destruction of the lungs not unfrequently occurs, accompanied with the usual symptoms of this disease, without a trace of tubercle." It is usually, he observes, a sequence of chronic bronchitis, terminating in indolent inflammation and partial induration of the lung. These indurated parts at last break down, leaving caverns and burrowing sinuses, which are characterized by a dark and sloughy aspect of their inner surface, and by the absence of any membranous lining (ARMSTRONG, GRAVES, STOKES, MACLACHLAN). "It constitutes the *ulcerous phthisis* of Bayle, the *pneumonic phthisis* of Addison;" and, in the experience of Dr. Maclachlan, it "is far from unfrequent in aged persons, the victims of intemperance or of chronic visceral disease." He believes it to be a form of inflammation, disorganizing the lung. In *senile pulmonary consumption*, when tubercles are found, the tubercular growths are generally confined to the lungs—commonly limited to one lung only; and to the upper and back portions—tubercles existing in the very apex or apices only, the remainder of the lung being healthy (MACLACHLAN, *op. cit.*, p. 334).

Many formations and lesions are constantly mistaken for tubercle, and no appearance is more often so regarded than the cut sections of bronchial tubes, thickened, softened, dilated, and containing a muco-purulent fluid in their opened cavity, as Addison long ago figured and described. Every condensation of lung-tissue, every cheesy metamorphoses of a previous formation, be it pus, solid exudation, or true miliary tubercle, have all and severally been regarded as tubercle; and the fact of their having become yellow and cheese-like (tyromatous) was regarded as evidence of their having been originally tubercle; more especially if the yellow cheese-like masses happened to co-exist with true miliary tubercle. They were then regarded as a diffuse growth of tubercle, or as a tubercular infiltration, like the infiltration of a cancer growth. But it is now well established that many consolidations, such as inspissated pus, old cancer masses, lymphatic glands enlarged by hyperplasia of their cell-elements, hæmorrhagic deposit, as well as the clear miliary tubercle, may each and all become yellow and cheese-like, and quite independent of the *scrofulous diathesis.* To this condition Dr. Craigie, of Edinburgh, proposed the names "*tyrosis*," "*tyroma*," "*tyromatous*" (from τυρος, cheese), to signify the carious aspect of such masses or accumulations, and without reference to the origin or causes of the lesion; but these names have never come into general use. (See his *Pathological Anatomy*, p. 1008.) Such cheesy transformation constitutes the "tuberculization" of Virchow, and is to be looked upon as a form of degeneration of new material to which the name *tyrosis* would be much more applicable, or at least less objectionable. All the morbid products in the lungs which lead to pulmonary phthisis, whether they be of pneumonic origin or true miliary tubercle, all tend to undergo this cheese-like transformation—*tyrosis*; and the cause of this change has been variously explained by the following theories:—

(*a.*) Constitutional predisposition—the evidence of which is based on the fact that in some animals all ordinary pus undergoes this change, as in rabbits and sheep.

(*b.*) Abstraction of the watery part of the pus or of the new material by absorption—a theory also supported by the occurrence of the change in those animals which rarely drink water and take little or no liquid food, their urine being highly concentrated and their excrement hard and dry.

(*c.*) Local anomaly of structure or function, such as may be expressed in greatest intensity in the lungs; as, for example, a relatively small amount of blood in the pulmonary vessels (WALDENBURG); and consesequent "dryness" of the pulmonary tissue. Hence certain diseases which keep the lungs over-supplied with blood have been observed to be antagonistic to phthisis—namely, certain diseases of the heart and great vessels, which hinder the outflow of blood, especially from the pulmonary veins (TRAUBE)—the venous and cyanotic blood-crases of Rokitansky. On the other hand, phthisis has been noticed as concurrent with stenosis of the pulmonary artery (TRAUBE, LEBERT, as well as older English and French observers, as mentioned by Dr. Gasquet, in his excellent article in the *British and Foreign Medico-Chirurgical Review*, p. 400, April, 1870).

The rarity of pulmonary phthisis in mountainous countries, and in persons whose chests have been thoroughly expanded, point in the same direction; as well as the immunity of those who suffer from chronic bronchitis; and the opposite effect of pleuritic compression as tending to induce phthisis.

Pneumonic lesions which lead to pulmonary phthisis:—There is no special or peculiar form of pneumonia which leads to phthisis; but the lesion is invariably a more or less prolonged lobular induration of the lung which undergoes the process of *tyrosis*, disintegration and breaking down into cavities (ADDISON, NIEMEYER, BURDON-SANDERSON).

Every form of pneumonia, as Addison has shown, may leave a residue which, under certain circumstances, terminates in *tyrosis;* but some cases of pneumonia are more liable than others to leave products of inflammation behind; and instead of the material of consolidation in pneumonia being liquified and re-absorbed, it becomes more dense, and finally is transformed into the cheese-like substance (*tyrosis*), which finally breaks up into a cavity. Such a termination is rare in cases of common acute (croupous) pneumonia in a healthy person. It is more frequently met with in catarrhal pneumonia, and in chronic catarrhal pneumonia to find it is almost the rule (NIEMEYER).

On this point Addison has written that when the matter thrown out into the lung is of the more plastic or organizable kind it fails to be entirely absorbed, and part of it remains forming deposits, indiscriminately called "tubercles," or "tubercular infiltration."

The doctrine of a specific *scrofulous pneumonia* ought now to be discarded.

But such forms of induration of the pulmonary tissue are altogether independent of tubercle; and may result from a simple pneumonia, or broncho-pneumonia, incident to all ages, in which there is little cough or expectoration, and therefore is often entirely over-looked. Dr. Addison recognized two forms—namely, *pneumonic phthisis* and *tuberculo-pneumonic phthisis;* they are now recognized as *acute pneumonic phthisis,* and *chronic pneumonic phthisis.*

(a.) *Acute Pneumonic Phthisis.*

How far a recent acute pneumonic lesion must have proceeded, so as to be beyond the limits of complete restoration to the normal condition of pulmonary tissue, is not exactly determined, The condition of the patient as to general health and the soundness of his constitution influence the result. Hale and healthy subjects recover from an extensive pneumonic lesion, which persons of a scrofulous or cachectic constitution would not recover from; but how far lungs in a stage of grey hepatization can be recovered from, so that its tissue is completely restored, is not known. When there is actual induration and obliteration of the lobules, perfect repair and restoration cannot be expected. All that can be hoped for is that it may remain quiescent, without any retrogression towards the cheesy degeneration (*tyrosis*). Cases are constantly seen, *post-mortem,* in which gradual changes are obviously passing from red to grey hepatization, with well-marked granulation on the surface of a section, or to cheesy degeneration (*tyrosis*).

Dr. Addison distinguished two varieties of pneumonic acute lesion tending to pulmonary phthisis—(1.) Inflammation more or less acute but slow and insidious in its course, manifesting some attempts at repair, as indicated by various stages and degrees of induration. But the induration, nevertheless, is not complete. The pulmonary tissue continues friable, and within a few weeks or months softens down and gives rise to cavities, generally by a slow ulcerative process, rarely by actual slough or death of greater or less portions of the indurated but friable tissue. (2.) Inflammation supervening upon, or around ancient induration, leading to disorganization either of the newly inflamed tissue, of the old induration itself, or of both at the same time.

The material accumulated in an *acute catarrhal pneumonia,* when it passes on to the cheesy *degeneration* (*tyrosis*), commences by the catarrh of the smallest bronchi—extending to the alveoli, or lobules, which become filled with young indifferent round cells, so that a whole lung, or parts of it may become disseminated with these minute nodules of lobular catarrhal pneumonia. Each nodule is more or less translucent, and the cell-like bodies which occupy the alveolar cavities are often called epithelial; but their exact relation to the epithelial lining of the alveolar walls is not known (SANDERSON). The individual masses of lobular pneumonia tend to coalesce, and the whole begins to undergo the same changes which fibrine and the cells imbedded in it generally undergo in ordinary acute pneumonia. They become filled with, or transformed into fat globules and then disintegrate, and so becoming fluid are re-absorbed. In the phthisical or destructive cases, however, the mass *caseates* at the centre (SANDERSON); *i. e.*, becomes opaque and soft, and fatty metamorphosis remains incomplete. The cells lose their round form, and by losing water, shrink into irregularly shaped corpuscles; and to the naked eye the consolidation of lung tissue appears of a dull grey, or reddish-grey homogeneous appearance gradually passing into a cheesy-like substance (*tyrosis*). From this point, disintegration may advance till a vomica is formed. Such terminations of lobular pneumonia are common in the course of measles and hooping-cough. It is a result also frequently met with in weakly and delicate individuals possessing but feeble powers of resistance against injurious influences—a delicate vulnerable constitution. There is a tendency in such persons to the cellular products of inflammation. (See vol. i., p. 40.)

Tuberculo-pneumonic phthisis is generally a rapidly fatal and common form of the disease, in which, although tubercles are present, the really efficient cause of the mischief is the pulmonic inflammation and its subsequent results in softening and cavern formation.

(b.) *Chronic Pneumonic Phthisis.*

Of this form, Dr. Addison describes two varieties, namely—(1.) That in which old indurations undergo a slow process of disintegration; and, (2.) That form of disease in which an insidious inflammation proceeds very slowly to convert a considerable portion of pulmonary tissue into grey induration. But, following Niemeyer, the lesion may be considered as the result of what he terms *chronic catarrhal pneumonia*, where the induration is of a gelatinous material, to which the names of "*tuber-culous infiltration*," "*tuberculer*," or "*cheesy pneumonia*," have been given.

Such an *infiltration of the tissues* by an albuminous fluid of a thick synovial-like character, which gradually degenerates into a firm greyish-red granular softened mass, containing portions of tissue within it, was first described by Dr. Baillie in his *Morbid Anatomy*. "In cutting into the lungs," he writes, "a considerable portion of their structure sometimes appears to be changed into a whitish soft matter, somewhat intermediate between a solid and a fluid, like a scrofulous gland just beginning to suppurate. This appearance is, I believe, produced by scrofulous matter being deposited in the cellular [areolar or parenchymatous] substance of a certain portion of the lungs, and advancing towards suppuration. It seems to be the same matter with that of tubercle, but only diffused uniformly over a considerable portion of the lungs, while the tubercle is circumscribed."

The process of chronic pneumonic phthisis not only involves the lobules, but also whole lobes, and the infiltration is not unlike frogs' spawn in colour and appearance. The surface on section is homogeneous and smooth from the material of a bronchial catarrhal secretion peculiarly rich in cells, tending to an ever-increasing accumulation in the alveoli, so that the cells, becoming densely packed together, encroach upon each other, shrink, and undergo necro-biosis—*i. e.*, die (VIRCHOW).

This pneumonic lession occurs in previously healthy lungs, as well as in those which already contain tubercles, induration, old cheesy deposits, or cavities. Occurring in healthy persons, it may be one step towards pulmonary phthisis; occurring in lungs already diseased, it contributes essentially to the further extension of consolidation and destruction.

The cells which accumulate in this variety of pneumonia tend, as a rule, to undergo the caseous change; but cavities are not invariably produced. More often the contents of cell-elements are absorbed; and cretaceous masses are left behind.

The most frequent mode of the formation of cavities in chronic phthisis occurs in those cases where the absorbed material is replaced by a growth of connective tissue. The lung tissue does not again become permeable to air, but is transformed into a tough indurated substance. This connective tissue, by gradually shrinking, occupies less space, the thorax contracts, and the bronchi dilate into oblong round cavities (*bronchiœctasis*).

One common form of pneumonic lesion which thus terminates is the fibroid or sclerosed lung. On section, it presents a smooth or a granulated surface; but generally the indurated part next the sound lung is granulated. The granulations resemble minute hard semi-transparent tubercles, each granule set in the midst of a hyperplasia of connective tissue. The interlobular connective tissue is very distinct, from its hyperplastic abundance, which divides and subdivides by innumerable ramifications, closing in minute polygonal spaces containing air-cells, and interspersed with much pigment. The cut section thus presents a bluish-grey colour, of different shades of iron-grey granite-like appearance, or nearly black. The microscopic texture of the indurated part is made up of cell-elements in various stages of development on to highly organized fibrous tissue. The granules enclosed consist of imperfect cell-elements (query—remains of catarrhal inflammation?) The interloublar connective tissue is greatly increased, and contains dark pigment matter.

In some cases the whole lung, or the greater part, is converted into a tough fibrous tissue, hard and contracted, and which on section looks striated, as if interwoven with fibrous filaments. There is great increase, by hyperplasia, of the connective tissue, and sometimes a granular appearance.

The cavities in the indurated texture are generally at the apex. Their walls are formed of more or less condensed lung-tissue, of irregular form, and containing generally an offensive dark-red fluid. Smaller cavities are generally scattered through the indurated tissue, having a thin membranous lining continuous with that of the bronchi. These cavities are generally, as already stated, formed from the dilated bronchial tubes.

Tyrosis, or cheesy degeneration of portions, may be seen interspersed among the consolidation. Miliary tubercles are not generally found in any part of the lung; but there is generally evidence of long existing disease of the bronchi, which are enlarged, and some may be dilated into cavities. The pleura is generally thickened over the indurated parts, sometimes to the extent of one-fourth or one-half of an inch; and fibrous bands may be seen to pass from the pleura into the texture of the lung. The bronchial glands are generally enlarged and hard.

There is generally granular degeneration or lesions of other organs, such as the kidney, liver, heart, pylorus, capsule of the spleen, bronchial glands, and skin (SUTTON, CLYMER).

"Microscopal examination of the indurated lung-tissue and hard nodules leads to the conclusions that,—(1.) There is a production of new tissue elements, and that these are what are usually considered as representing newly formed connective or fibroid tissue; (2.) That this new formation invades and destroys in part or wholly the lung-tissue, which undergoes fibroid change. Every new formation, according to Virchow, whether homologous or heterologous, is really destructive, and destroys something of what previously existed; (3.) These fibroid elements are found most highly developed in the connective tissue surrounding the minute bronchial tubes and lobules, as well as in the neighbourhood of the thickened pleura, and would seem to show that the new formation begins in the connective tissue of the lung, and extends in every direction amidst the elastic fibres of the air-sacs, until these become degenerate and are obliterated. With regard to the origin and nature of the yellow or cheesy matter found amidst the grey fibrous induration, the microscope shows it to consist of a large number of granules, having bright sparkling centres, and of irregularly shaped and apparently shrivelled or wasted cells, filled with what are commonly called fat-granules. They thus seem to be retrograde matamorphosis in the fibroid formation, and to result from an imperfect nutrition process. In fibroid growths elsewhere, as in the uterus, circumscribed fibroid deposits in the kidneys, syphilitic tumors in the lungs and liver, and in cancer of the lung, similar yellow cheesy centres occur" (CLYMER).

As to the nature of this fibroid change, or *sclerosis of the lung*, Dr. Clymer gives the following summary of opinions:—

"By many of the British and French authorities, sclerosis of the lung has been described as chronic pneumonia; but in chronic pulmonary inflammation, the lung is not shrivelled or contracted, and, although rather hard and elastic, is comparatively readily broken down. The lung-tissue is firmer and drier than in true chronic hepatization, scarcely yielding a trace of moisture when scraped, and often creaking under the knife. Dr. Fuller believes that these cases form a connecting link between chronic inflammatory consolidation and tuberculous infiltration, and should be classed under the head of phthisis, as being more nearly allied to that disease than to simple inflammation of the lung (*loc cit.*, p. 258). Dr. Wilson Fox thinks that it belongs to the tubercular constitution, and is a more frequent termination of the tubercular process than the cheesy change. Dr. C. J. B. Williams, who published two cases of the disease thirty-four years ago, looks upon it as a modification of ordinary phthisis, its distinctive character being its tendency to chronicity, to which it owes its comparative curability. Dr. J. Pollock is of opinion that it is not a specific entity, and cannot be separated from ordinary tubercular disease, of which it is a mere complication or concomitant, its peculiarity being rather one of progress and development than of nature; while Dr. Greenhow regards it as differing from pulmonary consumption in its origin, progress, and issue" (see discussion on "Fibroid Phthisis" in the Clinical Society of London, *Lancet*, vol. i., 1868).

By Dr. Andrew Clark the disease is regarded as a local expression of a constitutional affection, such as rheumatism, syphilis, chronic alcoholism. The tendency of the lesion is to hyperplasia; and it is a frequent form of senile phthisis (STOKES, MACLACHLAN, CLYMER). Irritation, prolonged from some cause, is generally a constantly acting cause, and there is generally a previous history of chronic catarrh, combined with excessive spirit-drinking. It is a frequent form of lesion in the consumption of stone-masons, grinders, and the like.

(c.) *Tuberculo-pneumonic Phthisis and Tubercular Phthisis.*

"*The greatest danger to most phthisical patients,*" writes Niemeyer, "*is the development of tubercles;*" and this statement leads to an account of the relation that may be traced between *tubercles* and *phthisis.*

It has been clearly shown, alike by Addison and by Niemeyer, that patients suffering from *pulmonary phthisis* do not have miliary tubercles in their lungs at the commencement of their illness; but many become tuberculous during the course of the original lung-lesion as it progresses onwards to phthisical destruction and to cavern formation.

The occurrence of miliary tubercles in such lungs must now be regarded as a *complication* (NIEMEYER); and from the frequent occurrence of tubercles in lungs which contain remains of chronic inflammation, as cheesy infiltration and cavities, and the rarity of tubercles in lungs which are free from such remains, it is now presumed that some *causal connection* exists between the tubercles and nutritive changes in the lungs preceding their development.

Generally, it may be stated, that the more frequently the products of any inflammation (whatever its form or kind) undergo a cheesy degeneration (*tyrosis*), the greater will be the chance of the deposit of miliary tubercle; and when tubercles are formed in a lung previously healthy, and free from cheesy deposits and cavities, cheesy products (*tyrosis*) in stages of disintegration are to be sought for and found in other organs.

In 23 cases of acute tuberculosis examined by Buhl, out of 280 *post-mortems* of *pulmonary phthisis*, he found in all, except one, pre-existing masses of caseous matter, or pulmonary cavities. Rokitansky had also already observed a similar connection. More recently, also, C. Hoffmann, in 1867, found that miliary tuberculosis almost invariably occurs where caseous masses pre-exist, these being found not merely in the lung, but in the urinary and genital organs, in the lymphatic glands, and among the products of suppurative peritonitis. Thus the very diverse origin of the masses of detritus (*tyroma*), which are the starting-point of the disease, suggests some common form or quality of the caseous masses, and not anything specific in them.

Niemeyer, therefore, from this point of view, regards *tuberculosis "as in most cases a secondary disease, originating in some unknown way to us, in the action of cheesy morbid products on the organism"*—perhaps, also, directly producible, as Gasquet suggests, by the absorption of decomposed blood; or, as appears to me, by the absorption of any solid particles sufficiently softened down to admit of such absorption through veins or lymphatics.

In what way does tubercle thus originate? This question has been answered to some extent by the direct experiments of inoculation. Such inoculation experiments have been especially and directly made by Drs. Villemin, Burdon Sanderson, Wilson Fox, Cohnheim Chauveau, and Waldenburg, on rabbits, guineapigs, hedgehogs, goats, horses, and cattle. The experiments are performed by making a small incision through the skin, and inserting a small portion of the substance to be inoculated in the subcutaneous cellular tissue, or underneath the conjunctiva, or into serous membranes—which latter Dr. Sanderson considers infinitely preferable. The result is the production of miliary tubercles in different parts of the animal's body; which tubercles having been examined by Virchow, it is stated on his conviction and authority that such miliary formations are identical with real tubercle.

The general results may be stated as follow:—

(1.) In one series of experiments, the successful cases were those in which the tuberculous matter inoculated was already softened or caseous; while those inoculated with unsoftened tubercle gave negative results.

(2.) More or less extensive tubercular deposit in more than one organ of each animal was produced by the inoculation of perfectly fresh cheesy matter from the non-tuberculous lymphatic gland of a living scrofulous patient. Dr. Sanderson's experiments show that of all inoculated materials, none is more certain or more active than material taken (as vaccinators say) hot from the diseased glands of a living animal already infected. The dose required is almost infinitesimal. If a diseased gland is squeezed into a little distilled water in a capsule, and the resulting slightly turbid liquid injected into a pleura or peritoneum, the usual results are certain.

(3.) The inoculation of pus produced successive tuberculous eruptions corresponding to the several successive inoculations.

(4.) The poison which, when inoculated, produces tubercle is unaltered by long keeping in spirits.

(5.) Dr. Waldenburg's experiments with the use of coloured products for inoculating shows that solid elements inoculated are actually conveyed to the seat of the miliary formation.

(6.) Generally, it is concluded by Waldenburg, that "*miliary tuberculosis is a resorption disease, consisting in the taking up into the circulation of very minute corpuscular elements, and in their deposition by nodular formation* in numerous separate parts *of the various organs. Hence tuberculosis is a general disease—in a certain sense also a blood disease—although not a specific one. It stands in the nosological system nearest to pyœmia.* (*Med.-Chir. Rev.*, April, 1870, p. 404). The great difference however is, that in *pyœmia* the particles taken up into the circulation are larger, and so produce embolism, stasis, abscess, and local gangrene, besides causing violent irritation and toxic symptoms from their putrid condition.

The next pathological question of importance concerns the nature of the tubercle when so developed by inoculation.

First, there is the evidence of Virchow, who has examined them, to prove that the miliary formations which so arise are identical with tubercle.

Next, there is the conclusive evidence by Drs. Sanderson, Fox, and other experimenters, which shows that the result of the process of inoculation is to produce a definite disease, affecting almost all the internal organs by producing in them nodules of new growth. This new growth has that peculiar structure which is common to all those diseased products which Virchow calls *lymphomas*, because they present a structure found in certain organs of the lymphatic system. Dr. Sanderson prefers the word "*adenoid*" to characterize this tissue; and he regards the tubercles which are produced artificially as overgrowths of adenoid tissue, and not as new growths; and the parts most apt to be infected by tubercle are those in which the adenoid structure exists naturally.

In the lung the inoculated tubercle assumes two forms. The lung becomes disseminated with minute nodules of lobular catarrhal pneumonia. Each nodule is extremely translucent. On making sections, it is found to consist of two materials entirely different from each other anatomically. On the one hand, the alveoli are choked with the ordinary roundish cells which are always found there—often called epithelial; but their relation to the epithelial lining of the alveolar walls is not known. On the other hand, the alveolar walls are thickened by the growth in them of the adenoid tissue. As the disease progresses these masses of lobular pneumonia coalesce. Each mass *caseates* at the centre—that is, becomes opaque and soft; and the disintegration goes on till a vomica is formed. The disease progresses not by continous growth, but by the distribution or dispersion of infective material from one point—which Dr. Sanderson calls the focus of infection. He assumes that the communication of the disease from a part primarily affected to the rest of the body takes place by such distribution or dispersion, so that we have to do with primary and secondary lesions. It is the nodule of induration produced by inoculation which is the primary lesion; and from this the infective agent proceeds as solid matter in a state of extremely fine division (WALDENBURG, BURDEN SANDERSON).

Thus, it appears that all recent researches tend to bring tuberculosis into the category of infective diseases; and lead us to believe that the infective material from which it proceeds is infinitely more common, and the conditions for its production of much more frequent occurrence, than those which generate other morbid poisons. *Tuberculosis* thus implies a certain degree of malignancy; and when an organ or part is *tuberculous*, it means that it is in a state of destructive induration—that is, that the organ is first consolidated, and then becomes indurated and softened. They are the remnants of a process analogous to that of pneumonia; and practically they are found to consist either in enlarged lymphatic glands from hyperplasia, which afterwards undergo cheesy degeneration (*tyrosis*); or they are the cheesy remnants of pleuritic, pericardial, or peritoneal effusions; or the products of chronic inflammations of bones and joints.

Tubercles in the lungs are thus generally developed in a slow and insidious manner:—(1.) In the form of the transparent grey granulations, confined to one portion of the lung, where they gradually increase in size and number; or, (2.) They suddenly and rapidly increase, and are found in many parts of a uniform size, generally called miliary tubercles, which are regularly distributed through the pulmonary tissue. They may present different stages of retrograde metamorphoses at different parts of the lung. The recent grey tubercles are soft and gelatinous, those of older date are firmer; and when the tissues are extensively invaded, they are softened and infiltrated with a thick serous effusion. The course of such cases is generally acute. In acute phthisis they may reach the size of a pea in three or four weeks (LOUIS); and when subjects in a state of scrofulous cachexia are exposed to violent irritation of the lungs, these granulations grow so rapidly and in such numbers throughout the lungs as to give rise to most alarming dyspnœa (SIR JAMES CLARK).

It has been much discussed (and still is discussed), in which texture *tubercle*-matter is first deposited, and whether, at the time of deposition, it is fluid or solid, and what is the cause of the deposition or growth. The deposit in the lungs is the one most frequently described; and while we have had some most minute and elaborate descriptions of the site of deposit of tubercle in the lungs, they do no more than confirm the account which Schroeder Van der Kolk gave forty-two years ago (1826), when he fixed the seat of tubercular *deposit* in the extremities of the bronchial tubes, or in what are named the pulmonic air-vesicles. Similar views were adopted by Carswell and Andral. Dr. Addison, in his observations on pneumonia, in 1843, records that the earliest and simplest form presented by tubercles in the lungs is that of minute semi-transparent and generally hard bodies, inseparably attached to, and apparently incorporated with, the parietes of the air-cells of the lungs, in the same manner as the small tubercles are incorporated with the peritoneum or pleura. Dr. Sieveking has since confirmed the observation by his own researches; Schroeder Van der Kolk has again elaborately described the more minute details as to how the process takes place; and Dr. Radclyffe Hall gives a similarly minute description. But the experiments already described fix the formation of miliary tubercles *in* the walls of the air-cells; and this growth is supplemented with the material within the lobule, which had been alone regarded as one form of tubercle, appearing, in the first instance, as a degeneration of the previously existing normal epithelium; shedding off this, and continuous replacement and degeneration, till the whole of the interior of the air-vesicle is filled with this mass of new matter. Subsequently the pulmonic fibres become enclosed and separated by the morbid growth and exudation, and free nuclei and granules are formed between and amongst them. According to Virchow, such epithelium becomes "cloudy," and the cells degenerate, and the process of tuberculization advances in the cavity of the air-vesicle. This is also consistent with the experience of Hasse, who is of opinion that the tuberculous matter is first deposited on the walls of the vesicles, and gradually presses towards their middle the epithelium-cells, or other contents of the air-vesicle, and then encloses the whole as a kind of soft, and sometimes dark-coloured nucleus, (see p. 10, *ante*).

The question of epithelium in air-cells is still the subject of discussion. Its existence there is disputed by Rainy, Todd, and Bowman, Mandl, Ecker, Deichler, Zenker, Luscha Henle, Waters, Badoky, and Munk; but its existence in the air-cells has been maintained by Addison, Rosignol, Kölliker, Eberth, Radclyffe Hall, Williams, Hirschmann, and Hasse. It is also asserted by Eberth that in the lungs of calves and pigs there is a very delicate unbroken epithelium (Virchow's *Archiv.*, 1862, p. 503, where figures are given). Preparing healthy human lungs from young subjects by boiling the lungs in dilute acetic acid, I believe I have succeeded in demonstrating the existence of a delicate film of pavement epithelium lining the air-cells.

Another form of isolated tubercle in the lungs occurs as an interstitial growth, situated in those points where a certain amount of connective tissue exists. Its occurrence is in the form of little knots, or separate grains massed together, but always in their early stage recognized as bright sparkling spots like sand (SIBSON)—the usual *miliary tubercle.*

The seat of tubercle in the lungs may therefore be the mucous membrane of the bronchial tubes entering the alveoli, the walls of the alveoli themselves, the connective-tissue of the lung, the delicate walls of the minute pulmonary arteries, or the surface of the pleura; but the observations of Dr. Burdon Sanderson would still more limit and define the actual seat of true miliary tubercle. He says, "If we examine the unsoftened part of an ordinary phthisical lung, we find structural changes of two distinct kinds,—those of one kind having their seat in the alveolar *septa,* those of the other in the alveolar cavities. The change in the alveolar septa consists in the development of the tissue, which he calls adenoid lymphoid corpuscles embedded in stroma. The change in the cavities consists in the accumulation of cellular elements.

The structural elements of the human lung undergo a change just at the point where the small bronchial tubes lose their cylindrical character and become expanded on all sides into the *lobular passsages, groups of recesses* and *dilatations* composing the *air-cells* or *alveoli.* The muscular layer of the small bronchial tubes disappears, the longitudinal elastic bundles are broken up into an interlacement of *areolar* and *elastic* tissue, which surrounds the air-cells and forms the basis of their walls. The mucous membrane thus becomes exceedingly delicate, consisting merely of a thin transparent membrane, covered by a stratum of squamous instead of cylindrical epithelium. The walls of the air-cells, their orifices, and the margins of the septa are supported and strengthened by scattered and coiled elastic fibres, in addition to which, according to Moleschott, Gerlach, and Hirschmann, there is likewise an admixture of muscular fibres (QUAIN, SHARPEY, and CLELAND'S *Anatomy,* p, 900). It is in the meshes of these delicate structures of the air-cells that true miliary tubercle grows.

Cases are rare in which the occurrence of tubercle is not at first latent in the lung; and from the numerous cicatrices that are found after death at the apices of the lungs.

The pneumonic lesions of the lungs occurring in early life frequently heal, although they generally return (sometimes at an advanced age) and ultimately prove fatal (SIR JAMES CLARK). There are few cases, also, in which the destructive and ulcerative progress of pulmonary phthisis is uniform. It is continually being checked, and for a time slumbers; and even in the worst specimens of pulmonary phthisis numerous cicatrices and evidences of attempts to heal may be recognized; but as one portion cicatrizes another becomes the seat of further induration, and softening, and cavities.

Cicatrices of healed lesions present different appearances, according as the cavities have been superficial or deep-seated. When superficial, the pleuræ are more or less adherent and thickened, frequently thus forming an external boundary to the cavity. When the walls of the cavern contract, the pleural surface of the lung is drawn inwards, and thus the irregular puckerings on its surface are produced. Occasionally no traces of scrofulous matter or tubercle are discovered either within or in the vicinity of these cicatrices; but more generally the contraction and puckering of parenchyma occur round indurations which have undergone various transformations, and sometimes a cyst encloses the mass. The enclosed growth may be found to have undergone any of the processes of transformation already noticed; and the cretaceous or calcareous concretions may remain an indefinite time in the parenchymatous substance of the lungs, or they may be evacuated through the bronchi with the sputa. Thus, masses of new material are sometimes absorbed or thrown off, and the evidence of this may be summed up under the following points:—(1.) Small puckered cicatrices, with loss of substance, after subsidence of the systemic disturbance characteristic of pulmonary phthisis. Strumous growths in the lungs of children seem capable of absorption without undergoing any change, such as cretification or softening. Most frequently it seems in them to affect mainly the small lymphatic glands along the outer walls of the air tubes. (2.) The conversion of the indurated lesions into cretaceous or horny masses, which are either expectorated or remain latent. (3.) Isolation of the growth by so-called plastic material; adhesion and thickening of the pleura; adhesions and lymph thickenings round cavities, analogous to cysts round clots of blood or musket balls. That such appearances are really evidences of arrest of the phthisical or destructive process is rendered apparent by the following facts:—

"1. A form of indurated and circumscribed lesion is frequently met with, gritty to the feel, which, on being dried, closely resembles cretaceous concretions.

"2. These concretions are found exactly in the same situations as the indurations of pulmonary phthisis—most commonly in the apex, and in both lungs. They frequently also occur in the bronchial, mesenteric, and other lymphatic glands, and in the psoas muscle, or other textures which have been the seat of inflammatory growths or scrofulous abscesses.

"3. When a lung is the seat of pneumonic infiltration throughout, which has passed into the condition of pulmonary phthisis, recent tubercle generally occupies the inferior portion, and older tubercle and perhaps cancer, the superior, whilst cretaceous and calcareous concretions will be found at the apex.

"4. A comparison of the opposite lungs will frequently show that whilst on one side there is fine encysted lobular infiltrations, partly transformed into cretaceous matter, on the other the transformation is perfect, and has occasionally even passed into a calcareous substance of stony hardness.

"5. Seeing that, according to the observations of Dr. W. T. Gairdner, cicatrices also may result from bronchial abscesses, the seat of cicatrices in the lungs may vary considerably" (Bennett).

In the words of Carswell, "pathological anatomy has perhaps never afforded more conclusive evidence in proof of the curability of a disease than it has in that of pulmonary phthisis." Pathology, therefore, teaches most distinctly that the aim of the physician must be to correct the conditions which lead to the further occurrence of tubercle. The most eminent physicians are agreed that it is peculiarly a disorder of childhood and youth, "when nutrition is directed to building up the tissues of the body," that such persons are frequently attacked with symptoms of phthisis, which under proper treatment cease, "and years elapse before there is any renewal of the disease, and that were advantage taken of the intervening period to correct the constitutional cachexia or vulnerability to morbid influences, the cure might prove complete." During remissions of the systemic disease, there is toleration of the lesions; but the remission ceases with renewal and extension of the local lesions.

The result of the course followed by pulmonary phthisis, when observed in patients under treatment in hospital, may be arranged, according to results obtained by Dr. Walshe, under the following heads:—

1. All the symptoms were removed and the physical signs reduced to a passive condition in about 4½ *per cent.* of the cases admitted, without reference to the stage of the disease, or to the severity of the primary or secondary morbid changes.

2. If the persistence of some active physical signs is disregarded, such as a continuance of cavernous rhonchus, all the general symptoms disappear in nearly 8 *per cent.;* and complete removal of all the symptoms was more frequently effected in the *male* than in the *female.*

3. More than half the cases of phthisis undergo temporary stages of improvement more or less permanent; and the time the disease has existed, rather than the stage the disease has reached, is an important element in calculating the probable benefit a patient may derive from treatment in hospital.

"In a given mass of cases the chances of favourable influence from sojourn in the hospital (at Brompton) will be greater, in a certain (undetermined) ratio, as the duration of the disease previous to admission has been greater; in other words, natural tendency to a slow course is a more important element of success in the treatment of the disease than the fact of that treatment having been undertaken at an early period" (*Med.-Chir. Review*, Jan., 1849).

General Symptoms of Pulmonary Phthisis.—These vary according as to whether the case be one following *acute inflammation of the lung alone,* or of *tuberculo-pneumonic phthisis,* or of tuberculosis only.

The several symptoms on which the diagnosis of pulmonary phthisis may generally be made are the following:—

(1.) *Increased frequency of respiration,* not always attended with *dyspnœa,* without dulness on percussion, and without bronchial breathing. Dyspnœa may be felt only at times—as when the respiratory wants are increased by fever—and therefore by increased tissue change. In some cases the increased frequency of respiration (48 to 54 per minute, the pulse being 104 to 112, with dyspnœa) may be the most troublesome symptoms of phthisis, and generally due to the diminution of the respiratory surface, from the filling up of the air-cells with tubercle, and the occlusion of bronchioli by the accompanying catarrh, or rarely to pain on respiration and to pyrexia, (100° to 102° Fahr.) Dyspnœa is only present when several of these causes act together. Miliary tubercles, *per se,* as a rule, escape detection by physical examination.

(2.) *Pains in the chest and shoulders* are often absent, or occur as dull, aching, "flying" pains about the collar-bones, or under one or both shoulderblades.

(3.) *Cough and expectoration precede pulmonary phthisis*, as a rule, by a longer or shorter time (two or three weeks), indicating a catarrh, which spreads to the alveoli, as a catarrhal pneumonia—the tyrosis and disintegration of which is the destructive process—phthisis or consumption of the lung and the body. This is the condition generally seen in measles, hooping-cough, and such cases as occur with the exanthemata.

(4.) *Character of the sputa expectorated.*—The finely and sharply marked deep yellow streaks of mucus indicate catarrh of the smallest bronchi, the inflammatory product being rich in cells,—a form likely to pass into the air-cells. Intimate admixture with blood indicates the beginning of a pneumonic process.

(5.) *A tedious and troublesome cough*, with little expectoration, and with pyrexia, are not so suspicious symptoms of pulmonary phthisis as a pneumonic process with tubercles of the mucous membrane of the bronchial tubes and of the alveoli.

(6.) *A hoarse or aphonic cough* signifies complication of pneumonic pulmonary phthisis with tubercle.

(7.) *Increase of temperature* indicates extension of catarrh from bronchioli air-cells; and its persistence testifies to the continuance of the pneumonic process. The difference of morning and evening temperature amounts, as a rule, to 1·8° to 3·6° Fahr.; rarely less—very often more. Frequently in the morning the temperature may be normal, while in the afternoon or evening it may be 102·2° Fahr., or even higher; but when tubercle complicates pneumonic pulmonary phthisis, the differences between morning and evening temperature are much less.

(8.) *Impoverishment of blood and emaciation* are constant and in relative proportion to the height and persistence of the fever.

(9.) *The physical signs* indicate one or other of two conditions, or a combination of both, but which latter condition is not constant. These conditions are either (*a.*) Tuberculosis; or (*b.*) Inflammatory processes which have led to infiltration, cheesy degeneration (tyrosis), disintegration, with shrinking or destruction of lung-tissue; or (*c.*) Both these conditions may be combined.

Physical examination of the apices yields negative results so long as peri-bronchitic and pneumonic deposits are not massed together into extensive consolidations, such as to produce dulness and bronchial breathing. But a protracted catarrhal affection of the apices must always be regarded as a grave sign, because it tends to such consolidations. It is chiefly from pyrexia being persistent, impairment of general health, loss of flesh, and pallor of the skin, that a diagnosis is made as to the presence of catarrhal inflammation of the bronchioles and air-cells; but from which, by careful management, recovery may yet be made. Catarrh of the apex of the lung is not therefore always a certain sign of pulmonary *tubercles*; but it is a sign *that the patient is in danger of suffering from pulmonary phthisis or consumption.*

Dulness on percussion, bronchial breathing, and consonant râles in the upper part of the thorax are probably never caused in the first instance by tubercles alone, but indicates consolidation, the remains, most probably, of a pneumonic process. Other symptoms require to be expressed in addition to these, before it can be said that tubercles also exist.

Flattening of the supra and infra-clavicular regions on one or both sides, and lower situation of the upper edge of the lung, with diminished respiratory movement, are always certain indications of a diminution in size of the apex of the lung by induration and shrinking; and may indicate thus far a curative process, which is only apt to be compromised by a fresh pneumonic attack, or a subsequent deposit of tubercle.

(1.) *Symptoms of Acute Pneumonic Phthisis.*

The inflammation of the lung which precedes the destructive process may itself be either acute or chronic, and be associated with hæmoptysis or extensive bronchial catarrh.

If the fever of an acute pneumonia does not cease at the end of the first, or beginning of the second week; if in the evening hours the fever rises considerably, when it only remits in the morning with considerable sweating; when percussion-dulness persists, and liquid clanging rhonchi are still heard over the affected part, then in such a case acute pulmonic phthisis may set in.

The sputa become muco-purulent; and when an examination, microscopically, is made, and the characteristic elastic fibres of the lung are to be seen in them, it is evident that the cheesy tissue of the diseased lung is breaking up, and the diagnosis of acute pulmonary phthisis is no longer doubtful, and which may prove fatal in a few weeks, especially if the fever is violent.

But sometimes the fever ceases, expectoration diminishes, and the patient improves, retaining signs of induration, however, and retraction of the parietes of the chest over the affected part, and often even signs of a cavern (generally from bronchiectasis) in the pulmonary substance.

When an acute catarrh is likely to terminate in pulmonary phthisis, that result is generally indicated only by the comparative gravity and endurance of the symptoms, rendered more certain by the sputa becoming rusty, and by physical signs of pleurisy or consolidation of the lung.

The chronic form of pneumonia which precedes acute pulmonary phthisis may begin very insidiously. A slight catarrh is usually the first indication of its commencement, in which the general health suffers, the patient becoming thin and losing his appetite. Then comes increased body-heat, with physical signs of consolidation and softening of the lung. This chronic form of catarrhal pneumonia may tend, on the one hand, to recovery or to extension, more or less rapid, of the induration; and the typical cases are such as find their way into hospital one winter after another, improving under the careful nursing, food, and medicines for the time; but each attack spreading the induration and subsequent softening over a greater extent of lung, till at last they die from a severe pneumonic process or extensive deposit of tubercle in the lung. This latter event is the greatest danger to most consumptive or phthisical patients. The deposit may occur at any stage; and hence no form of pulmonary phthisis can ever warrant an unconditionally favourable prognosis.

(b.) *Symptoms of Tuberculo-pneumonic Phthisis.*

These cases are generally more or less chronic, and the tubercular element is indicated in the commencement with some slight cough of a dry hacking character, most frequently induced on rising in the morning and going to bed at night. The cough at first seems simply intended to clear the throat, the irritation being mainly referred to the pharynx, which is often red, rough, and coated with tenacious mucus. As the cough becomes more fully expressed a scanty expectoration occurs, of ropy or glairy mucus, hardly discoloured, or only slightly streaked or stained by a trace of blood. Dyspepsia, sick headache, biliousness, and loss of appetite prevail. The patient is feeble, easily fatigued, feels unequal to his usual work, has burning heat of the soles of the feet at night, and some perspiration in the morning; his nights are restless, so that he rises in the morning weary and unrefreshed by sleep. He is irritable and often depressed in spirits, his appetite capricious, with dyspepsia—a most constant, important, and early symptom,—and he is convinced of a sensible loss of flesh. His muscles become flabby, the countenance pale, the conjunctiva becomes pearly-white, and the pupil of the eye dilated. These symptoms are accompanied by a permanently accelerated pulse, from 90 to 140. The cough after a time begins to recur at intervals during the day, and especially after the least exertion. He is now sensibly aware of being "short-winded," so that active exercise exhausts him, and he must rest at intervals if he walks fast or goes up a stair. The number of respirations per minute is now increased, the pulse accelerated, especially towards evening, and deficient in force. Febrile paroxysms are persistent and of daily occurrence; and elevation of temperature, as measured by the thermometer, is a constant phenomenon capable of being appreciated for many weeks before physical signs become decided (DAVY, RINGER). There is also correspondence between the elevation of temperature and the activity of the growth of tubercles in the lungs; such that when it goes on rapidly the elevation of temperature is high, and *vice versa.* This elevation of temperature is persistent, varying from 103° to 104° Fahr., for several weeks before diminished weight or physical signs indicate the undoubted presence of tubercle (RINGER). This stage or state of things may last a few weeks or a few months; and even the patient often revives, and seems, to an unpractised eye, for a short time to have recovered his good general health, were it not for a sense of weakness and undue exhaustion after such exercise as he has been daily accustomed to.

The disease, however, silently proceeds, and all the preceding symptoms are gradually but sensibly aggravated. A large amount of tubercle may have grown during the earlier stage; and after a time the intervening portions of lung become so congested that bronchial irritation, with intercurrent attacks of pneumonia, are of frequent occurrence. Rapid softening of the tubercles takes place in those in whom constitutional weakness or vulnerability is intense; and with the continued discharge of pus from the lungs, hectic fever becomes permanently established, and the sweat from the head and chest towards morning is often so profuse that the patient lies bathed in perspiration. The cough is then more distressing, the sputa purulent, and the pulse more frequent, perhaps from 90 to 110. The emaciation, consequently, is now well marked and decided. The duration of these phenomena is very indefinite—a few weeks to many months may end in a fatal issue. During its progress, however, the disease occasionally intermits and becomes latent, so that often there is for a time a marked amendment, and the patient regains some strength—a most important interruption, to be sedulously taken advantage of for the purpose of promoting the tendency to cure. But fresh tubercles may continue to grow during these intervals, so that the lung-tissue is still further encroached upon by the newly-formed growth; fresh local irritation is set up, all the general symptoms are aggravated; sickness and rejection of food are now often excited—the expectoration becoming more profuse, of a purulent character, and often streaked with blood.

When these symptoms attain their highest degree of intensity, and caverns are formed, the hectic is followed by cold clammy sweats and great exhaustion towards noon and again at night; the appetite is lost; a colliquative diarrhœa may supervene from the deposit of tubercles and ulceration of the intestines; the sputa are often pure pus, as from an abscess, but at length they become little more than a rusty sanguineous mucus; the pulse rapidly increases to 110 or 150; the emaciation is excessive; the hair falls off; catamenia cease; pleurisy or pneumothorax may supervene; ulceration of the larynx; and, amidst this general wreck of material existence, the Mind is often firm, collected, and even hopeful to the last.

As soon as the cavity which forms is in free communication with the bronchi, the cough is often greatly relieved. The duration of this stage is generally shorter than the former; but still, notwithstanding the existence of one or more cavities, it often lasts many months. As the fatal end approaches, the appetite fails more completely; sleep can only be obtained by narcotics; the integuments on which the patient lies are apt to become inflamed, sore, and even to die from the constant pressure; œdema of the feet and ankles sets in; and with the approach of death, suffocative dyspnœa may render the death-struggle extremely painful. In other cases a wandering delirium enshrouds the Mind, or coma supervenes; and the prolonged and weary illness is tranquilly ended by the gentle and welcome approach of death.

Such is a short outline of the course and phenomena of this destructive disease. It sometimes terminates life within a few weeks, or extends over six or eight months, while it occasionally lasts several years, with marked intermissions in its progress.

There are certain circumstances which promote the chances of a remission, and of which important advantages may be taken in the treatment—namely, (1.) Originally good constitution; (2.) Non-inheritance of scrofula; (3.) Direct inheritance of longevity; (4.) Limited extent of local lesion; (5.) Integrity of other organs, unimpaired digestion, absence of fever, vigorous nervous system, quiet pulse; (6.) Influence of age—the period when the young body is making its growth is the period of greater danger. The tolerance of lesions and the periods of remission are less during the growing age, and are increased just when the body has completed its process of increase.

The following is a short analysis of the principal local, constitutional, and stethoscopic signs of pulmonary phthisis.

Affections of the bronchial membrane and mucous membranes of the pharynx and larynx are certainly the most frequent concomitant symptoms of phthisis; but the part of the bronchial membrane affected is not always the same. Most commonly the mucous membrane of the smaller bronchial tubes is first affected; then that of the larger ones, the disease gradually ascending till it often ends in a *chronic laryngitis,* with a partial or total loss of voice. In a few cases, however, this order is inverted, and almost the first symptom is a *laryngitis,* with *hoarseness,* partial loss of voice, and constriction of the throat; after which the disease descends to the larger and then to the smaller bronchi, when the patient begins to expectorate; his pulse becomes hurried; he loses flesh; and all the unerring symptoms of phthisis are established; while *pleurisy, pneumothorax, pneumonia, bronchitis,* and *hæmoptysis* are the more important intercurrent complications; while amongst the more chronic are *ulcerations of the bowels, scrofulous inflammation of the epiglottis, larynx, trachea, bronchial glands, or tuberculous meningitis.*

The *expectoration* which takes place in phthisis from the bronchial membrane is usually purulent, the pus thrown up in the early stages being for the most part of good quality, and formed into "sputa," sometimes sinking, and sometimes swimming in water. It may be either of a sweet, insipid, or saltish taste, as experienced by the patient. As the disease advances, pus is often expectorated pure, as from an abscess, and without any separation into sputa, and is sometimes mixed with particles of a curdy substance.

In the last stages of consumption the sputa are often of a rusty green, a dirty sanies, or a rusty muciform serosity. The quantity expectorated varies greatly—sometimes only a few sputa, or not more than half an ounce in the twenty-four hours, and then perhaps more than a pint in the same period, so that in a few weeks the patient has often expectorated more than his own weight of pus. If a small abscess has burst into the bronchi, the sputa, though somewhat increased in quantity, are hardly changed in character; but if the abscess be large, the quantity thrown up is proportionally great.

Hæmoptysis.—Hæmorrhage may precede, or be contemporaneous with, or succeed to the pulmonary affection. If it precedes, the patient, being as he imagines in excellent health, is suddenly seized with *hæmoptysis,* followed by cough. This attack subsides, but a second and third follow, till the condition of pulmonary phthisis becomes undoubted; for in the majority of cases *hæmoptysis* is followed by a more or less serious irritation of the lung and pleura (pleuro-pneumonia), which may for once be resolved, but if often repeated, generally ends in phthisis. When such is the result, it is indicated by the continuance of fever, and proportional impairment of the general health. Slight pains are felt in the chest, and generally considered rheumatic by the patient; the respiration remains hurried, and muco-purulent expectoration commences. In most of the cases in which a healthy person thus suffers after an attack of hæmoptysis, it is presumed *that blood remains behind in the bronchi and alveoli, leading by its irritation to a pneumonia which undergoes the cheesy transformation* (tyrosis), *and afterwards breaks down.* From such causes numerous and excessive losses of blood by hæmoptysis are apt to occur, generally from erosion or bursting of a branch of the pulmonary artery running along in the wall of the cavity. In this respect, however, the hæmoptysis of phthisis must be distinguished from the recurrent and minute hæmoptysis of an aneurism opening into the air-passages.

Hæmoptysis more commonly, however, occurs later in pulmonary phthisis, increasing the debility, aggravating the symptoms, and hastening the fatal issue. The quantity of blood lost is sometimes only enough to streak the sputa, at others a few tea-spoonfuls, but in some instances so profuse as to amount to one, two, or more pints. In the still more advanced stages, though cases occur in which the quantity of blood thrown up is very great, yet more usually it is trifling, and more resembles a bloody sanies than pure blood.

An attack of hæmoptysis occurring without any obvious cause or injury is of bad omen. It shows some morbid friability of the branches of the bronchial arteries terminating in the bronchial mucous membrane, and associated with a tendency to inflammatory diseases of the pulmonary tissue.

The *dyspnœa* is generally great in phthisis, the patient being unable to make any active exertion, or even to read a few lines without pausing. The dyspnœa, however, is not always proportioned to the amount of mischief; for there are instances in which the respiration has been performed with facility, even when two-thirds of the lungs have been in the destructive disintegration of pulmonary phthisis. It is when the respiratory surface becomes considerably diminished by deposit of tubercles in the bronchi and air-cells that dyspnœa becomes most expressed; so that a high frequency of respiration, without percussion-dulness and without bronchial breathing, is one of the most important signs that the phthisis is "*tubercular.*" If a patient with consolidation and destruction going on of the lung has hitherto or only slightly been short of breath, is observed to get a high frequency of respiration and painful dyspnœa, without those phenomena being explained by an increase in the physical symptoms of consolidation and destruction of the lungs, or increase of pyrexia, then it is to be strongly suspected that the existing phthisis is complicated with tubercle.

The Stomach is more or less diseased in three-fifths of the cases of phthisis; yet it so seldom gives rise to any well-marked symptom, that for the most part any lesion there may be said to be latent. In the worst cases the symptoms are only a capricious appetite, indigestion, some pain in the epigastrium, and vomiting after coughing. The indigestion is peculiar. With reference to fat and fat meat, the dislike of consumptive patients to such food is unquestionable; so that the fat and the lean parts have to be adjusted to the likings of the patients; and it is generally of no use giving to them any but the leanest parts of the meat. They are partial to fish, and especially to soles.

The Intestinal Canal is at least as frequently affected as the stomach in phthisis; but in general the abdomen is without pain. The only marked circumstance is, that the stools are more copious than in health, the body being unable to appropriate the accustomed quantity of nutriment prepared by the stomach. As the disease advances, the patient often suffers from irritable bowels, or from diarrhœa alternating with constipation; while, towards the close of the disease, the diarrhœa often becomes colliquative, hastening the fatal result. In some few instances the peritoneum ruptures, and the patient dies of *peritonitis;* while in a somewhat larger number *ascites* is a common occurrence.

The Liver undergoes fatty degeneration in about one-third of the cases, and so remarkable a lesion might be expected to give rise to some particular symptoms; but this is not the case. It may occasionally be felt somewhat enlarged, but neither pain, nor altered state of the secretions, or other circumstances, denote its diseased condition. How far this condition results from the feeding on cod-liver oil and cream or milk, the most usual remedies in pulmonary phthisis, is not yet sufficiently appreciated; but so large and constant a supply of fat must have no small influence on the induction of fatty liver.

The emaciation so remarkable in this disease is common to nearly all the tissues of the body—the adipose tissue, the muscles, the bones, and even the intestines and skin are thinned. This emaciation often commences before the disease can be said to be well established, so that the patient has often lost one or two stones in weight before he applies for medical advice. In the more advanced stages emaciation progresses in a peculiar manner, the patient losing perhaps three pounds in one week, and gaining two pounds or more in the next; and this alternation of gain and loss goes on for many weeks or months, but generally leaving a balance against the patient. Towards the close of life the loss greatly surpasses the gain, and occasionally amounts to four, five, six, and seven pounds in a week. The total loss the patient sustains is perhaps from one-third to half his whole weight.

The Mind, though not capable of continued exertion, is generally perfect throughout the disease, or only wanders during the few last days of existence. It is seldom the patient dreads the future, or despairs of the present; for nature, however threatening his symptoms, has imparted a singular buoyancy to his hopes. He always says he is better; would be quite well but for his cough; feels able to take a long walk; and sometimes (in expectation merely) enjoys his meals;—yet, with all this, he may faint if he attempts to cross the room, or nauseate when his food is brought to him.

Such are the general and special symptoms of *pulmonary phthisis.* A physical examination of the chest will yield many valuable additions to our means of diagnosis, and enable us to determine, not only that the lung is diseased, but also the particular part, and the state in which it may be. Thus the discoveries of Laennec and Avenbrugger have now rendered the diagnosis of pulmonary lesions almost as perfect as though the disease was exposed to sight. Nevertheless, there is no *single sign* by which the existence of tubercles in the lungs is clearly indicated; therefore the general symptoms must invariably be judged of in combination and comparison with the information obtained by physical signs.

Not a little has been written as to the possibility of detecting what has been called a "pretubercular stage of phthisis" (E. SMITH and others). By this expression writers mean that there exists an abnormal physical condition of the lungs and of the body preceding the deposition of miliary tubercles in the lungs, which condition is capable of demonstration by certain signs or symptoms.

Some physicians believe that no local physical sign of disease is indicative of the existence of phthisis *before the growth of tubercle*, but that the tendency to the tuberculous state is to be inferred from the presence of a series of cachectic symptoms, which from experience are frequently found to terminate in tuberculous disease of the lungs. Others believe not only that such symptoms are present, but that they can detect such physical signs as are believed to indicate a peculiar local condition of the lung—*a stage anterior to the deposition of tubercle.* "The slightest subclavicular dull percussion sound, with lessened vesicular murmur, less forcible and deep inspiration, and flattening of the apex of the lungs," are described as the physical signs of this so-called *pretubercular* state (MARKHAM). But, however much practitioners may accustom themselves to detect the physical signs of known pathological conditions, there are states of the body between cachexia and local lesions so nicely balanced that no definite local morbid state can be ascribed to them, and therefore no physical signs can be associated with such a negative position apart from those of health. The so-called "pretubercular stage of phthisis is undoubtedly an instance of this kind. The best auscultators have admitted that there are no distinct and infallible signs by which we can with certainty diagnose the early existence of miliary tubercle; and it is also known that solitary growths of tubercle do not of themselves produce the slightest change in the percussion sound of the lungs." On the contrary, most physicians believe that the slightest subclavicular dull percussion sound, the less forcible, deep, harsh, or tubular inspiration, with lessened vesicular murmur, prolonged expiration, increased vocal resonance, and flattening of the apex of the lungs, when combined with the well-known general symptoms already mentioned, leave little doubt that the actual presence of destructive lesions and perhaps, but not necessarily, tubercle is indicated. While, therefore, it is imperative on the student and practitioner to educate their ear to the utmost in the detection and appreciation of the finest thoracic murmurs, it is unjust, as already observed, to expect through the stethoscope more information than it is fitted to convey. A too exclusive study of physical signs, to the almost disregard of general symptoms and the teachings of morbid anatomy, not only does injustice to the

Science of Medicine, but the lives of patients are endangered when treatment is solely founded on the former. It is by general symptoms, such as those which Todd, Clark, Bennett, Niemeyer, Ancell, Hutchinson, and Ringer have so fully elucidated, that the practitioner will be able to recognize a "pretubercular stage of phthisis," and not by local physical phenomena alone.

A very interesting illustration of this is afforded by some recent observations made by Dr. Sidney Ringer on the value of thermometric observation as indicative of a pretubercular stage of phthisis. He has been able to recognize a persistent elevation of temperature as the invariable precursor of the growth of tubercle in any organ. This persistent elevation of temperature existed for several weeks before diminished weight or other physical signs indicating the growth of tubercle in the lungs could be appreciated. After a certain time, however, growth of tubercle began to be apparent by physical signs in the apices of the lungs. This elevation of temperature is probably due either to the general condition of the body or to the deposition of tubercle in its various organs. It is probably due to the state of the body, rather than to the second of these processes; and if so, may not the rise of temperature be regarded in the light of the rise of temperature from an infective process going on, as in pyæmia? The temperature may be taken as a measure of the amount of the deposit, and any fluctuations in the temperature indicate corresponding fluctuations in the intensity of the disease. The temperature is a more accurate measure of the amount of lesion than either the physical signs or rational symptoms. By means of increased body-temperature we are able to detect the deposit of tubercle long before any physical sign is present, and when the symptoms are insufficient to justify such a diagnosis. By means of the body-temperature we are able to diagnosticate tuberculosis, even when throughout the course of the disorder there is no physical sign indicating the tuberculous deposition in any organ, and where the symptoms are insufficient to lead to a correct diagnosis. It is probably by means of the body-temperature that the abatement in the active general condition and the cessation of tubercular deposition may be ascertained, and the conclusion reached that the persistence of the physical signs is due to obsolescent tubercles, and to chronic thickening of the lung-tissue between the deposits. In illustration of this latter statement Dr. Ringer gives two cases. His observations were made on patients in University College Hospital; and he kindly permitted me to read, in MS., and to use, a summary of his observations.

Physical Signs.—In the following details a systematic examination of the patient is prescribed, after the method recommended by Dr. Fuller; otherwise the diagnosis is a matter of extreme uncertainty to those unpractised in conducting a physical examination of the chest. The physical signs are obtained by—(1.) *Inspection;* (2.) *Palpation;* (3.) *Mensuration;* (4.) *Percussion;* (5.) *Auscultation* of the chest.

The physical signs of pulmonary phthisis, in the order of their relative frequency, may be stated as follow:—*Dulness of percussion* (constant); *deficiency of respiratory murmur; bronchial voice; rough in-*

spiration; expiration prolonged; mobility of chest-wall lessened; flattening of chest-wall; tubular quality of respiration; dry crepitation; wavy inspiratory sound.

The physical signs denoting a few small scattered tubercles are,—(a.) *Inspiration rough and dry, and its intensity increased;* (b.) *Expiration prolonged—compared with its normal intensity and duration, as eight to two;* (c.) *Bronchophony in rare cases* (POLLOCK).

Of indurations, and perhaps tubercle in groups or masses, the physical signs are,—*Pulmonary crumpling sounds; dry crackling rhonchus; sonorous sibilant rhonchus, indicative of bronchial irritation; inspiration rough and dry, intensity increased; the intensity and duration of expiration increased, and its quality blowing; slight bronchophony; diminished local fremitus; slight dulness, localized and distinct; heart's sounds transmitted; and in certain cases a subclavian murmur* (POLLOCK).

In eighty cases of incipient phthisis observed by Dr. J. Pollock, the relative frequency of the physical signs was as follows, commencing with the most frequent:—*Mobility of chest-walls lessened; flattening of chest-walls; dulness on percussion; deficiency of respiratory murmur, rough inspiration; expiration prolonged; tubular quality of respiration; bronchial voice; dry crepitation; wavy inspiratory sound.*

If the chest of a patient labouring under incipient phthisis be carefully examined, we may at first observe nothing remarkable, unless the indurations shall be large in amount or confined to one side of the chest. Abnormally rapid respiratory movements indicate a large amount of tubercle; and there is decreased expansion of the chest in the *infra* and *supra*-clavicular regions on the affected side, or flattening of the chest-walls at the place affected; and if both sides are equally affected, this flattening is apt to escape attention. If the disease be further advanced, we find the patient emaciated, together with a singular immobility or incapacity of dilatation of that portion of the chest immediately below the clavicle, so that he breathes chiefly by his shoulders and diaphragm, and is unable to "fill his chest," especially during forced inspiration. The respirations are now greatly increased in frequency. In the latter stages of the disease the respiratory movements are still more rapid. The clavicles appear peculiarly prominent, in consequence of the flattening, depression, or falling in of the *supra* and *infra*-clavicular regions. A fluctuating impulsive movement may be obvious in the upper intercostal spaces, generally on the left of the sternum, due to the action of the pulmonary artery or base of the heart. At a still more advanced stage, if an abscess has burst into the cavity of the pleura, and caused *pneumothorax*, the affected side is not only motionless, but distended and, as it were, bulging out.

Palpation during the *early stage* may disclose deficient expansion in the *infra* or *supra*-clavicular regions; and if there is increased vocal fremitus at the apex of the lung, it betokens consolidation of the lung-tissue underneath. In the *advanced stages* of the disease, marked vocal fremitus betokens a large cavity, superficial, and in free communication with the air passages. It may be rhonchial, or gurgling, or like fluctuation. If the lung has shrunk, the heart may be felt beating out of its proper place.

Mensuration with calipers, in the early stage, may detect a slight diminution in the *antero-posterior diameter* of the *infra-clavicular* region, and a decrease in the local expansion movement. As the disease advances, the size of the side affected diminishes both in its *transverse* and *antero-posterior* diameter, probably due to atrophy and cicatricial-like contraction of the lung, collapse of air-cells, or firm pleural adhesions.

Percussion during the earlier stages, so long as air penetrates the pulmonary tissue equally on both sides of the chest, does not convey any definite information. Dulness does not always follow even when considerable masses of tubercle exist at the apex, unless the masses are very superficial, when the sense of resistance to the fingers is also increased. But if a portion of healthy or emphysematous lung intervenes between the consolidated lung and the chest-walls, there will still be clear resonance over the affected part. Percussion should be practised during deep inspiration, and then during deep expiration. If tubercles exist, it will then be found that in percussion, under a full inspiration, the resonance is *increased* on the affected side, but very *slightly* as compared with that on the healthy side; whereas, if percussion be made under a full expiration, the dulness will be far greater on the affected side than over the healthy lung (FULLER, pp. 42, 44, 365). It is differences rather than actual quality of sound on which an opinion must be based. In the more advanced stages, percussion elicits evidence of dulness more intense and more widely spread. In the latter stages, where *vomicæ* exist, percussion will be absolutely dull if one or more small vomicæ are filled by purulent material, or surrounded by consolidated lung. If, however, the same cavities are empty and superficial, without thickening of the pleural membrane, the dulness may be slight, or the sound on percussion resonant, though shallow or amphoric. "Large empty vomicæ, with thin tense walls, yield an amphoric or cracked-pot resonance, . . . and, except in rare instances of enormous superficial vomicæ, with thin tense walls, it is almost impossible to judge of the size of a cavity by the results of percussion" (FULLER, *op. cit.*, p. 369).

A superficial empty cavity, with resilient walls, having free communication with the upper air-passages, yields a cracked-pot sound, especially when the mouth is open, and all obstacle to the egress of air removed.

Auscultation yields extremely variable results. Slight harshness of respiration is the first indication noticeable, with prolongation of the *expiratory* murmur, and "jerking irregularity of the respiratory sounds." When these phenomena are persistent, and confined to one side of the chest, they indicate tubercle, especially if the phenomena of "dry clicking" be added. These phenomena are earliest marked in the *supra-scapular*, *supra-clavicular*, and *infra-clavicular* regions. If the physical signs of bronchitis (namely, coarseness of respiration, with small bubbling râles and sonorous sibilant rhonchi) are persistent in these regions, and inaudible below the second intercostal space, and still more so when confined to one lung, their existence warrants the suspicion of tubercle. If the disease is advanced, and the growth of tubercle involve bronchial tubes of considerable size, there is almost entire absence of vocal resonance and of respiratory murmur over the part affected, with the sounds of breathing exaggerated in the adjoining parts. If consolidation is extensive, the sounds of the heart and large vessels are transmitted to a greater extent than in health.

The diagnostic value of murmur in the pulmonary artery is variously estimated. It is often present in many phthisical patients at the second left sterno-costal articulation (FULLER, p. 366. See also Sieveking, in *Lancet*, Feb. 11, 1860).

As the disease advances into the second stage, auscultation indicates the presence of thin irregular-sized bubbling râles, from the passage of the inspired air through the softened and liquefied tubercles. It may also prove that coarse and hollow-sounding respiration exists over a more extensive surface than heretofore; or that the respiratory sounds are of a blowing character (bronchial breathing). In still more advanced conditions, with more or less empty cavities, the respiration is heard to be blowing or amphoric and metallic; and if the fluid contents of the cavity rise above the level of the permeable bronchi which lead into the cavity, large irregular bubbling râles or distinct gurgling may be heard. Well-marked pectoriloquy (from a mere whisper) is the form of vocal reasonance most pathognomonic of pulmonary tuberculosis (FULLER, l. c., pp. 363 to 370).

(c.) *Acute Miliary Tuberculosis, or Primary Tubercular Phthisis.*

This affection, from its rapid course, has also been named acute pulmonary consumption. It is expressed by febrile symptoms running an extremely rapid course, denoting the severity of the constitutional disturbance, proving fatal in from twenty days to ten or twelve weeks; and due to (*a.*) acute miliary tuberculosis, or (*b.*) acute pneumonic phthisis, or to both combined.

Acute miliary tuberculosis has usually been considered to be a comparatively rare disease; but it would appear that this fatal affection is unusually prevalent among the soldiers in Paris, among whom also the chronic form of phthisis is very common, probably more so than even in our own army. In 1861, M. Colin (Professeur agrégé au Val-de-Grâce) records *five* cases of *acute tuberculosis* in the soldiers of the garrison of Paris (Parkes, *Army Med. Dep. San. Report*, 1860, p. 357).

The disease occurs in two forms:—(1.) One form is connected with extensive infiltration of pneumonic material in the lungs, with irregular softenings in the centres of some, or with small excavations surrounded by patches of fresh hepatization. (2.) The form most characteristic of, or most commonly found in, acute phthisis is that in which there is general studding of both lungs with semi-transparent grey granulations, combined with pneumonia in its first stage, bright arterial injection, or hepatization (WALSHE). The two forms may co-exist in the same lung.

The Symptoms are those of an intense febrile affection. Dr. Parkes has favoured me with the notes of a case exhibiting the correlation of the temperature, the pulse, and the respiration during the course of a fatal case of acute phthisis. The records of temperature are as in the following diagram, fig. 53:—

DIAGRAM SHOWING THE RECORDS OF TEMPERATURE IN A CASE OF ACUTE PHTHISIS (Parkes).

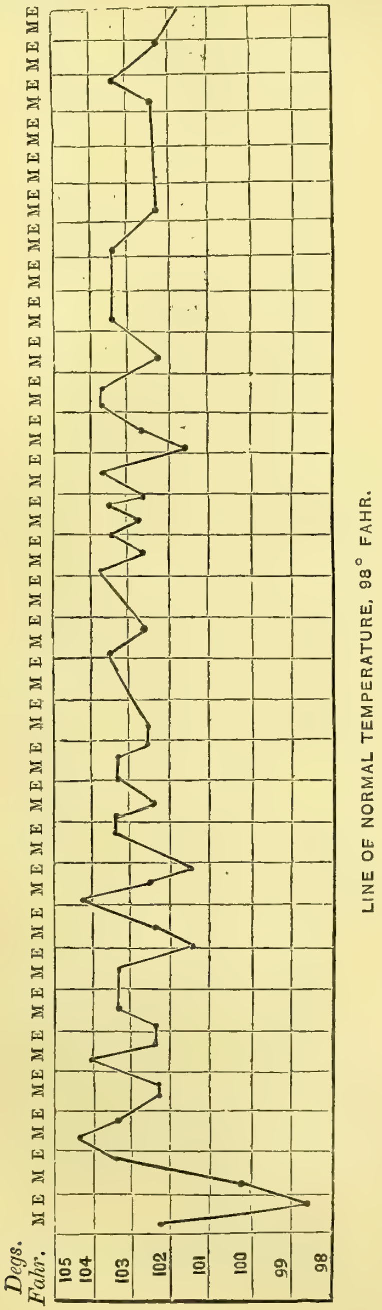

Fig. 53.

During *thirty* days the pulse ranged from 116, the lowest, to 178, the highest, and having a mean of 140, reckoned by the number of observations. The respirations *per minute* ranged from 36 to 60, having a mean of very nearly 50. In Dr. Walshe's cases the relationship of the pulse to the respiration has varied considerably. The average has been as 3 to 1.

The functions of the lungs are deeply impaired. The invasion appears to occur while the patient is in a state of health, or the fever may be remotely preceded by various depressing influences, and immediate exposure to cold and wet; after which rigors ensue, followed by acrid heat of skin. The rigors recur on several successive days, followed by perspirations, and sometimes crops of sudamina. Epistaxis, followed by coryza, may occur on the second day of seizure (Walshe). Prostration sets in early, so that in a few days the patient may be unable to stand. There is thirst, total anorexia, epigastric tenderness, dry lips and tongue, dental sordes—all signifying great intestinal disturbance. Diarrhœa is rare, and constipation may be extreme, even with abdominal pain and ulcerated intestines. Restlessness, *insomnia, cephalalgia, vertigo, tinnitus aurium,* diurnal wandering and nocturnal delirium bespeak cerebral complications and the probable growth of tubercles in the arachnoid membrane. The physical signs vary with the amount of tubercular growth or *pneumonic infiltration* in the lung, and are similar to those which have been described.

The following are some of the different modes in which phthisis pulmonalis makes its approach:—

1. There is sometimes to be noticed a latent, masked, or occult form, in which prodromal catarrh is absent—the real condition of the patient not being detected till the lungs are tuberculous to a considerable extent, unless recourse be had to careful thermometric observation, as described in the first volume, and as already often referred to here. Although the general symptoms may be slight, this very fact ought at once to excite suspicion, especially when the general symptoms denote considerable elevation of temperature with rapid consumption of the body. The slight cough, the shortness of breathing, the frequent pulse, increased on the slightest exertion, the languor of the frame, and general chilliness of the body with persistent increase of temperature, morning perspirations, and progressive emaciation, betray the insidious way in which this form of consumption commences. Such a case must be closely watched; for the local symptoms often remain obscure, and friends do not always see the importance of the illness till too late. It is most usually young persons between sixteen, eighteen, and twenty-five years of age who are the victims. They begin to lose flesh, becoming feeble, pale, and thin, and are attacked with a short tickling cough, often regularly excited by undressing at bedtime, and again on getting out of bed in the morning. Such a cough is dry, or followed by only a small quantity of mucus. The cheeks are the seat of hot, uneasy flushings, while the feet are cold, and towards morning there is generally a little more moisture than usual on the surface of the body, especially about the head, neck, and breast. The pulse is either considerably quicker than usual, varying from 90 to 110 or 120; or if it is natural, it is very readily accelerated. The respiration is in general more

frequent in a given time—usually from 24 to 28 in a minute—the inspiration being generally short, limited, and speedily checked, quickly succeeded by expiration; and the patient cannot take a full or deep inspiration without uneasiness, and without inducing coughing. The tone of the voice also becomes deep (CRAIGIE).

2. There is another form of consumption, attended with a severe and sudden accession of febrile disturbance (to be measured by the thermometer), occurring in persons of a scrofulous diathesis. To this form Sir James Clark gave the name of "*febrile consumption.*" Pulmonary symptoms are not generally manifest in such cases in the first instance; but what is commonly called biliousness more frequently prevails, or the case may present the symptoms of a *common catarrh.* Cough, however, generally soon appears, becomes urgent, and occurs often; and hurried breathing is one of the most remarkable symptoms. The cough speedily becomes more frequent, accompanied by expectoration, at first colourless, or of a thin transparent bluish jelly-like appearance; it subsequently assumes a yellowish or greenish hue, thick, opaque, dense, and puriform, and is occasionally streaked with blood. Fever continues unabated, and is out of all proportion to the other symptoms of pulmonary affection; and thus the true character of the disease may be overlooked. The pulse is seldom under 100, varying from 110 to 116, with some tension and sharpness in the beat. Gradually the fever assumes the hectic form, progressive wasting is established, and doubt can no longer be entertained regarding the nature of the case and the fatal issue. (See range of temperature in "Acute Tuberculosis.")

3. In another description of cases, repeated hæmoptyses are the first symptoms that attract attention and alarm the patient and his friends; which are followed by all those phenomena already noticed.

The cases of phthisis more usually rapid are such as arise from hereditary constitutional causes, or from the influence of exanthemata, especially measles, or of typhoid or other fevers. These run their course with implication of several organs at an early stage of the disease. In the more chronic forms of phthisis the lung in adults is the first seat of the disease, and other organs are secondarily affected.

Causes.—In appreciating the causes of phthisis several forms of pulmonary lesions ought to be distinguished which have been here described; and in the development of consumption or pulmonary phthisis in man, it will be found that there are three distinct agencies at work (BURDON-SANDERSON):—

(1). *An ill-defined agency, to which the name of constitutional tendency has been given.* It is exemplified in the fact of pulmonary phthisis so frequently attacking in succession several members of a large family shortly after adult life.

The question as to the evidence of *tubercle* being hereditary, in contradistinction to pulmonary phthisis, has also undergone some modification. Niemeyer considers the evidence uncertain, to this extent—that it would be necessary to show that one parent was *really* tuberculous at the time of conception, and that the child became so without having, or having had, any other disease or lesion capable of producing it.

Even tuberculosis of the meninges can generally be shown to be secondary to caseous formation elsewhere, especially in the bronchial glands.

An hereditary predisposition to *pulmonary phthisis* is not only, however, certain but frequent; and is chiefly due to congenital or inherent weakness and vulnerability of the constitution. Such vulnerability is evidenced by repeated illnesses during early life.

That the tuberculous constitution is actually transmitted from parent to child has long been a popular belief, and regarded as one of the best established points in the etiology of the disease. Actual *proof*, writes Dr. Walshe, has never yet been afforded of the justness of the general conviction; and as a step towards an *accurate* settlement of the question, Dr. Walshe analyzed and recorded (1849) the family history of 102 phthisical patients admitted into the Brompton Hospital for consumption. From these records it appears that about 26 *per cent.* of phthisical subjects in a given generation come of a tuberculous parent—a circumstance which may be predicated of any mass of individuals taken in hospital —namely, that 26 *per cent.* of them are of phthisical parents. On the other hand, while the general statement may be made that some cases of phthisis may be traced to hereditary influence, it is undoubted that *much phthisis is, in each generation, non-hereditary.*

It further appears that in *males* the malady exhibits itself at a mean period of about two years earlier, and in females at a mean period of about three years and a half earlier when there is a parental taint than when there is not. Phthisical persons spring from a phthisical source with a certain amount of frequency; and that freedom from taint in parentage is *probably* more rare, and the existence of such taint *probably* more common in phthisical than in non-phthisical patients; but it is possible that if investigation was extended to infancy, childhood, and youth, the ratio of cases of parental taint among the phthisical would be proportionably greater than it proves where inquiry is limited to adults.

It appears, also, that while about 9 *per cent.* more phthisical than non-phthisical persons come of a consumptive father or mother, on the other hand there are about 10 *per cent.* more phthisical than non-phthisical persons free from parental taint.

The final conclusion which Dr. Walshe arrives at, after a most careful and logical analysis of 446 cases of phthisical and non-phthisical cases, is this—That *phthisis in the adult hospital population of this country is, to a slight amount only, a disease demonstrably derived from parents;* and there is no reason to believe that the law differs among the middle and higher classes of society. But amongst the phthisical cases which form the subject of Dr. Walshe's inquiry, we have the tuberculous cachexia communicated, both as regards the parents and the generation following; but there is still a class of cases to be inquired into in a similar manner —namely, such as will show how far parents labouring under *scrofulous cachexia* merely entail on their offspring *a disposition* to tuberculous affections. It is now a well-known fact, emphatically insisted upon by Sir James Clark, that in the families of consumptive parents there are constantly to be met with instances of ill-health characteristic of the scrofulous constitution; and in general such instances are much more frequent and much more strongly marked in the younger than in the elder children; nay, there are families in which the elder children are healthy, and the younger ultimately become the subjects of tuberculous disease. In such cases it has been presumed that in some instances the health of the parents has become deteriorated during the increase of their family. The mere fact of the parents being unhealthy, and not necessarily tuberculous, appears in some instances as if sufficient to entail tuberculous diseases upon their children. This statement is in some measure borne out by a result obtained by Dr. Walshe, that as far as the mere phthisical or non-phthisical condition of parents is concerned, about 24 *per cent.* of tuberculous patients can trace the origin of their disease to either parental source.

(2.) *The agency of irritation.* The organs liable to the destructive changes of phthisis are, above all, the mucous cavities which communicate with the external air. A common bronchial catarrh, not differing in any respect except in its result from other bronchial catarrhs which terminate favourably, gradually assumes the character of consumption. Sometimes an obvious source of irritation is to be found in the nature of the work done by those who suffer and die of pulmonary consumption—as in the striking demonstration afforded by an observation long ago recorded by Dr. Home. In a stone mason who died of phthisis an earthy nucleus was found at the centre of many of the pulmonary condensations. This earthy nucleus turned out to be of precisely the same character as the stone of Craigleith quarry, where the man had been employed (Home, *Edin. Med. and Surg. Journal*, January, 1838).

Among townsmen, also, it is determined that there are certain classes of men more predisposed to phthisis than others by the irritant nature of their occupations. It has been observed to occur, for instance, in those workmen who suffer great vicissitudes of temperature, or who breathe an air loaded with particles of dust: as bakers, needle grinders, stone masons, quarrymen, cotton and wool carders, and bricklayers' labourers; and in this class of persons the disease has acquired the epithet of the "grinders' rot." As far as regards their state, much has been founded upon it to show how irritant substances may induce the local lesions. Professor Alfonso Corradi, of Venice, who wrote an essay in 1870 on the spread of pulmonary phthisis in Italian towns, mentions that the stone masons who have to work at granite near the *Lago di Garda*, nearly all of them die towards fifty years of age; and the same is true regarding those who work in slate in the caverns near Chiavari, and who suffer from the *Mal de Chiapperolo.* Those who do the same work in the open air are but little subject to the disorder; neither is the dust found in their lungs.

"Dust is undoubtedly a most deleterious agent, to which certain artizans are exposed, and would appear, from the investigations of Greenhow, Alison, Hannover, and others, to be a fertile cause of phthisis among those who are engaged in hackling flax, carding cotton, grinding steel; also workers in porcelain, makers of mother-of-pearl buttons and of matresses, chaff-cutters, stone masons, and saddlers, furriers, glovers (HANNOVER) who inhale the dust from animal tissues, and coal-miners.

Dr. Greenhow shows that the potter's consumption is chiefly caused by portions of clay being allowed to be trampled into dust on the floors of the work-rooms. Few men who enter certain rooms in cotton factories ever live to thirty-eight years; and out of twenty-seven men in a flax factory, twenty-three had pulmonary disease (GREENHOW). Dr. Alison asserted that in Edinburgh a stone mason scarcely ever reached fifty years without becoming consumptive. The noxious influence of varnishes, turpentine, and drying oils, in developing phthisis, was long ago pointed out by Lombard (*Annales de Hygiéne*, t. xi., 1834), and it is said to be very frequent among artizans who use solder, such as tinmen, coppersmiths, and goldsmiths" (CLYMER).

Similar instances of mechanical irritation are to be found in miners. It is in them that the form of *fibroid phthisis*, in which a chronic interstitial pneumonia leads to fibroid deposits with cheesy degeneration (tyrosis) of imprisoned portions of lung, and which constitutes one of the forms of pulmonary phthisis to which Dr. Andrew Clark has drawn especial attention—a form which he also connects and finds associated with rheumatic inflammation of interlobular tissue, chronic pleurisy, and such constitutional lesions as granular kidney and liver.

In the cases in which destructive processes ending in tyrosis originate from catarrhal inflammation of the genito-urinary organs, the same thing happens from irritation. Gonorrhœa leads to prostatitis, prostatitis to scrofulous catarrh of the bladder, which creeps upwards along one or both uretus, and produces induration or caseation of one kidney—that condition which is called *renal phthisis.*

Thus, alike in the lung and in the kidney, an indurative process, the result of inflammation, which ultimately becomes disintegrative (tyromatous)—*i.e.*, a tuberculosis—begins from a simple or specific catarrh of irritative origin.

(3.) *The agency of infection;* which means that whenever a chronic induration, due to over-corpusculation (hyperplasia), exists in any organ, it is apt to give rise to similar processes elsewhere. Thus infection has to do with the development of ordinary consumption—pulmonary phthisis—and independent of the deposit of true miliary tubercle, so that infiltrated forms of induration result, as well as true miliary tubercle, through an infective process or agency. Buhl showed this more than twelve years ago, in regard to miliary tubercle, to which he limited his statements. He showed that in persons who die of that variety of fever characterized by the general dissemination throughout the body of miliary granulation (*acute miliary tuberculosis*), masses of induration which have remained long enough in the body to become caseous, are in the great majority of cases to be found; and he also showed that in those cases in which miliary tubercles are sown over a much more limited region—as, *e.g.*, when they are confined to a single organ—they also spring from old lesions.

The results of the inoculation experiments of Sanderson, Fox, and others, already named, now extend the signification of the infective process to extensive indurations, as well as to miliary deposits; since by inoculation we obtain miliary granulation in the serous membranes and in the choroid, but interstitial and diffused lesions in the liver, lungs, and other massive organs.

To those agencies Niemeyer would also add—

(4.) *The agency of extreme bodily exertion,* which so disturbs the circulation and the influence of hæmoptysis—itself an evidence of an abnormal and disturbed condition of the bronchial circulation. Blood remaining in the bronchi has long been held—since the time of Hippocrates—as a frequent cause of *pulmonary phthisis.* It is followed by a more or less decided irritation of the lung and pleura—*consecutive pleuro-pneumonia*—in which, on the second or third day after hæmorrhage, an increased frequency of the pulse occurs, with a heightened temperature and general *malaise,* more or less severe lancinating pain in the lateral regions of the chest, and frequently fine bubbling rhonchi friction sounds, or a slight percussive dulness, with weakened vesicular murmur, or bronchial breathing. Such lesions may end in resolution, or gradually go on to cheesy degeneration (*tyrosis*), and perhaps the deposit of tubercle, as the direct result of absorption of the decomposed blood (*Phthisis ab hœmoptöe*).

With regard to climate or locality in the temperate zone, where the civilized inhabitants of the globe are located, it is calculated that *one-tenth* of the population die of this malady. In the mining districts of Cornwall and Devonshire, although those counties are considered among the most healthy portions of Great Britain, yet one-half of the whole number of the miners deprived of fresh air and light die of phthisis. In connection with this statement a most significant fact, of great practical importance, has been brought to light by Dr. Walshe. He has shown that improvement in cases of pulmonary phthisis was effected, by medical treatment in hospitals, in about 14 per 100 more frequently in persons following "open air" and "medium" occupations than in those whose trades were "confined;" but that *death* or *deterioration* was as frequent as improvement in those who followed confined trades. Minute analysis, such as that which has been instituted by Dr. Walshe, of the numbers that die of phthisis in the different ranks and classes of life, is greatly to be desired in illustration of the remote causes of phthisis. The late Professor Coleman was of opinion that by confining the horse in a dark and dirty stable, and by feeding him on bad provender, and neglecting to clean him, he could produce phthisis in that animal at will; and similar causes will probably be found to produce similar results in man. When, however, we consider how many persons there are who carry cleanliness to excess, whose diet is most studied, and whose every exercise is directed to health, and who nevertheless die of phthisis, it is plain that more secret and hidden circumstances still remain to be discovered to account for the existence of pulmonary phthisis in this country. The Reports of the Registrar-General show that, comparing the deaths from phthisis among the agriculturists and among the inhabitants of towns, the latter die in an increased ratio of 25 *per cent.* over the former; yet it is generally supposed that the dietary and general comforts of the townsman are greater than those of the countryman. The chances of improvement are 5 *per cent.* greater in persons who come from the country to a salubriously situated hospital in town than in townspeople (WALSHE).

The mortality numerically from consumption is much higher than from any other disease in this country; and amongst the class of society resorting to assurance offices the mortality seems to be about one-half of the whole.

The disease appears to be still more fatal to soldiers than civilians, producing nearly *one-half* of the whole mortality among the dragoon guards; while in the foot guards it has hitherto been nearly double of what takes place in the dragoon guards in this country. The habits of a soldier's life, his liability to febrile and inflammatory attacks, and the frequency with which he contracts venereal diseases, have hitherto been very favourable to the induction of the more aggravated forms of pulmonary indurations and phthisis. The history of phthisis in armies will at once show how materially the prevalence of such a disease influences the health, the wealth, and the military strength of a nation. In Prussia, phthisis caused 27 *per cent.* of the total mortality; in Austria, 25 *per cent.;* in France, 22·9; in Hanover, 39·4; in Belgium, 30; in Portugal, 22 *per cent.* In all these armies the same causes are in action, and the predominance of the disease is mainly to be sought for in the impure barrack air (Parkes's *Practical Hygiene*, p. 492). Some are inclined to ascribe this excessive amount of pulmonary disease and mortality to the night duties of the soldier—a statement in some measure supported by the large amount of mortality from consumption amongst night-watchmen generally; but when one looks to the age and height of the men enlisted for the regiments of the guards, and compares them with the physiological records regarding the stature and growth of the human frame, it will be seen how, sometimes, the combination of requirements for enlistment in the regiments of guards have been little calculated to secure a hardy and efficient body of men.* They are constitutionally vulnerable to an extreme degree. During 1859, 1860, and 1861, it appears that the mortality in the army hospitals from phthisis was not above that of the country generally, although it is clearly above that of the healthy districts; and there can be no doubt that there still is an excessive prevalence of tubercular disease in the army; and the astonishing disproportionate number of cases in the foot guards (18 to 20·6 per 1,000 of strength) is still as remarkable as it was twenty years ago (PARKES, l. c., p. 495). The tables prepared by Dr. Parkes, and given in his most valuable work on *Practical Hygiene*, clearly show that there must be a large amount of phthisis *generated* in the army; and in the foot guards nearly *four* times as much as among the civil male population of twenty-five to forty-five years of age.

In the American army the ratio varied (1861 to 1863) from 8 to 9 per 1,000, and the deaths from 1 in every 2·7 to 1 in 4·5 cases. In the British army, during 1861-62, the cases of phthisis were from 9 to 10 per 1,000 of mean strength; and the deaths 1 in 3. But neither in the American nor in the British army is it shown how many soldiers are annually invalided on account of pulmonary consumption, the greater number of whom die after their discharge, and swell the mortuary return of both countries (Clymer). The deaths from phthisis in the British navy during three years averaged 2·6 per 1,000 of strength, and the invaliding 3·9 per 1,000 (Milroy).

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* On this subject the reader is referred to a little book, by the Author, *On the Growth of the Recruit and Young Soldier*. Charles Griffin & Co., London.

The Science of Medicine is not unfrequently indebted to non-professional people for correcting prevailing errors of belief and establishing correct opinions. No one, perhaps, contributed more in this direction, in the discharge of his own professional duties, than the late Sir Alexander Tulloch. It was long a prevalent belief that consumption was limited by latitude, and that it never appeared in warm countries—for instance, south of the Mediterranean. But this is proved not to be the case; for the returns of the army, prepared by the above writer, have shown that phthisis is more frequent in the West Indies than even in this country—a statement first made by Sir James Clark, in his work *On Climate*, in illustration of the injurious effects of that climate on consumptive patients sent there from this country. According also to the recorded opinion of this author, great heat appears to have a powerful effect in predisposing to tuberculous diseases (probably by diminishing the exercise in the open air). That it is not the climate of the place which alone produces this result in the West Indies is shown by the fact that officers were attacked in infinitely smaller proportions than private soldiers; and in consonance with the views entertained regarding the nature of tuberculosis, it is more than probable that crowded barrack-rooms, a restriction to salt diet, and drinking spirits, may have produced the result.

It would appear that England and Wales, the Cape of Good Hope, and the Ionian Islands, are more exempt from phthisis than many countries which, from their higher temperature, have hitherto been supposed to enjoy a remarkable exemption from this complaint. The result of extended observation now entirely refutes the hypothesis that paludal districts are in an eminent degree exempted from phthisis—an opinion first promulgated by the late Dr. Wells, and advocated by M. Boudin. England and Wales, the Cape of Good Hope, Canada, and Malta—countries either the driest or the best drained, and consequently suffering the least from paludal diseases—are actually those countries the most free from phthisis. On the other hand, the influence of climate shows that phthisis is most frequent in low and damp situations; while it is far less so in the mountainous districts of all countries. Again, in whatever climate the disease breaks out, it is the opinion of many pathologists that its course is most rapid if the patient remains in that country; and therefore it is of the utmost importance to know the physical nature of the various climates of the world most suitable for the tuberculous patient. (See Appendix to the third edition of Dr. Walshe's work, *On Diseases of the Lungs;* and the late Sir James Clark's classic work *On Climate.*) The late Dr. Hennen's experience convinced him, when the disease broke out among our troops on the shores of the Mediterranean, that no other chance remained of prolonging the patient's life than by at once sending him back to this country.

Contagion of Phthisis.—Regarding the possible contagious propagation of pulmonary phthisis, Dr. Parkes thus expresses himself (having regard to the fact that purulent and epithelial cells have now been demonstrated as floating about in the air where numbers of persons are together):—"Considering that the pleuro-pneumonia of cattle is probably propagated through the pus and epithelium cells of the sputa passing into the air-cells of other cattle; that even in man there is some evidence of a pneumonic phthisical disease being contagious (Bryson, *Cases in Mediterranean Fleet*), the floating of these cells in the air is worthy of all attention. It may explain some of those curious instances of phthisis being apparently communicated" (*Prac. Hygiene*, p. 74).

Dr. Julius Petersen, District Physician in Copenhagen, has recently (1869) shown that a belief in the contagiousness of phthisis has a very ancient historical foundation; and he believes that the discrepancies existing in the views of European physicians on the subject is connected with well-marked climatic differences. In Southern Europe, the views in its favour decidedly preponderate; in France opinions are more divided; in the extreme North an anti-contagious theory prevails. Although unable to produce any absolute proof of the existence of contagion, he sums up the result of his inquiries as follows:—

(1.) That a contagious origin of some cases of phthisis cannot on sufficient grounds be denied; (2.) That phthisis caused by contagion is in general of a very dangerous and inflammatory character; that it must justly be considered hazardous to sleep in the non-disinfected bed of a phthisical patient, and to be habitually in too close contact with such a person; that this danger in Denmark seems to be greatest in the warm period of the year.

Dr. Clymer gives the following excellent summary of arguments for and against the doctrine:—

"Morgagni said *phthisicorum cadavera fugi adolescens, fugio etiam senex.* In Italy consumption has been, and still is, looked upon as a communicable disorder; a consumptive is shunned, and the vessels he may use in eating and drinking are avoided or destroyed, and his clothes burnt or buried. The opinion that long and continuous exposure to the body-effluvia of a tuberculotic patient puts a previously non-tuberculous person to the risk of the disease, by a predisposition to it, has been held by Jos. Frank, Laennec, Sir James Clark, and others. Andral went so far as to say that under certain conditions these tubercular emanations become a source of true contagion. Most medical practitioners of long experience have seen examples of the apparent contagion of consumption—a tuberculous husband infecting a wife, and the reverse. The late Dr. Leger lately communicated to Dr. Villemin some curious facts of this kind (*Gaz. Hébdomadaire,* 1868). That the tuberculous diathesis may be transmitted from the male to the female by the medium of the fœtus, and this even in such a way that children begotten by a second non-tuberculous husband may inherit the diathesis acquired from the first or tuberculous husband, would seem likely, from the many cases reported by Dr. Perroud of Bourdeaux (*De la Tuberculose,* &c., Paris, 1866), and by Dr. Alexander Harvey, of Aberdeen (*Edin. Medical Journal,* 1849, 1850, 1854). Although Sir Thomas Watson explicitly states that he does not believe phthisis to be contagious, he adds:—'Nevertheless I should, for obvious reasons, dissuade the occupation of the same bed, or even of the same sleeping apartment, by two persons, one of whom was known to labour under pulmonary consumption' (*Lectures on the Practice of Physic*). Dr. Fuller says—'But, though the non-infectious character of phthisis be admitted, it behooves the physician to warn the patient's friends of the dangers incident to long-continued attendance on him, especially if the disease be in an advanced stage. It would be the height of imprudence for a healthy person, and especially if young and of a scrofulous diathesis, to sleep in the same bed, or even in the same apartment, with a consumptive patient; for although the malady might not be communicated directly from one to the other, unless possibly under the condition of some tubercular matter being accidentally introduced into his air-passages or into some other part of his system, the surroundings and the air would be calculated to predispose him to the disease' (*On Diseases of the Lungs and Air-passages*, p. 431). Dr. Villemin suggests that besides the direct transmission, as by cohabitation, consumption may be contracted through indirect means, by clothes, bed-linen, water-closets, the vitiated air of rooms lived in by tuberculous persons, &c. The possible transmissibility of the disease in this manner merits, he thinks, the attention of medical officers of the army. A tuberculotic soldier dies in the hospital, and his clothes are returned to his company and worn by another; may not this, he asks, be one source of phthisis in the army? He is satisfied that the barrack is to the soldier in the production of consumption what the regimental stable is to the horse in the development of farcy, the contagion and transmissibility of which are at length accepted. Fournet, who in his work is a non-contagionist, still gives some weight to the possibility of infection from an atmosphere constantly breathed, and necessarily poisoned, by the consumptive."

"In the recent protracted and unfruitful debates upon phthisis in the French Academy of Medicine, the subject was fully discussed; the views of two of those to whose opinion we are disposed to attach value will be quoted. Dr. Jules Guérin believed that crude tubercle can never be contagious; but that when it is softened, and the ulcerated lung-surfaces are exposed to the air, the patient may become a source of infection to those about him, just as the pulmonary lesions he has may infect his own organism by the resorption of purulent and putrid products. Dr. Bouillaud's ideas would seem to be nearly the same. He said,—'During the course of pulmonary tuberculosis, when pus or other septic products are formed in parts which are accessible to the atmosphere, phthisis, like so many other affections in which similar purulent foci happen, becomes indirectly a cause of septic infection.'"

Treatment.—The prevention of pulmonary phthisis is only to be looked for in the unrestricted enjoyment of the open air. Continuous in-door labour is to be avoided; and measures to prevent bronchial catarrhs are to be taken, especially by sufficiently warm clothing and the constant use of flannel next the skin, in this country.

If, unfortunately, a pulmonary catarrh does not abate, and a pneumonia does not resolve itself within the usual period, but catarrhal symptoms prevail in the apices, and pyrexia is persistent, with the severe disturbance of the general health which has been described, everything must be done to protect the patient from injurious influences. Further extension of the pneumonic process must be prevented by absolute rest in bed for a time, talking being forbidden, and coughing as much as possible repressed; the chest to be covered with a poultice, and local bleeding by leeches, if pleuritic pains exist. Whenever considerable pyrexia exists in the evening, all duty occupations should be given up, strict rest, equable temperature, and application of poultices to the chest adopted, and moderate action of the skin maintained.

Should the pyrexia still continue considerable, *digitalis* and *quinine* are the remedies most successful in reducing temperature. The fever is the symptom which most of all demands treatment. Heim's pill is much employed by Niemeyer, as it is in Germany, for this purpose. Its main ingredient is *digitalis*, and its composition as follows:—

℞. Pulv. herb. Digitalis, gr. x.; Pulv. rad. Ipecac.; Pulv. Opii puri. *a a.*, gr. v.; Ext. Gentiani q. s.; misce et divide in pil. xx. *One pill three times a day.* It is of advantage that *quinine* to the extent of gr. xx. be added to this. Each pill will then contain *half a grain of digitalis; one quarter grain of opium; one quarter grain of ipecacuanha;* and *one grain of quinine;* and which may be held in form by *glycerine* or *syrup.*

The general principles of treatment having been already laid down under "Scrofula," p. 30, it only remains here to warn the student (as Dr. T. K. Chambers has so ably done in his Lectures, p. 270) against the use of so-called "cough-medicines," *antimony, ipecacuanha*, and *squill* especially; also against the use of *mercury, purgatives*, and *neutral salts.*

To foster and cherish an appetite for food must be the great aim of treatment. One of the best tonics, if pyrexia does not exist, is the syrup of *iron, quinia*, and *strychnia*—of which the formula by the late Professor Easton, of Glasgow, has been given at p. 86, *ante.* Let it be given in very small doses at first; and followed up by the use of cod-liver oil "of the most agreeable, clearest, sweetest, and most scentless kind." The *brown oil* ought never to be prescribed, except, perhaps, to an Esquimaux. The oil should be given in tea-spoonful doses at first. Phthisical patients require the richest diet that will agree with them.

Dr. B. W. Foster, of Birmingham, has kindly drawn my attention to the advantages of *æther* in promoting the digestion and absorption of fatty food, and especially of cod-liver oil. A stomach once intolerant of all fat will good-naturedly accept full doses of cod-liver oil after the use of æther. Sometimes it has been given separately, before or after the oil; but Dr. Foster now prefers to give it combined with the oil—a mixture now generally known as "*Ætherized cod-liver oil.*" To every two drachms of the oil, ten, fifteen, or twenty minims of æther are to be added, according to the wants of the case. The *Æther purus of the British Pharmacopœa* must be used, so that the oil may not be rendered muddy, as it will be if the æther contains alcohol or water. The ordinary dose of oil ought not to exceed two drachms, and to which more than twenty drops of æther may be added, if necessary. The æther is indicated wherever there is inability to digest fat. It makes an emulsion of the fat; and fat when emulsified is more easily digested than in the other form. It seems also to determine, when introduced into the stomach, a considerable flow of pancreatic juice, as Claude Bernard demonstrated, whose experiments led Dr. Foster to propose its use as an aid to cod-liver oil in the treatment of *pulmonary phthisis.* (See *Brit. Med. Journal*, Nov. 21st and 28th, 1868; also, *Medical Press and Circular*, 1869.)

A pint of milk, if possible, "*warm from the cow*," ought to be taken as often as possible. It is so prescribed by Niemeyer, in order that the cream may not be removed by skimming, but the entire milk obtained.

Extract of malt (TROUSSEAU) is also a remedy of recent repute. It is a genuine extract, resembling other officinal extracts, consisting of the soluble constituents of the malt and of the bitter extractive matter of the hop. Two or three table-spoonfuls of it are to be taken daily, diluted in spring water or in warm milk, or in any other liquid that may be desired. There should be a careful choice of meats and drinks. Meats rich in *osmazone* and roasted are to be preferred. Full-bodied wines and malt liquors are better than mere alcoholic fluids. Broth made of coarsely brown rye meal, beef, mutton, or other flesh meat, boiled to make a soup, is also of service as a diet. I have found the following soup also very serviceable:—Take of *linseed* half an ounce; *fine bran*, one ounce; *water*, one quart. Boil these for two hours, and strain; then add beef, mutton, or any other meat that may be fancied, to the amount of one pound, and boil to a soup, with vegetables, to which celery seed or other flavouring may also be added. The whole quantity ought to be reduced one-third.

Dr. Clymer, after many years' experience, has come to regard *arsenic* as an efficient improver of the general nutritive and assimilative functions. He gives *one* or *two* drops of Fowler's solution, or $\frac{1}{50}$th of a grain of arsenious acid, once, twice, or thrice a day, *during the meal*, continuing it for months, with occasional intermissions. Decided benefit sometimes follows the use of *iodide of potassium*, which may be combined with the *ammonio-citrate of iron*, or *tincture of the muriate*, in a bitter infusion like *columba* or *cascarrilla.*

To allay cough, *opium* in its many forms is the best medicine, when absolutely indispensable; but its use should be deferred as long as possible.

Hard rubbing the skin is an excellent tonic; and for the relief of the many local conditions of discomfort, pain, and distress for which the consumptive patient most frequently applies to the physician, Dr. Clymer advises as follows:—"The most common are cough, stitch-pains, and sweating. For the first, the so-called nauseating expectorants, so generally prescribed, do harm by deranging digestion, which it is so important to keep whole. It arises from so many causes that no general rules for its management can be laid down. If there is an elongated uvula, the end should be snipped off; if there is irritation about the pharynx and upper part of the larynx, it may be relieved by the inhalation of some of the atomized fluids; cough is often much under the will of the patient, and may be controlled by an effort; opium is to be avoided when possible, and the *bromide of potassium, tincture of cimicifuga,* and *prussic acid,* first tried. Of the preparations of opium, *codeia* is probably the least harmful in this complaint; it may be given in combination with *prussic acid.*

"For the local pulmonary congestions, dry cupping and derivatives may be used, though the tendency to them is best hindered by restoring the skin-function and equalizing the circulation. The attacks of intercurrent bronchitis and pneumonia may be treated with *muriate of ammonia;* and in the more chronic and asthenic forms some patients have found great relief from the wine of tar: it is of value when it is borne by the stomach. Counter-irritation to the chest-wall is a stereotype method of cure in this condition, and when properly used in the earlier stages, before there is much loss of strength, it is undoubtedly beneficial; but later it is weakening and annoying. Croton oil liniment is the chief favourite; but a prompt and not too severe application is the following ointment, recommended by Dr. Fuller:—

"℞. Hydrarg. Chlor. Mit., gr. viij.; Iodinii, ʒss.; Alcohol, fʒiss.; Unguent. Simp., ℥j. M. Rub in a portion over the affected lung morning and evening until a pustular eruption comes out.

"A solution of the nitrate of silver (thirty grains to the ounce of distilled water) may be painted on the skin beneath the clavicles every evening until the skin is darkened, and repeated after the cuticle peels off. Gentle and continuous irritation of the skin may be kept up by wearing constantly on the chest a piece of flannel wet with a weak solution of iodine in glycerine and water, covering the cloth with oilskin, to prevent too rapid evaporation and soiling of the clothes.

"The night-sweats, so often annoying and profuse, diminish under general systemic improvement and restored cutaneous functions. Bathing with vinegar and water, alum and water, or diluted alcoholic liquors, are well-known remedies, as well as gallic and tannic acids, nitrate and oxide of silver, and oxide of zinc, and infusion of common garden sage.

"Little can be done to check or even relieve the diarrhœa when it once sets in. A flannel bandage around the abdomen should be worn. The traditional treatment is by bismuth, astringents, and opiates. When it is possible, let the stomach be spared, and drugs be given in enemata."

SECTION VI.—DISEASES OF THE PLEURA.

PLEURISY.

LATIN EQ., *Pleuritis;* FRENCH EQ., *Pleurésie;* GERMAN EQ., *Pleuritis*—Syn., *Rippenfellent-zündung;* ITALIAN EQ., *Pleuritide.*

Definition.—*Inflammation of the serous membrane that lines the cavity* *and covers the viscera of the thorax. It is characterized at its outset by a febrile chill, followed by an acute sharp pain in some part of the chest, frequently called* "a stitch in the side," *as it is usually confined to one spot about the lateral regions of the thorax. The acts of respiration are performed rapidly, and are not completed. A dry short cough supervenes, and the pulse is hard and quick. The natural serous secretion of the pleural sac is arrested in the first instance, but soon becomes increased in quantity, and of an inflammatory type, the exudation having a great tendency to assume the* corpuscular *character, when the effusion more or less rapidly increases, and may ultimately assume a sero-purulent character, the parietes of the corresponding side of the chest being dilated accordingly.*

Pathology and Morbid Anatomy.—The inflammatory phenomena begin in the sub-pleural tissue, whose vessels enlarge and admit red blood, and shortly afterwards the red blood penetrates the web of the pleura itself, and the process is more or less diffuse. At first a number of red dots may be visible, which at length are so multiplied as to become confluent, and form large patches, which spread till perhaps the whole of the *pleura pulmonalis and costalis* is in one continuous state of inflammation. The membrane is in all cases of a bright red or arterial colour, slightly thickened and swollen from interstitial infiltration. The epithelium is easily detached, or already cast off.

If the diffuse inflammation be of any intensity, the secretion from its surface is in general suspended at first, and the membrane is dry, with its free surface rough, from inflammatory products and granulations, consisting of newly formed fusiform cells, filaments of connective tissue, and the whole supplied with elongated delicate capillary vessels, coiled up into loops within the new growths (FOERSTER). In this state the inflammation may terminate by adhesion, or by resolution, or fluid effusion may be poured out.

Thus, with pleurisy as with pericarditis, there are at least two distinctive forms, namely—(*a.*) Where the pleura thickens, and the material of inflammation is of the adhesive kind; (*b.*) Where the pleura also thickens, but the material of exudations, after pushing off the epithelium, continues to produce corpuscular forms by interstitial exudation, and to the accumulation of fluid within the cavity of the pleural sac. (For a detail of the "Minute Morbid Anatomy," the reader is referred back to p. 550.)

The quantity of fluid thus effused is extremely various. In some cases it hardly exceeds a very few ounces, while in other instances it amounts to many pints, separating the usually opposed surfaces of the membrane, and distending the cavity of the pleura, and compressing the lung. Laennec is of opinion that the time of effusion after the commencement of the inflammation is often very short, as he has detected ægophony and absence of respiration, as well as of thoracic resonance, an hour after the patient has first felt pain in the side. If the effusion be considerable, the lung becomes collapsed, contains no air, and therefore no longer crepitates; the vessels are devoid of blood, while the bronchi, even to the large trunks, are evidently contracted; still, if this lung be inflated, it enlarges more or less perfectly. Again, should the pleuritic effusion be less in quantity, some fluid appears spread all over the lung; but the greater quantity is collected at the lower portions of the chest.

Accompanying either of the previous forms, or existing, *per se*, the *fibrinous inflammatory lymph* may predominate, and adhesion of the opposed surfaces ensue. In many cases the lymph is loose and watery, rendering the serum turbid or flocculent; but in other cases it is more solid, and adheres with great tenacity to the opposite membrane, becoming organized at both surfaces. The organization of these membranes is rapid, and is often effected in the course of forty-eight or even twenty-four hours.

If the patient dies shortly after an attack of acute inflammation, these adhesions are found soft, easily lacerable, and extensible. If, however, he survives a longer period, the adhesions are often of great tenacity, indurated, and with difficulty separated from their attachments. The extent of membrane affected with adhesions is sometimes limited to a small portion, and sometimes extends over the whole surfaces of the cavity; but their most common seat is over the anterior lobes of the lungs, or the portion of pleura from the mamma to the axilla.

Niemeyer arranges pleurisy into the following forms:—

(1.) *Cases in which no symptoms occur*, dry pleurisy, or pleurisy with purely nutritive exudation, characterized by extensive *adhesions.*

(2.) *Cases in which the exudation is scanty but fibrinous.* It generally accompanies acute or chronic pneumonia, or other chronic affections of the lung, as sclerosis or other forms of pulmonary phthisis, as already described. Extremely delicate membranous coagula of fibrin coat the inflamed surface, causing the pleura to appear opaque, 'and hiding its redness beneath, which may be disclosed by scraping off the loose exudation. Sometimes the membrane is soft and white, like croup. Fluid does not generally co-exist in the cavity. The products of effusion are apt to undergo the cheesy transformation (*tyrosis*), to liquify and be absorbed; after which adhesions of opposed surfaces generally ensue.

(3.) *Pleurisy with abundant sero-fibrinous exudation*, involving the tissues of the parietal and costal pleura in very extensive lesions, and with effusion of serum into the pleural sac, equal in amount to two, three, and even ten, twelve, or more pounds of fluid.

This fluid exudation consists of two component parts, namely—(*a.*) A yellowish green serum; (*b.*) A quantity of coagulated fibrinous masses which float in the serum in the form of flakes or lumps, or traverse the serum in a loose net-work, or it is precipitated on the pleura, upon which it lies in the form of a membrane. A few pus-corpuscles may be found in this form of pleurisy, both in the fibrinous deposit and in the serum, and the transition to empyema is very gradual. The serum is turbid in proportion to the quantity of the pus, and the deposit is then more yellow.

General Symptoms of Pleurisy.—Like other inflammations of the lungs, pleurisy may be acute or chronic. The acute form of this disease may be preceded by fever, but often no such antecedent is present. Its local symptoms, however, in most cases, are strongly marked, the patient suffering with severe *continued pain* in the affected side, of "a dragging, shooting character," which is greatly exasperated by coughing or forced inspirations, movement, pressure, and percussion, so that the lungs can only be imperfectly filled with air. The seat of the pain, however extensive the inflammation, is generally limited to one point; and this point is usually about the centre of the mamma, or just below that part, towards the lateral attachments of the diaphragm. While the pain is constant, it nevertheless sometimes remits, and with the occurrence of effusion often totally disappears. It is often doubtful whether the pain is due to pleurisy, to rheumatism, to neuralgia, or to shingles. When the fibrinous effusion is scanty the pain is generally piercing when a "breath is fetched," and greater during ordinary than forced respiration. Coughing and sneezing are especially painful; and pressure from without or from within greatly increases the pain. Respiration is imperfectly carried out, and the body is bent towards the affected side. The tongue is commonly white, but the pulse varies, perhaps according to the form of the inflammation and its intensity. If the disease be limited to an effusion of lymph, the pulse is seldom more than 90 to 110, but "hard and concentrated in impulse." Either form of *pleurisy* is generally accompanied by a short troublesome cough, and some expectoration. The respirations are increased in frequency—phenomena of more constant occurrence than even the local pain—and, unless dyspnœa exist, are unnoticed by the patient. While one *respiration* is performed, only *three* beats of the heart, in place of *five* or *six*, occur. The patient likewise is for the most part restless, and lies on the affected side. If the effusion is great of sero-fibrinous exudation, the symptoms are acute, and run an acute course. Severe rigors usher in intense fever, with full and frequent pulse, headache, coated tongue and throat, pains in the back and limbs. The chills are often recurrent, thus differing from pneumonia, so that the disease may be mistaken for ague. At the commencement also pain in the side is sharp; but abates as the fluid effusion increases in the sac; and often ceases altogether before the effusion is complete. Dyspnœa is constant, and often becomes severe, but it may abate as the fever abates. The disease advances for six or eight days, when improvement ought to take place from rapid abatement of the fever, leaving the fluid effusion to be reabsorbed; which absorption ought to begin immediately in favourable cases, and progress rapidly (NIEMEYER); but even after many weeks a considerable remainder of fluid may still exist.

In another class of cases, the fever, being acute at first, moderates merely towards the end of the first week, or a little later, but does not wholly subside; and although exudation does not continue, yet absorption remains in abeyance. By and by a reaccession of all the symptoms takes place, and effusion again occurs to a greater extent than before. In this way the disease subsides and recurs, thus fluctuating for months, and at last, as a rule, terminates fatally. Another form develops in a latent and slow way, without inflammatory fever, and without pain, shortness of breath being the only source of distress to the patient; but who ultimately seeks advice on account of failing strength, and having become pale and thin. The abdomen, too, may be tumid, from pressure downwards of the diaphragm. In such cases the amount of fluid in the pleura is generally very great and highly albuminous.

Prostration and debility is extreme, from the constant fever that at last sets in; and finally such cases generally end in pulmonary phthisis (NIEMEYER).

But the physical signs of pleurisy are much more delicately varied according to the anatomical conditions of the disease. For example, at the most early stage, when the serous secretion is at first arrested, the expansion of the walls of the chest is diminished, as may be proved by measurement. The percussion-sound, however, is not perceptibly altered, and respiration is weak, because imperfectly performed. The characteristic *friction-sound* of inflamed serous membranes may, perhaps, now be detected, if listened for in the *infra-mammary* or *infra-axillary* regions. When the secretion has returned, increased in quantity, the signs continue as described, but the clearness of the percussion-sound becomes diminished, and the *friction-sound* is of a rubbing or grating character. The patient himself sometimes experiences a distinct sensation of friction. The period of inflammatory effusion is now established, and the infra-mammary and infra-axillary regions become more or less bulging; the projection of the intercostal spaces of the affected side during both respiratory acts becomes most obvious; the thoracic vibration from the voice is abolished where the fluid intervenes, and so also are friction-murmurs there. The area of dulness, and of the peculiar sounds, may be changed by altering the position of the patient. The natural respiratory murmurs become greatly intensified above the level of the effusion. When the effusion exists on the right side, the sounds of the heart are more clearly audible than in the natural state in the right axillary region, because the lung is more solidified by the pressure of the effused fluid.

Such are the more salient symptoms which mark the progress of pleurisy. The disease, however, may exist without any of the general symptoms. There may be neither local pain, cough, dyspnœa, nor febrile action, and yet effusion may have occurred to such an extent as to have reached the clavicle, while the patient remains utterly unaware that his chest was the seat of disease (WALSHE). The physical signs alone reveal the disorder—which is termed *latent pleurisy*—a form of disease which had no existence in nosology previous to the time of Laennec. Whenever, therefore, the least suspicion exists of disease in the chest, especially in elderly persons or those liable to constitutional affections, percussion and auscultation must never be neglected.

Causes.—The causes of pleurisy have an influence over its course; therefore a due appreciation of them is essential to a correct prognosis and treatment. The inflammation is sometimes said to acknowledge an idiopathic origin, as when the pleurisy is believed to arise from exposure to cold, or to the action of other atmospheric influences of which we know nothing. Exposure to cold, especially to currents of air when the person is heated, is a frequent exciting cause; but many now express their entire disbelief in cold being able to establish pleurisy in a healthy person (HYDE SALTER, FULLER). When exposure to cold is followed by inflammation of the pleura, the disease is most likely associated with some morbid condition of the blood, as in rheumatism (*rheumatic pleurisy*), and without doubt blood-poisoning is one of the commonest morbific causes of serous pleuritic inflammations. For example *anœmia, pyœmia,* the specific toxæmia of *eruptive fevers,* the *materies morbi* of *rheumatism, gout, scrofula, carcinoma, Bright's disease, alcoholism, syphilis, or even retained excreta, with pyrexia as the result of over-feeding,* are all apt to set up serous inflammation. All serous inflammations, in truth, especially point to states of blood-poisoning; as during the progress of typhus or puerperal fever, or during Bright's disease. Or the pleurisy may acknowledge some adjacent irritation, as *pneumonia,* constituting *pleuro-pneumonia.* In such cases the inflammation seems to spread by continuity from the neighbouring organs. But there are also numerous cases in which some growth in the lungs, such as *tubercle* or *cancer* advances to the pleura.

Prognosis.—Simple idiopathic pleurisy on one side of the chest, occurring in a person whose lungs are not chronically diseased, almost always terminates favourably, if taken in time and treated judiciously and with energy, and if the effusion has not been copious; but when it occurs as a complication in other diseases, the result may be doubtful, especially if air finds its way into the cavity (*pneumo-thorax*).

Treatment of Pleurisy.—In acute pleurisy, during the first stage, or that of *hyperœmia,* the best practitioners of all times and of all countries have taken blood from the arm, provided the strength be good and the symptoms sthenic; and if, says Laennec, after one or two bleedings, the pain in the side and fever have not abated, blood should be taken from the side by leeches or by cupping. Niemeyer, however, believes that, with the exception of a few rare cases, venesection can be dispensed with in the treatment of pleurisy. He is convinced that it neither cuts short the malady nor prevents the effusion. He considers the practice more dangerous than in pneumonia. He only resorts to it in cases of dyspnœa, arising from collateral hyperæmia, if the compressed part of the lung exists with œdema. The practitioner should also remember that effusion often takes place after bleeding, during the subsidence of the inflammation, so that the breathing is often more oppressed and the symptoms for a time aggravated, although the condition of the patient may appear to be improved.

At the commencement, Niemeyer recommends very strongly the use of cold compresses, and of local blood-letting by leeches or cupping, to be repeated in the course of a day or two until relief becomes permanent. The local blood-letting is only to be had recourse to if the cold compresses do not relieve the pain and the dyspnœa within two or three hours. Large hot poultices should be always employed when pain on inspiration is present, of a "catching" or "stabbing" nature. Niemeyer also recommends that *half a drachm of mercurial ointment* be rubbed into the affected side of the chest twice daily; but the remedy is to be suspended the moment mercurial fetor can be perceived in the breath.

Tartar emetic, says Laennec, is in general well supported in pleurisy, and contributes powerfully to subdue the inflammatory tendency; but, nevertheless, when the pain in the side and fever have ceased, it loses further power over the disease; at least it does not appear to promote the removal of the fluid effused, so that its use must generally be abandoned as soon as the acute symptoms have passed away. *Digitalis* is recommended by Niemeyer rather than *tartar emetic.* In recent cases he gives it in the form of *infusion;* but in the more latent cases *one grain* of the *powder of the leaves* combined with a grain of *quinine.*

With respect to the application of blisters, Laennec objects to their use until the acute stage is past; but when the pain has ceased for some days, and absorption of the fluid proceeds slowly, and the disease promises to become chronic, a succession of blisters may be applied. On the other hand, Niemeyer recommends the early application of large blisters in certain cases, as the fever is not aggravated by their use (MEYER and NIEMEYER).

The points aimed at by venesection being relief of dyspnœa from congestion and œdema of the compressed lung, and of pain; and to secure moderation in the force and frequency of the pulse, the patient ought to be bled in the upright posture if possible, and the blood should be allowed to flow in a full stream until he can take a deep breath freely, or till he feels faint and exhausted. From ten to twenty ounces may be necessary to accomplish this end, according to the severity of the case and the nature of the constitution. After free evacuation of the bowels has been effected, *calomel,* to the extent of producing the *slightest* mercurialization, is the most beneficial line of treatment, to be followed *in certain cases only*—namely, those which do not acknowledge any *constitutional disease* as their cause. The more rapidly slight mercurialization can be produced the better; and hence, writes Dr. Walshe, during the first six hours, small doses of *calomel with opium* (*a grain and a half of the former,* combined with *a sixth of a grain of the latter,* or more, if the pain continues acute) should be given every half hour, while mercurial ointment is rubbed into the skin of the affected side near the axilla every fourth hour. Dr. Fuller recommends *half grain* or *grain doses* of *opium* every *three* or *four* hours, in combination with *one* or *two grains of calomel, and half a grain of digitalis,* and to have *the whole side covered with a piece of linen spread with mercurial ointment:* over this is placed a poultice covered with oiled silk (l. c., p. 184). The patient must be carefully watched, so that neither *ptyalism* nor *narcotism* is produced. The moment mercurial action has been established, the further administration of the mineral must cease.

Opium and *digitalis* are advised to be continued after the use of calomel has been suspended; and with them may be given two grains of *squills* or of *nitrate of potash,* which will act beneficially as a diuretic; or after twelve or more hours very small quantities of *tartar emetic* in solution may be given at night, combined with small doses of *opium* and *ipecacuanha,* to allay cough and general irritation.

After the febrile action has in some measure subsided, and the active stage of the disease is at an end, a blister may be applied over the lateral region of the chest, but *not* over the seat of pain; and if the fluid continues to accumulate, the blisters ought to be repeated, so as to maintain a surface at a distance from the affected part in a constant state of counter-irritation. Diuretics ought at the same time to be freely given. The *compound tincture of iodine,* in doses of *twenty minims freely diluted,* is a valuable medicine at this juncture; and so also is the *liquor iodi of the British Pharmacopœa of* 1867.

In the chronic stage of pleurisy, physicians are agreed as to the necessity of generous diet and tonic remedies in aid of any diuretics and absorbents which may be employed. If the patient be kept too low after the stage of active inflammation has subsided, or be unduly depressed, it will not only be impossible to induce absorption of the fluid, but there will be great danger of its becoming sero-purulent in character. The unfavourable issue of pleurisy in its chronic stage is in many cases attributable to a want of tone in the system, caused by the treatment adopted—the patient being kept too low, or he is overmuch purged, or is in some other way unduly depressed, so that the system is unable to exercise its reparative power. A more generous diet ought to be given, and the general health sustained by quinine and other tonics. The use of diuretics and absorbents ought, at the same time, to be steadily persevered in. When a succession of blisters has failed in relieving the patient, *iodureted lotions*, or *iodureted ointments*, combined with the internal administration of *cinchona* with *tincture of iodine, iodide of potassium*, and small doses of *bichloride of mercury, nitre, acetate of potash, squills, digitalis, and cantharides*, have each in their turn effected the desired object (l. c., p. 186). The fluid has been gradually reabsorbed, and recovery has ensued by a combination of *iodine* remedies, taken internally and applied externally. *Syrup of the iodide of iron* (ʒii.), with *simple syrup* (℥ii.), and a tea-spoonful taken every two hours, in conjunction with the external application of a weak compound solution of iodine (*iodine*, ʒss.; *potass. iodidi*, ʒii.; aq. distil., ℥ii.) upon the affected side of the chest (Niemeyer). The formulæ most useful in such cases are—(1.) A diuretic in the form of a pill composed of *digitalis, squills*, and the mass of *pill hydrargyri*, of each a grain and a half; *nitrate of potash* (twenty grains), combined with *tincture of the perchloride of iron* (fifteen minims), three times a day, may at the same time be given (Fuller, Chambers). Professor W. T. Gairdner recommends as a diuretic the *cream of tartar* electuary, in which the *cream of tartar* is mixed in equal proportions with treacle, honey, or marmalade, and in some cases flavoured with a few drops of peppermint oil. The dose is a teaspoonful repeated as often as the stomach will bear it, or as the urgency of the case demands.

(2.) A lotion, to be applied over the chest by *spongeopiline*, or by *lint* covered with *oiled silk*, composed as follows:—

℞. Hyd. Bichloridi, gr. iv.; Tinct. Iodinii Co., ʒiv.—ʒvi.; Glycerini, ℥iii.; Aquæ Distillatæ, ℥ivss.; Ft. Lotio.

(3.) One or other of the following ointments may also be rubbed in upon the skin, over the side of the chest: namely—

℞. Hyd. Bichloridi, gr. iv.—v.; Ungt. Iodinii Co., ʒiv.—ʒvi.; Adipis, ʒiv.—℥i.; Ft. Ung.; or—

℞. Hyd. Bichloridi, gr. iv.—v.; Potassii Iodidi, ʒii.; Aquæ Distillatæ, q. s. ut Hyd. Bichlorid. et. Potassii Iod. solventur; Adipis, ℥i.; Ft. Ung.

Niemeyer, however, considers that remedies which merely aim at promoting re-absorption of the fluid deserve little reliance; but mainly on account of the action of diuretics being uncertain: and he rightly objects to drastic purgatives on account of their pernicious effect upon digestion and assimilation. Feeding the patient upon the driest possible diet, and withholding water as much as possible, has been successful in some cases (SCHROTH).

Professor W. T. Gairdner and Dr. Fuller are the two most recent authorities who notice the treatment of pleurisy by puncturing the chest and letting out the fluid—an operation to which the name of *paracentesis* has been given. With regard to acute pleurisy, Dr. Gairdner infers, from his own experience, that "for the mere saving of life in the acute stage of the disease, the operation is not necessary in any but a very small minority of the cases which have not yielded to remedies" (*Clinical Medicine*, p. 374). But a large proportion of the cases recorded in Dr. Gairdner's excellent work, just quoted, occurred before he began to adopt the method of withdrawing the fluid practised so successfully by Dr. Bowditch, an American physician. In consequence of the great facilities afforded by this method for withdrawing fluid from the chest, Dr. Gairdner's views regarding the operation have undergone considerable qualification. He now substantially gives in his adhesion to the principles and practices of Dr. Bowditch, and claims for therapeutics a correspondingly extended field of usefulness. He has only performed the operation in acute pleurisy in cases of *great distension*, after other remedies had a fair trial without effect; and has avoided doing it in cases of partial or moderate effusion.

As a means of relieving the chest in chronic pleurisy and empyema, the operation is a useful one; and the practical question is as to the time when the operation should be performed. The condition of the patient's health and respiration, and the absorption or non-absorption of the effused fluid, are the aids to a decision for or against the performance of the operation. As long, writes Dr. Fuller, as the breathing is not seriously embarrassed, and the general health does not decline, so long are we justified in making full trial of our remedies. But as soon as extreme shortness and distress of breathing, or lividity and anxiety of the countenance, denotes serious interference with the functions of life, delay is no longer justifiable, and it becomes our duty at once to give our patient the chance which the operation affords.

The space between the fifth and sixth ribs, counting from above downwards, should be selected (if adhesions are ascertained not to exist there), being the most depending part of the chest when the patient lies on his left side—the more usual position in this disease.

The operation is described under the subject of hydrothorax:—

EMPYEMA.

LATIN EQ., *Empyema;* FRENCH EQ., *Empyème;* GERMAN EQ., *Empyem;* ITALIAN EQ., *Empiema.*

Definition.—*Pleuritis, with purulent exudation.*

Pathology.—The pleuritic inflammation sometimes terminates in suppuration; and should the pus be in such quantity as to accumulate in the cavity of the chest, the disease is termed *empyema.* Empyema may be true or false; it is said to be true when the pus is secreted by the pleura, and false when it results from the bursting of an abscess of the lung into the cavity of the chest (*pyothorax*). The quality of the pus in true empyema varies from a genuine laudable pus to a sero-purulent fluid. In quantity, also, it varies from a few ounces to many quarts, filling the entire cavity of the chest. Under these latter circumstances the side of the chest is dilated, and the intercostal spaces are widely separated and bulging.

Effusion of pus may take place into either cavity of the chest, but the left, perhaps, is the more common. The phenomena accompanying empyema of the left side are remarkable; for, besides the lung being found collapsed, and not so big as the fist, the heart is sometimes seen transposed as far over on the left side as it usually is on the right. In cases, however, in which *paracentesis* has been performed, and the pus has been drawn off, the heart is observed to return to its place, while the lung, less completely collapsed, may be bound down to the upper and lower portion of the chest by long and multiple adhesions.

Symptoms of Empyema.—Again, if the inflammation is about to issue in the formation of pus, the pulse is extremely small and frequent (from 120 to 150), while the restlessness and anxiety of the patient are greatly increased. There are cases, however, of empyema in which the commencement is very insidious, owing to the serious implication of the general system and blunted condition of the sensorium (NIEMEYER). The patient, in such cases, suffers little pain, or any more marked symptom than usually awaits the last stages of phthisis. In some instances he is for a time even capable of walking about the ward of an hospital or in a bedroom. Supposing, however, empyema to have occurred in a case of pleurisy, any acute pain which may have existed subsides, but the anxiety of the patient is increased, and his state of collapse shows his imminent danger. If the constitution be less affected, the symptoms vary according to the side of the chest which is the seat of the empyema. If it be on the left side, for example, the heart is often transposed, and felt beating as far over on the right side as it usually does on the left, and the pulse is small and frequent. If we now bare the chest of the patient, we find the affected side enlarged, sometimes œdematous, with projecting intercostal spaces. As the lung is now greatly compressed, no respiratory action is seen on that side, which is entirely at rest. If *paracentesis* be now performed, the heart is restored to its place as the pus flows; but as the lung for the most part only imperfectly expands, the affected side, even in the most favourable cases, contracts, and the spinal column, pressed upon by an unequal weight, acquires a lateral curvature, the shoulder sinks, and the patient is greatly and permanently deformed.

Auscultation and percussion are equally valuable in determining the amount of effusion. If serum or pus be effused to the amount of a pint, for instance, the lung is displaced to that extent; and consequently the lower portion of the chest, when struck, returns a dull sound, which extends as high as the level of the fluid. If we now auscultate the patient, the respiration is also lost below the level of the fluid. Besides these results, the voice gives very striking indications of the lung becoming so far condensed from the pressure of the fluids; for we very constantly have *bronchophony*, and occasionally *ægophony*.

If the chest be completely filled in empyema, the respiratory sound is altogether wanting; so is *ægophony* and *bronchophony*, and the containing cavity returns a dull sound at whatever part percussed. Under these circumstances, and especially if the heart be displaced, the affected side will be seen entirely motionless, rounded, and distended; and when these signs are present, there can be no doubt that the distension is due to the presence of serous effusion or pus.

HYDROTHORAX.

LATIN EQ., *Hydrothorax;* FRENCH EQ., *Hydrothorax;* GERMAN EQ., *Hydrothorax*—Syn., *Brustwassersuch;* ITALIAN EQ., *Idrotorace.*

Definition.—*Watery fluid or serum in the cavity of the pleura (of either or both sides), rarely occurring in the absence of pre-existing disease of the pleuræ, lungs, heart, or great vessels, nor without the influence of some specific or constitutional disease—not the result of exudation, but a "passive dropsy of the pleura."*

Pathology.—In hydrothorax the cavity of the chest, on being opened after death, is found more or less full of watery fluid or serum, which being removed, the pleura is seen sometimes healthy-looking, but more generally of a dark colour, in consequence of a quantity of venous blood being congested in the vessels. The fluid may be effused into one or into both, generally into both, cavities. It may also be limpid and colourless, like water; but more commonly it is citron-coloured, and contains fibrin, much albumen, and sometimes urea, in cases of *Bright's disease*. "*Water on the chest*," as it is commonly called, is generally traceable to increase of lateral pressure within the veins, especially obstructed action of the right side of the heart, and decrease in the amount of albumen in the serum of the blood (NIEMEYER). Like other dropsies, therefore, it is a symptom of a disease rather than a substantive disease itself; and in statistical returns it ought to be returned under the primary disease, of which it is found to be a secondary affection. It is one of the lesions in *general dropsy*. The quantity of fluid effused varies from a few ounces to many pints: eight and nine pints are not unusual; and Laennec states that he once removed twelve pints from the right side of the pleural cavity. When the quanity is large, the lung is compressed towards its roots, and placed generally in the groove formed between the sides of the bodies of the vertebræ and the heads of the ribs to their angles. It is sometimes so flat as not to be more than half an inch in thickness. It may be compressed against the sternum if previously-existing adhesions to that region have fixed its position there. When hydrothorax is secondary, almost every chronic affection, either of the liver, kidney, or heart, may be found co-existing at the same time. The fluid of hydrothorax is distinguished from the fluid of pleuritis by the absence of fibrinous coagula, and of inflammatory changes in the pleural surfaces. Occasionally it is the result of extremely slight pleuritis, rarely of severe pleurisy; and in these cases the serum is more flocculent, contains more albumen, and portions of lymph are often also seen adherent to the *pleura pulmonalis* or *pleura costalis;* the two pleuræ are also often more or less united; and the fluid encapsuled in old adhesions.

Symptoms.—The effusion may take place either gradually or suddenly. In the former case it may be so slow that the lung is able to adapt itself to the presence of the accumulating fluid, and the symptoms will consequently be much less marked, although the effusion be large. In the latter case the functions of the lung are almost at once suspended, the countenance livid, and the breathing greatly disturbed. When the effusion is slow, the symptoms are,—difficulty of respiration, which is carried on rather by the shoulders and diaphragm than by the intercostal muscles, some expectoration, lividity of the face or lip, œdema of the legs, and either a very full labouring pulse, or one that is small, frequent, and intermitting: the urine also is extremely scanty. As long as the effusion is moderate the patient can lie flat in his bed without experiencing any inconvenience. In the event, however, of the effusion being so considerable that the function of the lung is entirely suspended, the patient is unable to lie down, from the sense of suffocation produced by the fluid gravitating towards the root of the lung, and compressing the larger bronchi, and he therefore sits propped up by pillows, with his head bent forwards. When hydrothorax is symptomatic, or consecutive to affection of the heart or of other disease, it is generally preceded by swelling of the legs or eyelids, by the urine being plentiful and albuminous, or scanty, high-coloured, and loaded with the usual salts, and indeed by most of the symptons of dropsy generally. In these cases the effusion seldom takes place into the chest till a few days before death, rendering the agony of death doubly painful and suffocating.

When the effusion is moderate, auscultation gives bronchial respiration, some mucous rhonchus, and *bronchophony*, and occasionally that condition called *œgophony*, which is a broken sound like the bleating of a goat, or the notes used in the exhibition of "Punch," and which is heard as though the patient was speaking at the end of the stethoscope, but not through it. This singular phenomenon is heard only when the instrument is placed about the level of the effused fluid. When the effusion is more considerable, the respiration is almost tracheal; there is neither *bronchophony* nor *œgophony*, and a dull sound is returned over a greater part of the chest. Again, if the patient's chest be bared, there is no expansion on the side of the seat of the effusion, the respiration of that part being carried on altogether by the shoulders and diaphragm; and should the effusion be excessive, the affected side bulges out, as in empyema, and its intercostal spaces are enlarged and prominent. If air exists as well as fluid, succussion of the patient gives the sound of the splashing of fluid, and sometimes the patient can produce this phenomenon by shaking his body himself.

Diagnosis.—The absence of pain and of the other symptoms of inıammation distinguishes this disease from *acute pleurisy.* Should, however, the pleurisy be chronic, it is impossible to distinguish the two diseases except by the previous history. The diagnosis, also, between *hydrothorax* and *œdema of the lung* is not always easy.

Prognosis.—Some cases of hydrothorax recover, but the prognosis is in all cases extremely grave and doubtful.

Treatment.—The treatment of hydrothorax is of great difficulty, from the many causes on which the effusion may depend, and also from the almost uniformly intractable nature of the disease. The general principles of the medicinal treatment to promote absorption of the fluid have been given under the section on the treatment of pleurisy; and it only remains here to notice the great facilities afforded for the removal of the fluid by the operation recommended by Dr. Bowditch, with the apparatus devised by Dr. Morrill Wyman, of Cambridge, U.S. The apparatus consists of a trocar a little larger than the ordinary exploring trocar, and a silver canula, with a stop-cock in silver, as light and small as possible, capable of being connected with a syringe by an intermediate piece of brass, also provided with a stop-cock, the two cocks working the same way, and acting as checks upon each other. Such instruments may be had of Mr. Kemp, Philosophical Instrument Maker, Infirmary Street, Edinburgh; and a drawing of the instrument may be seen in vol. xxiii., p. 348 of the *American Journal of Medical Science;* also for Jan., 1863; also vol. xx., Oct., 1850.

During the operation the patient should be seated, when possible, sideways on a chair, or astride the chair, with his face to the back of it; or, if unable to rise, he ought to be brought so that the affected side may be made to incline slightly over the edge of the bed. The most appropriate spot for puncture is between the seventh and eighth, or the eighth and ninth, or the ninth and tenth ribs, in a line let fall from the lower angle of the scapula; but as a rule let the trocar be introduced as low down as possible, consistently with the safety of important organs in the chest or abdomen. The exact position of the liver and spleen must be determined first in every instance. Laennec himself once transfixed the diaphragm and pierced the liver, and that through the *fifth* intercostal space. An enlarged liver or spleen may be detected as high as the fifth rib. Dr. Watson once witnessed an operation in which the trocar was pushed through the diaphragm into the spleen, which was unusually large. The patient died a day or two afterwards of peritonitis. Just under the lower angle of the scapula is a spot easily reached, and where the muscles are thin. But in selecting the precise intercostal space, Dr. Bowditch chooses one about an *inch and a half higher* than the line on a level with the lowest point at which the respiratory murmur can be heard in the healthy lung of the opposite pleural cavity. Having pressed the forefinger of the left hand deeply into the intercostal space, the trocar, with its canula, should be plunged through the tissues at the depressed part, keeping as near as possible to the upper edge of the lower of the two ribs. The point of the instrument should be raised rather than depressed, so as to avoid injury to the diaphragm, liver, or spleen. The skin need not be incised before puncture. Having withdrawn the trocar, leaving the canula, the double-valve syringe is to be applied, and the effusion slowly drawn away, until the lung has undergone as much expansion as it can endure with safety. This will be indicated by a sense of dragging distension or pain. When the canula is removed, the wound contracts and closes so completely that no lint or dressing is required.

In the hands of the American physician, Dr. Bowditch, and Professor W. T. Gairdner, of Glasgow, and Dr. Budd, of London, this operation has been the means of saving many lives. It is comparatively harmless, gives but little pain, and in the opinion of these eminent physicians it is an operation which ought never to be allowed to fall into disuse by the profession. The effect of the operation is to relieve the mind as well as the lung of the patient from great oppression. Although before the operation he is quite weak, he is often able after it to get up and walk. Digestion becomes at once improved, and strength is rapidly regained. The cough, however, is apt to augment during the first few days; the pulse also retains its quickness; friction-sounds occasionally become developed; and several months may elapse before the vesicular murmur becomes properly re-established in the lung.

Whenever the pleural cavity has become distended with fluid, and the dyspnœa is great, Dr. Bowditch recommends operation without delay. When thus performed early, it prevents a long and tedious illness and future contraction of the chest. It should also be resorted to in all chronic cases when the effusion does not disappear after a reasonable time and the use of appropriate remedies. Dr. Bowditch has performed the operation 150 times on 75 persons, and has seen it done in 10 other cases. Out of the 75 cases, 29 recovered completely, and apparently in consequence of the operation, which was generally performed after severe symptoms had set in; and in all these cases the tapping seemed to be the first step towards recovery. In 26 of the 75 cases the fluid obtained at the first tapping was *serum;* and 21 of these cases made good recoveries. If the fluid afterwards became *purulent*, an almost certain fatality attended such a change. Of 6 such cases, 4 died; and the two others were likely to die when Dr. Bowditch wrote. In 24 of the 75 cases, *pus* flowed at the first tapping; and 7 of these recovered, and 7 died. In such cases relief is always obtained; but the tendency remains to a fistulous opening or to phthisis. If the fluid at the first tapping is sanguinolent, thin, and of a dark red colour, not coagulating, it forebodes an almost certainly fatal result; and is generally associated with some malignant disease of the lung or pleura. Of 7 such cases, 6 died; and the seventh was still lingering when Dr. Bowditch wrote. A mixture of bloody purulent fluid at the first operation is usually fatal. A fœtid gangrenous fluid is very rare, is obviously of bad omen, and betokens gangrene of the pleura and lung.

The operation may require to be repeated. Dr. Bowditch has done it *eight times* in six weeks to the same patient—himself a physician. One lady he tapped *nine times* in eight months and a half, commencing when she was four months and a half pregnant, and when orthopnœa was threatening death. She was delivered of a living child at the full time, and recovered. In one remarkable case a youth had obscure symptoms for nine months. Dr. Bowditch recognized by the physical signs "latent pleurisy," or "idiopathic hydrothorax," as some may style it. Four pints of fluid were removed at one time, and the lung was fully expanded in forty-eight hours. In three weeks the patient was well, and he continued so.

Dr. Bowditch, in a letter to Dr. Gairdner, thus states the general results to which he has arrived in the use of this operation (*Clinical Medicine*, p. 720):—"I now never operate unless I find some distension or rounding out of the chest, and filling up of some of the intercostal spaces, so that the chest presents a uniform curve, and not alternate depressions and elevations, as in the healthy chest. I operate under the following circumstances when I feel certain there is fluid:—

"1. When there is *severe permanent dyspnœa—orthopnœa*—however acute the disease, if I find fluid filling the pleural cavity, or nearly filling it.

"2. When there are occasional attacks of *orthopnœa* threatening death, even if there be not sufficient to fill more than half of the cavity. If the fluid seems to be the cause of the dyspnœa, I operate, because occasionally I have lost a patient when waiting for more extensive physical signs. This rule I apply to acute and chronic cases.

"3. I use the trocar after three or four weeks of ineffectual treatment, without any absorption being produced.

"4. In chronic idiopathic hydrothorax, a latent pleurisy, with simply physical signs to indicate *extensive* effusion, but when the rational signs are either very slight or none at all, save a general malaise and weakness."

In the experience of Sir Thomas Watson the operation of *paracentesis thoracis* in simple pleurisy is not to be performed unless the life of the patient is in jeopardy—that is, in cases "in which the effusion continues and increases, and the side, instead of shrinking, enlarges; the functions of the lung on that side are entirely abolished; nay, the use of the remaining lung is greatly interfered with by the pushing over of the mediastinum; and the patient is in imminent danger of suffocation." In such cases the oppressed lung must be relieved by "*letting the fluid out*" (*Lectures*, 4th edit., vol. ii., p. 128). Again he writes, "Life is plainly in jeopardy when the vital functions of the lungs or of the heart are greatly hindered; when symptoms present themselves of approaching death by *apnœa* or by *syncope.* If we discover no cause for those symptoms except the increasing pressure of liquid pent up in the pleura, we are warranted in ascribing them to such pressure, and bound to act upon that persuasion." Also, if death by *asthenia* appears inevitable, the patient losing ground from day to day, and when all other means of getting rid of the pent up fluid have failed, the patient should not be denied the *chance* which the operation affords. Also, "whenever the effused liquid consists of *pus*, it should be let out" (l. c., p. 130). With the arrangements of Dr. Bowditch's syringe we are enabled to remove fluid *before* the *false membrane* thickens over the compressed lung, and so makes it difficult to expand again. The trocar and small canula take the place of an exploring needle, which Sir Thomas Watson does not object to use, regarding its use as a "minor diagnostic puncture;" and if the lung is not covered by an inexpansible *false membrane*, it will gradually expand as the fluid is withdrawn, the operation being done with the greatest slowness possible.

By the peculiar construction of the exhausting syringe, Dr. Bowditch has been enabled to evacuate the chest much more completely than by any other method, and to prevent entirely (and with absolute certainty) the admission of air. "It appears to me," says Dr. Gairdner, "to be in every respect an improvement so important that it may be said to open up a new history for the operation of *paracentesis thoracis;* and I trust it will receive in this country the attention which is due to it" (*Clinical Medicine,* p. 380). On the other hand, and by Dr. Bowditch's own showing, it does not appear that the entrance of air into the cavity of the pleura produces dangerous symptoms. He has never found this to be the case, even when air has been pumped into the chest. Why, therefore, resort to so elaborate an arrangement of stop-cocks, syringes, and canulæ, and tubes! Dr. Fuller regards such instruments as practically inoperative. "The lung," he says, "is seldom able to expand freely at once; and as the chest-walls will not yield beyond a certain point, air must be allowed to find ingress into the pleural cavity, or the fluid would not flow out. I have seen two of the most perfect of these stop-cock instruments employed; and so long as precautions were taken to exclude air from the pleura, so long they failed to draw off more than a few ounces of the fluid; directly air was admitted, the liquid flowed through them freely. Therefore, however desirable it may be theoretically to exclude air from the pleura, it is practically impossible to do so if we wish to relieve our patient" (FULLER, l. c., p. 192). But Dr. Fuller agrees in the view that "when acute inflammatory action has subsided, the admission of air does not necessarily excite suppurative action of the pleura,—nor is it found to interfere with the re-expansion of the lung." He believes that the lung is very slow in regaining its due expansion—more than five days. Yet he agrees also that every precaution should be taken to prevent admission of air (as it is certainly conducive to suppurative inflammation) when the grooved needle has shown that the effused liquid is serous (l. c., p. 193). Hence, I think, we come back to Bowditch's plan as the best. "When, on the contrary, the grooved needle has proved the existence of pus," Dr. Fuller believes "that the admission of air is not of the slightest importance." But here, again, we know that pus formed internally, and not in contact with air, undergoes changes when exposed to air which renders it not less imperative that we should, if possible, prevent its admission; therefore, if the lungs are not bound down by adhesion, or prevented from expanding by the thickness of the *false membrane* which covers it, here again Bowditch's syringe and stop-cocks enable the fluid, even when purulent, to be withdrawn without air being admitted. Mr. Lister's method and precautions as to opening an abscess ought to be adopted.

But when air has been admitted, and when the fluid within the cavity of the chest has become changed thereby—is purulent and perhaps fetid—the evacuation of the contents by "drainage" seems the most efficient remedy. This operation consists of introducing, through the opening made in the chest for removing the fluid, "a fine, long iron probe, somewhat bent." It "is then directed towards the lower and back part of the pleural cavity,—the lower the better. If the end of the probe be made to press against the side of the thoracic walls, it can be felt from the outside, through the intercostal spaces, though perhaps obscurely, owing to thickness and toughness of the false membrane. The lowest and most appropriate site in which the probe can be felt having been selected, an incision is made upon the end of the probe, which is then brought through the opening thus made. A strong piece of silk thread is passed into the eye of the probe and drawn through the two openings, and the drainage tube—an india-rubber tube, perforated at frequent intervals, in the way recommended by Chassignac for the treating of sinuses—being firmly tied to one end, is then drawn through by means of the silk: the ends of the tube are then tied together, and the operation is complete. . . . The openings in the chest walls are thus always free; the matter is discharged drop by drop as it forms, so that if the tube be suitably placed, there is never any collection of pus in the thorax; no time is given for decomposition, and the pus, therefore, is discharged in a healthy and pure state," (Goodfellow and De Morgan, in *Med.-Chir. Trans.*, vol. xlii.) Fetid fluid may thus be got rid of, and the cavity of the pleura washed out with warm water, containing a weak solution of *permanganate of potash* (Condy's fluid), of the strength of *two fluid drachms* to the pint of water (Fuller). Sir Thomas Watson is of opinion that if the corrupt and corrupting mass, in two cases which he cites, had been duly removed, the patients would have had a much better chance of life. With such a tube and a syringe, offensive gases and fluids may be got rid of.

Mr. Lister's antiseptic methods of treatment are of special advantage in such cases.

PNEUMOTHORAX.

Latin Eq., *Pneumothorax;* French Eq., *Pneumothorax;* German Eq., *Pneumothorax;* Italian Eq., *Pneumotorace.*

Definition.—*A collection of air or gas in the cavity of the pleura, generally co-existing with fluid in the same cavity,—when the condition is sometimes termed* hydro-pneumothorax.

Pathology.—Such a collection of gas or air within the cavity of the pleura, on either or both sides, occurs under any of the following conditions (Fuller, l. c., p. 199):—

1. When no communication exists between the pleura and the external air, it is then presumed to be due either to the spontaneous evolution of gas from decomposing fluid in the pleura, from gangrene of the pleural membrane, or from the secretion, generation, or exhalation of air from the pleura—a phenomenon of doubtful occurrence *per se.*

2. When a communication takes place between the pleura and the alimentary canal, as in cases of softening and perforation of the œsophagus or stomach.

3. When a communication occurs between the pleura and the atmo-

sphere, through an opening in the chest-wall, the result of penetrating wounds of the thorax or of parietal abscess.

4. When a communication occurs between the pleura and the bronchi, either the *result of violence,* rupturing the lung-substance, and tearing the pulmonary pleura; or the *result of disease,* causing perforation of the pulmonary pleura from without inwards, as in cases of *empyema;* or from within outwards, by ulceration. Such ulceration may be due to *tuberculous disease,* to *hydatid cysts, cancerous growths, pneumonic gangrene, metastatic abscess* or *bronchial gland abscess;* or, lastly, the lesion may be the consequence of *emphysema,* or *pulmonary apoplexy,* or minute bronchial abscesses perforating the lung during the course of typhoid fever (GAIRDNER).

Numerous cases of pneumothorax are to be met with from the bursting of a pulmonary cavity during inspiration, or from the opening of a pleural effusion into the lung. Indeed, tubercular ulceration of the pulmonary pleura is the efficient cause of pnemothorax in 90 *per cent.* of the cases in which air is found in the pleura (WALSHE, FULLER). Cases also are on record of pneumothorax ending in recovery, with remarkable absence of bad symptoms. Such a case has been described by Dr. Thorburn (*Brit. Med. Journal,* June 2, 1860), and is probably a unique example of pneumothorax occurring in a previously healthy man, and terminating in a recovery virtually complete, running its course without fever, and with singularly little pain or dyspnœa. Dr. Gairdner also, in commenting on Dr. Thorburn's case, observes that the acute and terrible symptoms so characteristic of pneumothorax in typical cases are occasionally absent individually, or at least not of diagnostic value absolutely. The disease, therefore, is one which may be overlooked in such cases where the symptoms are so latent that the date of invasion cannot be determined; and instances of the occurrence of pneumothorax being marked by the severe sufferings caused by another disease are so common as to be now well known (GAIRDNER). Cases of phthisis which do not seem to depart from their ordinary course, yet during the course of which aggravations are apparent, are just the kind of cases in which we may find, on examination, the pleura of one side more or less filled with air, but in which no date can be assigned to the pneumothorax, which has thus been detected at a period more or less remote from its actual occurrence (GAIRDNER, l. c., p. 391). To detect the existence of any minute opening in the pleura after death, the side of the chest affected should be filled with water, and if the lung is then gently inflated, bubbles of air will escape if a perforation exists, however minute. It commonly occurs in the area comprised between the third and sixth ribs (WALSHE, FULLER, CHAMBERS), "motion being greater there than in any other part of the chest," and therefore the pleura is less likely to be protected by adhesions there.

Symptoms.—The signs of pneumothorax are, marked deficiency of respiratory murmur, without dull percussion, and with metallic phenomena, such as "a clear mixing click, as of water dropping into a well" (*tintement métallique*), audible with almost every inspiration, and succussion sound in a more or less well-marked degree. Nevertheless, "it is extremely difficult," as Dr. Gairdner observes, "to say precisely what, in the present state of science, constitutes complete evidence, in a clinical sense, of the existence of air in the cavity of the pleura. The combination of *very marked* deficiency of respiratory murmur, with very marked euphonic respiration or metallic tinkling, would probably be accepted by the sceptical critic."

These phenomena only present themselves when the pneumothorax is very extensive; and such cases rarely occur except in conjunction with pulmonary tuberculosis, when it is generally fatal. "Pain may be only moderate in degree, or not characteristic. Dyspnœa may be merged in a more chronic affection of the chest; fever may hardly be observed amid the hectic of phthisis or of empyema; while the more special sensation of rupture, alluded to by some authorities, and the suddenly-felt rush of fluid or of cold air into the chest, as described by others, are certainly quite as often absent as not" (GAIRDNER, l. c., p. 387).

The signs of water, as well as air in the chest, may be discovered within a few hours after the presence of air has excited sufficient irritation to establish the pleuritis and serous effusion.

"The general symptoms in typical cases are comprised in physical distress and mental anxiety, as expressed in the countenance. The complexion is pale and dusky, and the lips more or less livid. The voice is weak; the skin moist, and often covered with a cold clammy perspiration; the pulse is quick and feeble; and the respiration is extremely hurried" (FULLER, l. c., p. 204).

The physical signs in typical cases comprehend—

> "Convexity of the affected side, with obliteration, widening, and even bulging of the intercostal spaces, immobility or diminished movement of the chest-walls, and inaction or diminished movement of the intercostal muscles, contrasting forcibly with the increased play of the opposite side of the chest and the energy of its intercostal action. *Palpation* informs us that *vocal fremitus* is diminished or altogether annihilated; that the intercostal spaces are more than usually elastic or resilient; and that the heart is more or less displaced. *Mensuration* confirms our impression respecting enlargement of the affected side and the increased width of the intercostal side. *Percussion* elicits a clear tympanitic resonance, which sometimes changes its character and becomes amphoric and of a metallic quality over the trachea and larger bronchi . . . and the area of clear resonance on percussion may extend considerably beyond its normal limits, as the mediastinum, the heart, and the diaphragm are more or less displaced. . . . *Auscultation* furnishes different results, according as the amount of the effused air is larger or smaller. If the quantity of air be small, the respiration-sounds are weak and distant, and the vocal resonance is weak; if it be great, so that the lung is thoroughly compressed, the respiratory sounds and the vocal resonance are almost or altogether absent, except in the interscapular region, at the root of the larger bronchi, where diffused blowing respiration and diffused but loud vocal resonance may still be audible" (FULLER, l. c., p. 204).

Certain signs, resulting from the co-existence of air and serous or other fluid in the same cavity, which are not met with either in pleurisy or pneumothorax when they exist independently of each other, occur with the combined condition named *hydro-pneumothorax,* and are characteristic of it. These are described by Dr. Fuller as follow:—(1.) *Fluctuation*, which is felt by the patient as well as by the observer, when the patient's body is abruptly jerked or shaken; (2.) A ringing, splashing sound—the succussion sound of Hippocrates—which is heard under the same circumstances; (3.) A remarkable metallic tinkling, which sometimes accompanies succussion of the patient, but which is also apt to accompany cough, or inspiration, or a sudden change in the patient's posture (l. c.. p. 205).

Prognosis.—When it affects the whole, or nearly the whole, of one side in tubercular disease, it is usually fatal after periods varying from minutes to weeks. *Limited* pneumothorax, however, is less fatal, as in cases, for example, in which the lung gives way by a mere *pin-hole perforation;* but the pre-existence or rapid formation of adhesions limits the escape of air to a part only of the cavity, and the pin-hole opening is sealed up. Pleurisy in such cases, therefore, in relation to pneumothorax, is not to be regarded as a fatal complication, but as a healing power (GAIRDNER, l. c., p. 396), the formation of adhesions often anticipating perforation of the pulmonary pleura in cases of tubercular phthisis. These adhesions limit the escape of air, prevent the utter collapse of the lung when air does escape, and so maintain to some extent the function of the lung, which otherwise would be destroyed as a consequence of such collapse. Prognosis is most unfavourable in perforation cases resulting from disease in the tissue of the lung, as contrasted with the more favourable prognosis in traumatic cases, in which the chest-walls are wounded, but in which the lung is not wounded. And those cases, *cæteris paribus*, are most apt to run an untoward course where there is great accumulation of air and great displacement of the thoracic organs (FULLER).

Treatment.—Puncturing the chest-walls, to relieve tension, may give temporary relief, by allowing the air to escape; but as the relief is only temporary, the operation ought only to be done in cases where the dyspnœa is urgent, and the displacement of the viscera such as demands that relief which the operation is calculated to give. Otherwise, the treatment of pneumothorax is best conducted by small doses of morphia —*i. e.*, stimulant doses—frequently repeated, combined or not by *æther* or *alcohol* in small doses, with a view to overcome collapse, relieve dyspnœa, and subdue pain. When re-action follows the collapse, as indicated by heat of skin, strength and hardness of the pulse, soreness and pain of the affected side, local blood-letting by leeches, with saline aperients, may be used if the strength of the patient warrants such a line of treatment. Turpentine and poppy fomentations, followed by blisters, are also indicated if life is sufficiently prolonged after the primary inflammatory symptoms are subdued. In other respects the treatment is similar to that stated under pleurisy.

CHAPTER XX.

DISEASES OF THE DIGESTIVE SYSTEM.

SECTION I.—DISEASES OF THE MOUTH.

STOMATITIS.

LATIN EQ., *Stomatitis;* FRENCH EQ., *Stomatite;* GERMAN EQ., *Stomatitis* —Syn., *Entzündung der Mundschleimhaut;* ITALIAN EQ., *Stomatitide.*

Definition.—*Inflammation of the mouth.*

Pathology.—*The mouth* is liable to various forms of inflammation, especially in children. They have received the name of "stomatitis." These inflammations may be either *simple, ulcerative,* or *vesicular.*

Simple erythematous inflammation occurs in patches, and is generally due to hot or acrid substances taken into the mouth; to cold, or the irritation of the teeth; or to tartar upon them. It may be also due to gastric derangement. Small doses of *magnesia,* or of *rhubarb with soda,* will generally correct the stomach derangement, when the stomatitis will subside.

ULCERATIVE STOMATITIS.

LATIN EQ., *Stomatitis exulcerans;* FRENCH EQ., *Stomatite ulcéreuse;* GERMAN EQ., *Ulcerative stomatitis;* ITALIAN EQ., *Stomatitide ulcerosa.*

Definition.—*Ulcerative inflammation of the mouth.*

Pathology.—In its milder form the disease is also known by the name of *noma;* but there are different grades of the ulcerative lesions, from *noma* up to *cancrum oris.*

Noma, or the milder form, generally commences at the edges of the gums opposite the incisors of the lower jaw. At these points the gums appear white, become spongy, and separate from the teeth, as if mercury had produced its specific effects. Ulceration begins and extends along the gums until the jaws are implicated; and as the disease advances the cheeks and lips begin to swell, so as to form a tense indurated tumefaction. The teeth are apt to fall out; and the gums assuming a gangrenous condition, the breath becomes intolerably fetid. There is generally enlargement, with tenderness of the submaxillary glands.

THRUSH—*Syn.,* APHTHA, VESICULAR STOMATITIS.

LATIN EQ., *Aphthæ*—Idem valet, *Stomatitis vesiculosa;* FRENCH EQ., *Aphtheuse;* GERMAN EQ., *Soor*—Syn., *Schwämmchen Aphtha;* ITALIAN EQ., *Afte.*

Definition.—*Vesicular or follicular inflammation or aphtha of the mouth.*

Pathology and Symptoms.—This disease usually commences as a simple stomatitis; but very soon small, round, transparent, greyish or white vesicles appear, and at the base of each is an elevated marginal ring, which is pale and firm. Fluid soon escapes from the ruptured vesicle; an ulcer forms, which spreads, bounded by a red circle and an elevated border. These ulcers are sometimes covered by a pultaceous formation, in which the secretion of the mouth is greatly altered and increased. The disease chiefly attacks the new-born infant; but a similar condition is sometimes seen in the adult towards the termination of long wasting diseases, and especially in phthisis. The whole surface of the mouth exhibits unusual redness, with here and there cord-like exudation in irregular patches, preceded by vesicles, especially behind the lips, and about the tip of the tongue. These patches are thrown off, to be renewed again; and the mucous membrane below is of a bright red colour. The adjacent glands are apt to become tumid and tender. The skin is commonly hot and dry; thirst is considerable; swallowing seems to give pain; and diarrhœa may ensue to a degree which soon proves fatal in an infant. The disease frequently occurs as a sequela of measles. In some forms of the affection microscopical parasitic plants occur. The parasite usually found in the mouth is the *oidium albicans,* or so-called *thrush fungus.* The general health of the little patient is frequently much disturbed, and diarrhœa, with very offensive evacuations, is a common accompaniment.

Treatment.—The mouth should be frequently washed with emollient fluids, such as *linseed infusion, diluted glycerine,* and *biborate of soda,* or *honey* mixed with *biborate of soda.*

Dr. Tanner recommends the following lotion:—

℞. Sodæ Biboratis, fl. dr.; Glycerinii, fl. oz. ii.; Aquæ Rosæ, fl. oz. iv.; *misce.* To be painted over the lips and mucous membrane of the mouth and tongue.

Creosote, vinegar, carbolic acid, glycerine, and *alcohol* are also recommended as local applications.

The late Dr. Symonds, of Bristol, recommended combinations of *turpentine,* with *glycerine* and *alcohol.* Equal parts of *turpentine* and *glycerine,* applied with a soft brush two or three times a day is a curative application, whether the lining of the mouth be red and puffed, or pale and œdematous, or studded with superficial ulcers, or opaque yellow accumulation of secretion or epithelium. He also recommends that *follicular ulcers* on the inside of the lips and cheeks and tip of the tongue be treated by *sulphate of copper* applied once or twice a day (*Brit. Med. Journal,* March 13, 1868.)

In severe cases, where the breath becomes fetid, with the submaxillary glands large and swollen, the lips and gums tumid, the face flushed and swollen, and the fever intense, *chlorate of potash* must be freely given internally to the extent of *five* grains every four or six hours.

Besides the remedies already recommended, it will be found that the solution of the *pernitrate* of iron, internally, has a beneficial effect upon the sores. It is prescribed in the following formula (DUNGLISON):—

℞. Liq. Ferri. Pernitratis, gtt. xl.; Syrup. Aurant, fl. oz. ss.; Aquæ, fl. oz. vss.; *misce.* A fourth part may be given to a child three or four years of age four times a day.

In cases where parasitic vegetable productions abound (*parasitic thrush*), the application of a solution of *sulphite of soda* (fl. dr. i. to fl. oz. i. of water) removes the lesion in twenty-four hours (JENNER). The secretions of the mouth being acid, the salt is decomposed, and sulphurous acid is set free, which destroys the parasite. A change of air is often absolutely necessary to restore the patient to health; and *arsenic* and *iodine* are useful restorative agents in repairing, with good diet, the faulty nutrition of the child.

ABSCESS OF THE CHEEK.

LATIN EQ., *Abscessus buccarum;* FRENCH EQ., *Abcès de la joue;* GERMAN EQ., *Abscess der Wange;* ITALIAN EQ. *Ascesso della guancia.*

Definition.—*A limited collection of pus in the textures or region of the cheek.*

Pathology.—Such abscesses may be caused by blows on the side of the face, but are more often the result of irritation from carious teeth. In some cases the disease is confined to the *Antrum* of *Highmore.*

The **Symptoms** are acute pain in the cheek, with subsequent constant aching pain, and difficulty of mastication. There is considerable swelling of the cheek, sometimes to such an extent that the nostril becomes closed. the lachrymal duct obstructed, the eye extruded, and the teeth loosened. If unrelieved, the abscess may point externally, or it may burst into the mouth or into the nostril.

Treatment consists in making a free aperture as soon as pus is formed. All diseased teeth should be removed; and if the abscess becomes chronic, search should be made for necrosed bone. When the abscess is confined to the *antrum*, one of the molar teeth should be withdrawn, and a trocar passed up through the socket.

CANCRUM ORIS—*Syn.*, GANGRENOUS STOMATITIS.

LATIN EQ., *Gangræna oris* — Idem valet., *Stomatitis gangrænosa;* FRENCH EQ., *Gangrène de la bouche;* GERMAN EQ., *Noma*—Syn., *Wasserkrebs;* ITALIAN EQ., *Cancro della bocca.*

Definition.—*The more severe form of inflammation of the mouth.*

Pathology.—It occurs in children of debilitated habits, between *two* and *five* years of age especially.

A copious flow of saliva, a tumid appearance of a cheek, with fetor of breath, ought to suggest an examination of the mouth, when small vesicles of a greyish-red or even black appearance may be seen on the inside of the lips or on the tumid cheek.

These vesicles are surrounded by a red base, and swelling, surrounding hardness, heat, and pain increase. An ash-coloured eschar may then appear in the centre of the cheek, within the cavity of the mouth, surrounded by a glossy tumefaction of the parts; and on the inside of the cheek a hard indolent swelling. So rapid may be the spread of this destructive disease that in a few days the lips, cheeks, tonsils, palate, tongue, and even half the face may become gangrenous—the teeth falling from their sockets, a horribly fetid saliva and fluid flowing from the parts.

Treatment.—Analogous to hospital gangrene, an early recognition of the nature of the disease will suggest an efficient application of *strong nitric acid* to the slough. The mouth must be frequently syringed with a solution of carbolic acid in the proportion of half a drachm dissolved in a gallon of boiling water, and allowed to become warm or tepid.

Tonics and antiseptics must be freely given. Beef-tea, wine, brandy, quinine, and chlorate of potash are the remedies indicated.

RANULA.

LATIN EQ., *Ranula;* FRENCH EQ., *Grenouillette;* GERMAN EQ., *Ranula;* ITALIAN EQ., *Ranula.*

Definition.—*A semi-transparent fluctuating swelling situated under the tongue.*

Pathology.—The swelling is generally caused by an obstruction of the duct of the *sub-maxillary gland,* but it may also proceed from a similar condition of the *sub-lingual,* or from dilatation of a *bursa mucosa* over the *genio-hyoglossus* muscle.

There are great doubts entertained as to whether simple dilatation of a small duct can produce tumors of the very large size often attained to in these regions; and although it is not denied that the smaller tumors may be produced from duct-obstruction, it is probable that they are much more frequently actual new formations—in point of fact, cystic tumors. They appear as fluctuating more or less transparent livid blue swellings, situated under the tongue, but occasionally extending under the *sterno-mastoid* muscle for a very considerable distance below the jaw —quite half-way down the neck. The contained fluid of these cysts may be thin, colourless, and transparent; but it is more frequently thick, albuminous, and of a pale straw-colour, and very much resembling uncooked white of egg. In cases of an enlarged *bursa* the fluid is clear and serous, in which sometimes blood discs may be detected.

Treatment of all cystic tumors of the cheek and mouth is essentially the same—namely, to empty the contents, and to set up such an inflammatory process as will destroy the secreting surface, and so prevent a re-formation of the tumor. This end may be attained by injection of *iodine,* by the application of *nitric acid,* or *nitrate of silver,* or by removing a piece of the cyst wall; but in most cases the introduction of a seton will be found the most convenient, as it is also the surest method. There are, however, instances in which, in spite of all milder measures of treatment, these cysts will re-fill, and in such cases the practitioner may endeavour to remove the cyst entire.

SECTION II.—DISEASES OF THE TONGUE.

GLOSSITIS.

LATIN EQ., *Glossitis;* FRENCH EQ., *Glossite;* GERMAN EQ., *Zungenentzündung;* ITALIAN EQ., *Glossitide.*

Definition.—*Inflammation of the tongue.*

Pathology and Causes.—The tongue is liable to various forms of inflammation of its covering and of its substance generally, resulting in various forms of ulcers. The inflammation may occur spontaneously, or from occult atmospheric causes, which give rise to idiopathic inflammation of other organs; or it may result from acrid substances taken into the mouth, or from the specific action of mercury, or of scarlet fever, or of small-pox.

Symptoms.—There is generally great tumefaction from infiltration of serum, while fever, mental depression, and general weakness prevail, with pain and heat of the tongue, the colour of which is of a deeper red than usual. The swelling may be so great as to cause the tongue to project beyond the teeth, or even to project so far back as to cause dyspnœa. Dr. Graves relates a case of inflammation affecting one-half the tongue, the median line forming the boundary between the swollen and the healthy parts.

Treatment.—Active cathartics are generally of great service, and they are to be given as *enemata.* Blood must be taken directly from the tongue in such cases. Incisions along the superior surface of the inflamed organ, followed by the action of the vapour of hot water, may reduce the swelling and relieve congestion. The relief afforded by these measures is frequently almost instantaneous. In Dr. Graves's case, two or three applications of six leeches at a time to the inflamed parts produced a speedy decrease of the tumor.

If suffocation is imminent, *tracheotomy* or *laryngotomy* may be performed.

In the experience of the late Dr. Symonds, of Bristol, erythematous inflammation of the tongue will yield to a combination of *bismuth* in *glycerine,* such as *bismuth subnitratis,* gr. xx.; *glycerine,* ʒi.; *aquæ,* ℥vii. Of these make a lotion with which to wash the mouth and tongue.

Morbid sensibility of the tongue may be soothed by a weak solution of *bromide of potassium,* ʒss. ʒi. to ℥vi. of water, with which to wash the mouth (SYMONDS).

ULCER OF THE TONGUE.

LATIN EQ., *Ulcus;* FRENCH EQ., *Ulcère;* GERMAN EQ., *Geschwür;* ITALIAN EQ., *Ulcera.*

Definition.—*Inflammation, simple or specific, terminating in ulcers.*

Pathology.—Chronic ulceration of the tongue is the most frequent affection of this organ. It may occur as—(*a.*) *Simple ulceration;* (*b.*) *Syphilitic and mercurial ulceration;* (*c.*) *Cancerous ulceration.*

(*a.*) *Simple ulceration of the tongue* is in most instances caused by derangement of the digestive system. In young subjects the apthous patches already described may be noticed. In adult patients such ulcerations will generally be observed about the tip or edges of the organ. The remainder of the tongue will be furred, and there will be the general symptoms of dyspepsia. This simple ulceration may pass off under the use of suitable constitutional and dietetic remedies; but it may be also very obstinate, and resist every effort at local or general treatment.

(*b.*) *Syphilitic ulceration* may be primary, secondary, or tertiary.

When primary disease of the tongue occurs, it must be treated in the same manner as a primary chancre in other situations.

Secondary ulceration of the tongue is one of the earliest sequelæ of syphilis. There are two forms in which this ulceration appears:—(1.) As a simple denudation of epithelium, giving rise to a glossy psoriasis-like patch, which creeps from place to place, and which heals and breaks out again repeatedly. Accompanying it there are frequently similar patches on the mucous membrane lining the cheeks, and on the inner surface of the lips. (2.) Condylomatous patches on the tongue are not at all uncommon in secondary syphilis. They generally occur at the back part of the tongue, and are associated with similar patches on the tonsils and fauces. The epithelial denudation is mostly seen on the anterior part of the tongue: in both cases the ulcers are symmetrical; and this last symptom is a most valuable diagnostic sign of secondary syphilitic ulceration of the mouth and tongue.

Tertiary ulceration of the tongue may occur in the form of deep fissures, in a longitudinal direction, with enormous hypertrophy of the papillæ along the edges of the fissure; or as nodes,—the gummata of modern syphilographists.

These nodes occur as an inelastic induration on the upper surface of the tongue. Sometimes they are situated so far back as to require the laryngeal mirror to bring them into view. (Several such lesions are to be seen in the Museum of the Army Medical Department at Netley.)

These nodes frequently soften, burst, and leave a large sloughy ulcer, with ragged, thickened, and hardened edges.

Ulcers of the tongue resulting from the action of mercury are usually associated with similar ulcerations of the gums and mercurial fetor of the breath.

The **Treatment** of syphilitic ulcerations of the tongue varies, of course, according to the severity and form of the affection. In the secondary varieties local application of solutions of *chloride of zinc,* with washes of *chlorate* or *permanganate of potash,* are frequently sufficient; but sometimes the application of *solid nitrate of silver* is desirable.

In the tertiary forms of syphilitic ulceration, *solid nitrate of silver* must be applied daily, and large doses of *iodide of potassium* frequently administered.

In mercurial ulcers, constitutional treatment of a tonic character, disinfectant gargles, and generous diet are indicated. It may here be mentioned that in all cases of diseases of the tongue, all irritants, as spices and pepper, as well as smoking, sucking of lozenges, or any other measure calculated to increase the flow of saliva, must be strictly prohibited.

(*c.*) Concerning cancer of the tongue, the reader is referred to the subject of cancer.

ABSCESS OF THE TONGUE.

Latin Eq., *Abscessus;* French Eq., *Abcès;* German Eq., *Abscess;* Italian Eq., *Ascesso.*

Definition.—*A limited collection of pus in the substance of the tongue.*

Pathology.—Though rare, abscess of the tongue is sufficiently frequent to merit consideration, more especially as it is liable to be mistaken for *carcinoma.* It is generally seen as a tumor, varying in size from that of a pea to a plum, embedded in the deeper tissues of the organ.

It is generally semi-elastic to the touch, but it is not always that fluctuation can be detected. The growth is slow, and need not be preceded by any acute inflammation.

The diagnosis of these simple lesions from the more serious diseases of cancer may be made,—First, by the fact that cancer generally attacks one or other side of the tongue,—abscesses are almost invariably in the centre. Secondly, the pain is more localized in the case of an abscess. Thirdly, there is no glandular enlargement. In addition, the age of the patient, family history, and constitutional condition will assist in arriving at a right conclusion.

The nodes or gummata, previously mentioned as appearing in this region, will be differentiated from simple abscess by the history of the case and by their more superficial appearance.

The **Treatment** consists in simple incision.

HYPERTROPHY.

Latin Eq., *Hypertrophia;* French Eq., *Hypertrophie;* German Eq., *Hypertrophie;* Italian Eq., *Ipertrofia.*

Definition.—*Increase in the muscular substance of the tongue.*

Pathology.—Enlargement of the tongue is a condition occasionally seen as a chronic disease; and it is also rarely met with as a congenital affection. The same pathological problem is here involved as in other hypertrophies—namely, how far the enlargement is due to *hyperplasia.* (See subject of hypertrophy, vol. i.)

Surgical treatment, although not satisfactory, is of more use than medicine. Where, however, it is the result of chronic inflammation, *iodide of potassium* should be administered with perseverance.

VASCULAR TUMOR.

Latin Eq., *Tumor vasculosus;* French Eq., *Tumeur vasculaire;* German Eq., *Gefässgeschwul'st;* Italian Eq., *Tumore vascoloso.*

Definition.—*A tumor mainly composed of blood-vessels.*

Pathology.—Vascular tumor and nævus of the tongue are both fortunately of rare occurrence.

It is only necessary to remark that their treatment is difficult. Such growths in this region can neither be ligatured nor extirpated without considerable trouble; and any operation is likely to be followed by œdema, pain, or hæmorrhage.

TONGUE-TIE.

Latin Eq., *Lingua frenata;* French Eq., *Filet;* German Eq., *Anwachsung der Zunge;* Italian Eq., *Brevità del frenulo.*

Definition.—*A condition where the frænum linguæ extends to the very tip of the tongue, and so ties it down.*

Pathology.—This condition is very much less frequent than is generally supposed; and in most cases it exists only in the imagination of the child's mother. Hence it is seldom met with except amongst the ignorant classes. It is much better to overcome the prejudice than to pander to it. Should any operation be really required, snipping the frænum is at least harmless, though the method of performing it which is usually recommended in text-books is objected to by Mr. Holmes Coote. This distinguished surgeon says (Holmes's *System of Surgery,* vol. iv., page 215) that it must be "remembered that the artery of the frænum may proceed from the sublingual, and not from the ranine arteries; and that the rule given to keep the point of the scissors downwards towards the floor of the mouth is hazardous. It is better to use blunt-pointed scissors, and to cut as little as possible, and directly backwards."

Section III.—Diseases of the Fauces and Palate.

QUINSY—*Syn.,* CYNANCHE TONSILARIS.

Latin Eq., *Cynanche tonsillaris;* French Eq., *Esquinancie;* German Eq., *Angina tonsillaris*—Syn., *Parenchymatöse und phlegmonöse Entzündung des Rachens;* Italian Eq., *Angina tonsillare.*

Definition.—*Acute inflammation of the tonsils, which may or may not lead to suppuration.*

Pathology and Causes.—Exposure to cold is almost always the cause of the attack; but in some cases it appears to be due to constitutional disturbance. Whether the predisposing habit of the patient be rheumatic or gouty, as some believe, it is undoubtedly true that some people are much more liable to quinsy than others; and a patient who has had one attack rarely escapes a second, unless radical treatment has in the meantime been adopted. In adults enlarged tonsils commonly predispose to the affection.

Moreover, in persons subject to quinsy there is almost invariably disease of the follicles of the tonsils, so that the mucus, instead of being freely secreted, becomes hard and cheesy, and, blocking up the follicles, strongly predisposes to inflammation of the gland whenever the slightest cold is taken.

Spring and autumn are the periods of the year in which the affection most frequently appears. It is rare in children, and is seldom met with before puberty. It is also rare in the aged, and appears to be a disorder mainly confined to youth and middle life. According to the Mortality Returns of England and Wales, nearly 400 persons die annually from the effects of quinsy; but this may be due to an error of diagnosis—cases of death from scarlatina being probably returned under the head of quinsy. It is exceedingly improbable that a death has ever occurred from simple uncomplicated tonsillitis.

Symptoms.—Inflammation of the tonsils is usually preceded by some shivering and fever, which is succeeded in a few hours by the sensation of a sore throat. The symptoms now increase in severity and with great rapidity. The patient experiences great pain in deglutition, and on attempting to swallow, drink is sometimes ejected through the nostrils. There is a continual dull aching when the throat is at rest; the voice is altered, being thick and nasal, and the patient can hardly breathe except through his nose; he has ear-ache, and frequently is somewhat deaf. There is a constant flow of saliva from the half-open mouth; and there is a frequent desire to clear the throat of the viscid mucus which adheres to it. These symptoms, combined with feverishness and loss of appetite, increase in severity, until either resolution takes place, or pus having been formed, escapes. The degree of prostration which attends tonsillitis is usually out of all proportion to the severity of the local lesion. The attack generally subsides in a week or ten days, and rarely lasts a fortnight.

It is often very difficulty to examine the throat, because the patient is unable to open his mouth widely. Should a view be obtained, one or both tonsils will be seen to be red and swollen, and the passage through the fauces more or less completely blocked up. Although all the soft structures are swollen and œdematous, only one tonsil is generally affected at a time; but it is not uncommon for the inflammation as it subsides in one tonsil to attack the opposite side. The tongue is covered with a thick clammy fur, and the breath is very offensive. The sub-maxillary glands are usually sympathetically enlarged.

Treatment must depend entirely on the stage at which the disease comes under notice.

In the early period of an attack resolution may almost invariably be brought about by administration of *guaiacum*, in the form of lozenges. This remedy has been given in the form of mixture for many years, by those who believe that quinsy is due to a rheumatic diathesis; but it is probably by its local action that guaiacum is of service.

Dr. Mackenzie prescribes a lozenge containing three grains of the extract of guaiacum mixed with black currant paste, every two or three hours. If the approach of a quinsy be early recognized, and even when the tonsils are considerably enlarged, the *gum resin of guaiacum* is of great service, combined in the following formula:—

> ℞. *Magnes. Sulph.*, ʒvi.; solve in aquæ ℥viii.; *Adde Pulv. Guaiaci*, ʒiss.; *Pulv. Gum. Tragacanth Co.*, ℈ii.; *misce bene.* One-sixth part of this mixture may be given every four hours till the bowels are freely moved.

In addition to this, ice sucked constantly is often most grateful to the patient. In other cases, however, relief is experienced by holding hot water in the mouth, and by the inhalation of hot steam, combined with sedatives—such as *benzoin* or *conium.* Ice in lumps, or in iced drinks, or cold thick gruel, if ice cannot be obtained, often afford relief. A mixture of mucilage or gruel containing *nitrate of potash* or *borate of soda,* and a small quantity of *syrup of poppy, Batley's solution of opium,* or *prussic acid,* should be frequently but slowly swallowed in small quantities—in tea or table-spoonfuls; *the amount of narcotic ingredient being carefully regulated,* so that a definite quantity be consumed in a given time.

Externally, a mustard poultice should be first applied, and afterwards linseed poultices. The latter may be constantly repeated until the attack subsides—spongiopiline often answers even better than the linseed poultices, and is more cleanly.

If suppuration has commenced before the practitioner sees the case, great relief will be afforded by puncturing the abscess with a guarded knife: the incision should always be made towards the median line.

After the abscess has been relieved, the recovery of the patient is usually very rapid; but the prostration is sometimes considerable, and an abundance of nourishment and stimulants are required.

It has been already remarked that a patient who has once suffered from quinsy is likely to suffer again; and the question naturally arises as to what preventive treatment should be adopted? In those cases in which there is any chronic enlargement of the tonsils, excision should be performed; but in some cases the glands, though extensively diseased, do not remain enlarged when the acute inflammation has passed away. Under these circumstances the tonsil may be amputated during the attack of quinsy. This plan of treatment is commonly adopted in Germany on its own merits, without any question as to recurrent inflammation.

SLOUGHING SORE THROAT—*Syn.*, PUTRID SORE THROAT; CYNANCHE MALIGNA.

LATIN EQ., *Angina putris*—Idem valet, *Cynanche maligna;* FRENCH EQ., *Angina gangrêneuse*—Syn., *Angina maligne;* GERMAN EQ., *Angina maligna oder gangrænosa;* ITALIAN EQ., *Angina maligna.*

Definition.—*Ulceration of an acute form attacking the tonsils and rapidly running into sloughing of the fauces.*

Pathology and Symptoms.—The disease is a very grave—often a most intractable—affection, and as rapid as it is grave (POLLOCK).

The first symptoms are those of quinsy; but on examination at an early stage, the whole throat will be seen to be generally œdematous, of a dusky-red colour, and covered with patches of grey or yellowish membrane. As these sloughs spread there is a discharge of a most offensive character through the nose—the patient is unable to swallow, and all fluids are ejected through the nostrils. With the progress of the disease the sloughs separate, and with them the uvula, or large portions of the soft palate may also come away. If the patient recovers, swallowing is for a long time difficult, and the voice permanently nasal.

This form of sore throat is probably in all cases either the result of syphilis or dependent on scarlet fever. The symptoms are not always so serious as just described. It occasionally happens that after an incision into a suppurating tonsil the wound takes on a sloughy appearance.

Diagnosis.—The affection must be distinguished from malignant scarlet fever.

Treatment.—In all cases of sloughing sore throat a stimulating and tonic treatment must be pursued. Tincture of the *perchloride of iron* and *chlorate of potash* are the most suitable internal remedies. Disinfecting gargles of *carbolic acid* or *permanganate of potash* are not only agreeable to the patient, but very useful in checking the offensive discharges.

ENLARGED TONSILS.

LATIN EQ., *Tonsillæ intumescentes;* FRENCH EQ., *Hypertrophie des amygdales;* GERMAN EQ., *Hypertrophie der Tonsillen;* ITALIAN EQ., *Tonsille ipertrofiche.*

Definition.—*Hypertrophy of the tonsil, generally with induration.*

Pathology.—Hypertrophy of the tonsils is most common in early life; but the causes of this condition have not been satisfactorily ascertained. Rich and poor are alike subject to it, and probably a strumous diathesis is the strongest predisposent. In some cases it is probably due to a very slow interstitial inflammation. Enlargement of the tonsils occasionally occurs as a sequel of exanthematous fevers.

The development of the disease is usually unattended with pain; and attention is generally first drawn to the enlargement by the symptoms. These are, thickness of articulation and breathing, snoring in sleep, and sometimes deafness. On examination, the glands will be seen to be more or less hypertrophied—the mucous membrane covering them will be much thickened and unevenly pitted, from enlargement of the follicles. These follicles will sometimes be blocked up with cheesy-like matter.

Treatment.—Although enlarged tonsils may be the result of constitutional derangement, they in themselves probably also interfere with the proper development and health of the body. Thus, they constantly obstruct the free passage of air into the chest; they give rise to an unhealthy secretion in the mouth; they sometimes interfere with sleep; and, in the case of adults, they subject the patient to repeated attacks of quinsy, with its associated conditions of difficult deglutition and pain.

The importance of removing the diseased and redundant portions of the affected gland will be at once evident. Tonics, sea-bathing, and other constitutional means, though constantly recommended both by writers and practitioners, do not appear to exercise any beneficial effect in either diminishing or arresting the growth of tonsils predisposed to hypertrophy; nor are the applications of caustics of the slightest value, unless they are so strong as to destroy a large portion of the gland. It is better, therefore, to excise the enlarged tonsil in all cases when the respiration is at all affected, or where there is disease of the follicles predisposing to frequent attacks of inflammation and suppuration. The best method of removal is with a guillotine or wire écraseur. Dr. Mackenzie has lately invented a double guillotine, by which, in one operation, both tonsils are removed. This is a great desideratum in the case of children; as, after one tonsil has been removed, the practitioner has great difficulty in inducing his little patient to allow a second similar operation.

Hæmorrhage seldom happens after removal of the tonsils. Should it occur, however, ice will generally check it; and if this does not answer, a few tea-spoonfuls of a saturated solution of tannin (℥ss. in fl. oz. iij. of water) slowly sipped, will be certain to stop all bleeding.

ELONGATED UVULA.

Latin Eq., *Uva descendens;* French Eq., *Elongation de la luette;* German Eq., *Verlängerung des zäpfchens;* Italian Eq., *Ugola allungata.*

Definition.—*Relaxation or paralysis of the uvula, which becomes so elongated that its tip may touch or rest upon the surface of the tongue.*

Pathology and Causes.—Relaxation of the uvula is a most common result of inflammation of the throat. It is also met with among clergymen, officers of the Army and Navy, singers, and other persons who are obliged to make great vocal efforts, or to continue the use of their voice, even when suffering from catarrh. The condition generally depends on simple relaxation of the mucous membrane, but the *azygos uvulæ* muscle may also form some part of the increased growth.

The **Symptoms** of elongation of the uvula are always inconvenient, and sometimes even serious. The patient may experience nothing more than a slight tickling cough, accompanied by the sensation of a foreign body, and these symptoms will only be inconveniently felt at intervals. In other cases, the cough will be constant and troublesome, and followed by frequent expectoration of little pellets of mucus. The constant tickling of the fauces and pharynx may produce a sensation of nausea, and occasionally vomiting takes place on waking in the morning. On lying down at night the symptoms are often increased, and the patient may suddenly awake with spasm of the glottis, brought on by the elongated uvula.

These constant attacks bring the patient into an extremely nervous state; and the want of sleep and constant cough often give rise to so much emaciation and weakness, that at first sight the patient appears to be the subject of phthisis or other serious disease.

On examination of the throat the cause of these symptoms will be at once evident; but it is important to observe the length of the uvula in relation to the level of the back of the tongue. The importance of an enlongated uvula entirely depends on the relation of the various parts at the back of the mouth; for that which in one patient would be but slight and harmless relaxation, in another would give rise to serious and distressing symptoms. Again, when a patient opens his mouth, he always at the same time takes an inspiration, so that the uvula being drawn up, and to a certain extent invaginated, it may appear much shorter than it really is. In examining the throat, therefore, it is a good plan to tell the patient to EXspire, so that the real length of the uvula may be ascertained. The larynx of patients suffering from this condition is generally more or less congested.

The **Treatment** of relaxed uvula must depend on the degree of relaxation present and the previous duration of the affection. Slight and recent cases often get well by the persevering use of astringents. Gargles, such as *tannin*, *rhatany*, and *perchloride of iron*, lozenges, and local applications may all be used with advantage; and, indeed, it is found convenient to brace up the mucous membrane by employing these different methods at the same time. When the uvula has been considerably relaxed for some months the elongated portion should be snipped off.

While it may be said that hardly any slight affection of the throat produces such serious symptoms as elongation of the uvula, it is equally true that there is no slight operation that gives such complete and permanent relief as removal of the elongated extremity. Should any hæmorrhage follow the operation, treatment similar to that recommended after excision of the tonsil must be adopted.

SECTION IV.—DISEASES OF THE PHARYNX.

PHARYNGITIS.

LATIN EQ., *Pharyngitis;* FRENCH EQ., *Pharyngite;* GERMAN EQ., *Schlundentzündung;* ITALIAN EQ., *Faringitide.*

Definition.—*Inflammation of the textures of the pharynx.*

Pathology.—Inflammation of the pharynx may be either *acute, subacute,* or *chronic.*

Acute pharyngitis is generally met with in association with a similar condition of the fauces, and is but very rarely seen to exist alone.

The form of inflammation is most frequently of an œdematous character, and it occasionally extends to the larynx.

The **Symptoms** are, great pain and difficulty of swallowing, with more or less dyspnœa if the swelling extends over the wind-pipe.

When the larynx is affected the symptoms are those of œdematous laryngitis. With the laryngoscope, that part of the larynx which can be seen will appear red and œdematous.

Prognosis.—The opinion given should always be guarded; but the disease is seldom serious unless it extends also to the larynx.

Treatment should consist in hot steam inhalations of a sedative character, poultices externally, and scarification if œdema takes place. In those cases in which the uvula is much swollen and infiltrated with serum it should be at once removed.

Sub-acute Pharyngitis may occur either as an early stage of the acute form or as a less serious inflammatory affection. In this condition it is the mucous membrane alone, not the areolar tissue, which is inflamed. The *symptoms* are less severe than in acute pharyngitis; and *treatment* consists in the application of astringent solutions, and the use of astringent lozenges. The frequent sucking of small pieces of ice is of great service in reducing the hyperæmic condition.

Chronic Pharyngitis is of three forms,—

(1.) *Granular*, (2.) *Follicular*, and (3.) *Herpetic.*

Symptoms.—(1.) In the *granular* form there is uneasiness and a certain amount of soreness in swallowing, especially if hot drinks or piquant food are taken.

The mucous membrane of the pharynx is seen to be red, rough, and uneven in appearance.

(2.) In *follicular pharyngitis* there is not often pain, but there is a constant desire to clear the throat of a foreign body. On examination, the mucous membrane will be seen to be studded here and there with white raised spots, similar in appearance to those observed in follicular disease of the tonsils. These spots occasionally burst and discharge a white sebaceous matter.

(3.) In the so-called *herpetic form of pharyngitis*, the appearance presented is that of raised solid papules of a redder colour than the mucous membrane, covering the posterior wall of the pharynx. This condition has nothing of the vesicular character which in England attaches to the term herpetic. The symptoms are much the same as that of the follicular form, and all these varieties are often associated with, and probably sometimes caused by, relaxation of the uvula.

Treatment.—*Chronic pharyngitis* is always difficult to cure, and the herpetic variety is, above all, very obstinate. *Tonics*, such as *iron*, *quinine*, and *strychnia* are mainly indicated. Locally, the treatment must vary according to the especial character of the disease. In the *granular* form, astringent solutions, such as *zinc* and *iron*, may be applied either with the brush or in the form of an atomized inhalation. Gargles are of no use when the disease is situated behind the anterior pillars of the fauces; but lozenges are most serviceable in all forms of pharyngeal disease. Those of *tannin*, *rhatany*, and *kino* are very beneficial in granular pharyngitis.

In *follicular pharyngitis* the spots should be scraped, the secretion emptied, and solid *nitrate of silver* applied to each diseased follicle.

Lozenges of *chlorate of potash* and of *bitartrate of potash* are useful in stimulating the follicular secretion to a healthy condition.

In *herpetic pharyngitis* the raised papules must be destroyed. The best application for this purpose is a paste recommended by Dr. Mackenzie. It consists of equal parts of *caustic soda* and *unslaked lime*, which is mixed with water, as required, into a creamy paste, and should be applied to each papule with a finely-pointed glass or aluminium rod. The caustic effect of this application is instantaneous. The patient therefore should be directed always to wash his mouth with water immediately after the application, so as to prevent any other part of the throat being burnt by contact with the caustic.

In all cases of pharyngeal disease, every form of spice, pepper, mustard, and piquant food should be strictly prohibited, and all fluids should be taken at a moderate temperature.

ULCER OF PHARYNX.

LATIN EQ., *Ulcus;* FRENCH EQ., *Ulcère;* GERMAN EQ., *Geschwür;* ITALIAN EQ., *Ulcera.*

Definition.—*Inflammation of the pharynx, resulting in loss of substance, in the form of an ulcer.*

Pathology.—*Ulceration of the pharynx* is generally the result of hereditary or acquired syphilis; but occasionally it appears to be due to a low strumous inflammation. Two forms are recognized by the College of Physicians, namely—(a.) *Superficial ulcer;* (b.) *Perforating ulcer.*

The **Symptoms** are, offensive discharge from the nose and mouth, with a constant desire to expectorate the secretion covering the ulcerated surface. There is more or less difficulty of swallowing, but seldom much pain. On opening the mouth the disease is readily recognized.

It generally attacks the posterior wall of the pharynx, but it may also involve the velum and tonsils. Not unfrequently there is also ulceration of the posterior nares.

In the syphilitic cases the *treatment* consists in the daily application of solid nitrate of silver to the ulcerated surface, and the free internal administration of *iodide of potassium.* In the case of children, *iodide of iron* and *cod-liver oil* are also indicated.

ABSCESS OF THE PHARYNX.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abcès;* GERMAN EQ., *Abscess;* ITALIAN EQ., *Ascesso.*

Definition.—*Inflammation of the pharynx, resulting in a limited collection of pus, in the form of an abscess.*

Pathology.—*Abscess of the pharynx* is a most serious affection, and is generally caused by disease of the vertebræ, or of one of the cartilages of the larynx. Diffuse suppuration of the pharynx is fortunately a very rare occurrence.

The **Symptoms** are, difficulty and pain in swallowing. If the abscess burst into the larynx, the usual symptoms of food "going the wrong way" will be experienced. In such cases it may be advisable to feed the patient by means of a tube passed beyond the seat of fistulous communication. No other method of treatment is of any benefit; and the efforts of the practitioner must be directed to relieving the symptoms of a distressing and almost invariably fatal disease.

SLOUGHING OF THE PHARYNX.

LATIN EQ., *Sphacelus;* FRENCH EQ., *Escharre;* GERMAN EQ., *Gangrän;* ITALIAN EQ., *Gangrena.*

Definition.—*Inflammation of the pharynx, resulting in loss of substance by death of portion in the form of a slough.*

Pathology.—*Sloughing of the pharynx* is usually the accompaniment of syphilitic ulceration.

It is occasionally found as a complication of inflammation of the fauces accompanying typhus or enteric fever. The *treatment* consists in the administration of tonics, stimulants, and nourishment. Locally, antiseptic applications, as *carbolic acid* and *permanganate of potash,* are of service.

ADHESION OF THE SOFT PALATE.

LATIN EQ., *Palatum molle adhærens;* FRENCH EQ., *Adhérence du palais;* GERMAN EQ., *Verwachsung des weichen Gaumens;* ITALIAN EQ., *Aderenza del palato molle.*

Definition.—*Union, by adhesive inflammation, of the soft parts of the palate and pharynx.*

Pathology.—*Adhesion of the soft palate to the pharynx* is an occasional sequel of syphilitic ulceration of the pharynx.

When cicatrization takes place there is great displacement of the soft parts, and adhesion of the soft palate to the wall may occur.

Under these circumstances the sense of smell is much impaired, and the patient experiences great difficulty in clearing the nose.

Treatment is not of much use, as the tissues in these syphilitic cases are exceedingly prone to contract, and thus the passage to the nose becomes again obstructed.

DILATATION OF THE PHARYNX.

LATIN EQ., *Dilatatio;* FRENCH EQ., *Dilatation;* GERMAN EQ., *Erwiterung;* ITALIAN EQ., *Dilatazione.*

Definition.—*Expansion of the walls, or of a portion of the wall, into the form of a pouch.*

Pathology.—*Dilatation of the pharynx* is occasionally noticed. It usually happens that a pouch is formed in which the food collects. This gives rise to dysphagia, and subsequently to vomiting of the obstructed food. Dilatation throughout the entire length of the pharynx is rare.

Treatment.—Where the exact situation of the obstruction is ascertained, the patient should be recommended to press externally against the pouch, by which means the food is often passed into the œsophagus.

SECTION V.—TREATMENT OF DISEASES OF LARYNX AND PHARYNX BY THE USE OF ATOMIZED FLUIDS.

The treatment of diseases of the *pharynx* and of the *larynx* by the use of *atomized fluids*, in which medicated vapour and gases are brought to act upon the parts, has recently been used with advantage, since better appliances have been devised for using such remedial agents. The aim of such instruments is to atomize or pulverize, or very minutely to

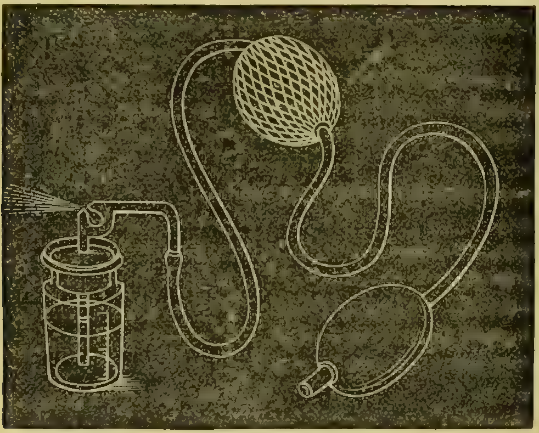

Fig. 54.

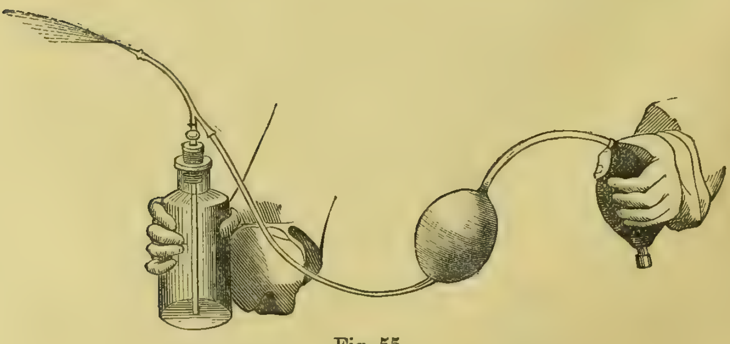

Fig. 55.

divide the fluid, by causing it to be thrown as a fine shower or spray, so as to be inhaled as such by the patient.

Dr. Andrew Clark's hand-balls (fig. 54), fitted with Bergoon's tubes and Maunder's atomizer, constitute an efficient and useful arrangement for applying fluids in this way up the nostrils, to the back of the throat, or over the entrance to the larynx (Da Costa).

Dr. Richardson's well-known instrument (fig. 55), for the production of local anæsthesia is also an excellent atomizer of fluid, and may be used for a similar purpose.

Where it is desirable to make the topical application directly upon the diseased part, these are the most simple instruments that can be used without loss of time by the physician; but where *inhalation* simply of vapour or heated air variously medicated is all that is required, nothing can be better than that described at p. 739, used by Dr. Fergus, of Glasgow.

Siegle's apparatus, supplied by Krohne & Sesemann, of 241 Whitechapel Road, and 8 Duke Street, Manchester Square, London, as well as Dr. Morell-Mackenzie's eclectic inhaler (page 705, *ante*), are each as useful and valuable instruments for inhalation.

On this most useful mode of treatment Dr. Da Costa makes the following observations:—

"Inhalations by means of atomized fluids are an unquestionable addition to our therapeutic means; but they are nothing but an addition, and not a substitute for all other treatment.

"In most acute diseases of the larynx, and still more so in acute disorders of the lungs, their value, save in so far as those of water may tend to relieve the sense of distress, &c., and aid expectoration, is very doubtful; though in some acute affections, such as in œdema of the glottis and in croup, medicated inhalations have strong claims to consideration.

"In certain chronic morbid states of the larynx, particularly those of a catarrhal kind, and in chronic bronchitis, they have proved themselves of great value.

"In the earlier stages of phthisis, too, they may be of decided advantage; and at any stage they may be a valuable aid in treating the symptoms of this malady.

"Their influence on such affections as hooping-cough and asthma is not satisfactorily proved.

"They furnish a decided and unexpected augmentation of our resources in the treatment of pulmonary hæmorrhage.

"They require care in their employment; and in acute affections we should consider whether, as they have to be used frequently to be of service, the patient's strength justifies the disturbance or the annoyance their frequent use may cause.

"In any case, to be of service, they ought to be carried on as a treatment with a distinct object, and not intermittingly or spasmodically resorted to."

Table of Doses for Inhalation (Da Costa).

Alum, 10 *to* 20 *Grains.*—In this dose, it is suitable to chronic catarrhal affections of pharynx and air-tubes, particularly in bronchial affections with excessive secretion, when, as in most inflammatory conditions of the respiratory mucous membrane, it may be advantageously united with opium. In rather larger doses, 30 grains to the ounce, it is useful in pulmonary hæmorrhage. As an astringent it is generally more of a sedative, and more suited to conditions of irritation than tannin (FIEBER).

Tannin, 1 *to* 20 *Grains.*—Useful for the same affections as alum. Employed in cases of laryngeal ulceration and excrescences, in œdema of the glottis (TROUSSEAU), and in croup. Here, as well as in pulmonary hæmorrhages, it is used in large doses. In ordinary cases of laryngeal or bronchial disease, begin with a small dose—five grains to one fluid ounce of water. If the remedy occasion much heat and dryness, it is not be employed.

Iron (perchloride of), $\frac{1}{8}$ *to* 2 *Grains.*—In earlier stages of phthisis. In chronic pharyngitis or laryngitis it may be used stronger—three grains to one fluid ounce of water. As a weak inhalation it is useful in hysterical aphonia. Of greatest strength it is useful in pulmonary hæmorrhage—2 to 10 grains to the ounce. The lactate, citrate, or phosphate may, in ordinary cases in which we wish a non-astringent salt of iron, be also used, though they are not, on the whole, as available as the chloride.

Nitrate of Silver, 1 *to* 10 *Grains.*—Is useful in ulcerations of pharynx and larynx, and in follicular pharyngitis. A face-shield is always to be used. Ten grains to the ounce only to be used in cases of ulceration.

Sulphate of Zinc, 1 *to* 6 *Grains.*—In bronchial catarrh with excessive secretion. In aphonia connected with chronic laryngeal catarrh.

Chloride of Sodium, 5 *to* 20 *Grains.*—Promotes expectoration and diminishes sputa; employed in phthisis.

Chlorinated Soda (*Liquor Sodæ Chlorinatæ*), $\frac{1}{2}$ *to* 1 *Drachm.*—In bronchitis with offensive and copious expectoration; in phthisis.

Muriate of Ammonia 10 *to* 20 *Grains.*—In laryngeal and bronchial catarrh, acute as well as chronic. To promote expectoration; also in capillary bronchitis. The dose best borne is not above 10 grains to the ounce.

Opium (watery extract of), $\frac{1}{4}$ *to* $\frac{1}{2}$ *a Grain.*—In irritative coughs, and as an adjunct to allay irritation. Also for its constitutional effects. Dose of *tincture of opium*, 3 to 10 drops. *Acetate of morphia*, one-twelfth to one-eighth of a grain has been administered; but large doses require much caution.

Conium (fluid extract of), 3 *to* 8 *Minims.*—Irritative cough; asthma; feeling of irritation in larynx.

Hyoscyamus (fluid extract of), 3 *to* 10 *Minims.*—Spasmodic coughs; hooping-cough. One-half grain of the extract, gradually increased; or the tincture may be employed.

Cannabis Indica (tincture of), 5 *to* 10 *Minims.*—In spasmodic coughs; phthisis.

Iodine Liq. (*Iodinii Compos.*), 2 *to* 15 *Minims.*—In chronic bronchitis; in phthisis.

Arsenic (*Liq. Potass. Arsenit.*), 1 *to* 20 *Minims.*—Nervous asthma (TROUSSEAU).

Tar Water, 1 *to* 2 *Drachms* of officinal solution.—In offensive secretions from bronchial tubes; in tuberculosis; as an antiseptic in gangrene of lungs.

Turpentine, 1 to 2 Minims.—In chronic bronchitis with offensive secretions; in bronchorrhœa; in gangrene of lungs.

Lime Water, used of officinal strength, or stronger.—In diphtheria; in membranous croup.

Water, Distilled.—Cold, in pulmonary hæmorrhage. Warm water in asthma; in croup; in bronchitis.

For various other modes of applying topical applications to the larynx, the student is referred to Dr. Morell-Mackenzie's work *On the Laryngoscope,* third edition.

SECTION VI.—DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

LATIN EQ., *Œsophagitis;* FRENCH EQ., *Œsophagite;* GERMAN EQ., *Entzündung der Speiseröhre;* ITALIAN EQ., *Esofagitide.*

Definition.—*Inflammation of the textures of the Œsophagus.*

Pathology.—Inflammation of the œsophagus is a rare disease, for morbid poisons seem to have little influence over this portion of the alimentary canal, and atmospheric vicissitudes are in like manner seldom followed by inflammatory affections of this part. The most frequent causes of inflammation of the œsophagus are, accidentally drinking boiling water; swallowing corrosive liquids, as the mineral acids; and wounds, most commonly inflicted in the act of committing suicide. Children a few days old are sometimes affected with slight inflammatory affections of the œsophagus.

Inflammation of the mucous membrane of the œsophagus is characterized by a deep redness of the part, generally terminating by resolution, but occasionally followed by separation of the cuticle. Lymph may be thrown out. In new-born children points of lymph are often found lying on the mucous membrane of the œsophagus, being apparently an extension of the thrush affecting the mouth and pharynx. Andral has seen, in a girl twelve years old, lymph thrown out after the manner of broad bands in the pharynx, œsophagus, and stomach. After puberty this form of inflammation is still more rare, but there are some few instances. Cruveilhier says that he found among the preparations of Dupuytren a very remarkable example of inflammation of the œsophagus, terminating in the formation of a false membrane, which coated this canal throughout its whole length. Dr. Abercrombie also gives the case of a gentleman, aged twenty-six, who caught cold, and died in about three weeks. The whole of the pharynx was covered by a loose adventitious membrane, which extended over the epiglottis, and portions of it were found lying in small irregular masses within the larynx at the upper part. A similar membrane was traced through the whole extent of the inner surface of the œsophagus quite to the cardiac orifice.

Besides lymph being thrown out, the mucous membrane of the œsophagus may also ulcerate, especially as a result of irritant poisons.

These ulcers in general form on the anterior portion of the œsophagus; and by continued extension they at last penetrate the posterior surface of the larynx, so that the patient often dies suffocated from the escape of food into the lungs. Occasionally the ulceration takes place from without inwards. The cicatrices are very apt to induce stricture. In cases of poisoning with the mineral acids, the whole œsophageal canal may become constricted and narrowed, and the mucous membrane puckered up and contracted, so as greatly to diminish the calibre of the canal generally. More commonly the stricture is partial, one circular muscular fibre perhaps having been abnormally contracted, and in this state bound down by adhesive inflammation, diminishing the diameter of the canal at that part to at least one-half. Dr. Bailie mentions a case in which, from this cause, the diameter of the œsophagus was so reduced as hardly to allow a garden pea to pass; yet in all other respects the œsophagus was healthy.

Symptoms.—The symptoms of *œsophagitis* are almost entirely local, and consist principally of pain, of dysphagia, of the expectoration of a thick viscid mucus, and perhaps of vomiting. Emaciation follows the loss of nutrition, and the patient ultimately dies from inanition. Stricture may be induced by the careless introduction of a probang.

Diagnosis.—The diseases with which it may be confounded are similar states of the stomach; and the diagnosis in these cases is often difficult and perplexing. Stricture may be confounded with the spasmodic affections caused by an irritated state of the lung or trachea.

Prognosis.—Simple *œsophagitis* is probably often recovered from, as is seen after wounds of the throat partially dividing the œsophagus; but the chronic forms of inflammation probably often lay the foundation of disease leading to the ultimate death of the patient. Ulceration extending into the thoracic or pericardial cavity has been in all cases fatal.

Treatment.—The treatment of *œsophagitis* is by small local bleedings, by warm cataplasms to the neck, and by moderately acting on the bowels. In the treatment of the more chronic forms some sedative is essential. The use of the probang must be left to the discretion of the practitioner. There is always some danger, in its use, of rupturing the canal, or of causing an ulcer. When the case is hopeless, from the small quantity of aliment which reaches the stomach, life may yet be prolonged by enemata of soups, milk, egg, wine, or other nutritious fluid matters.

SECTION VII.—RELATION OF THE ABDOMINAL VISCERA TO THE WALLS OF THE ABDOMEN.

The regions into which the abdomen is usually divided by lines are like those of the thorax, already described, quite arbitrary; and figs. 7, 8, 9, already given (p. 480), are referred to here; likewise the description given at p. 479, as to the fixed skeleton points which determine the direction of these lines, and the regions mapped out by them.

The vertical lines which have referrence to the abdomen are five in number, and run as follow:—(1.) From the insertion of Poupart's ligament in the external tubercle of the pubes to the acromial extremity of the clavicle (right and left side); (2.) From the posterior boundary of the axilla (the inferior edge of *latissimus dorsi*) to that point of the crest of the ileum on which it falls vertically, fig. 8 (right and left side); (3.) Along the spinous processes of the vertebræ from the sacrum to the nape of the neck.

The transverse lines are four in number, and indicate horizontal planes as follow:—(1.) On the point of the xyphoid cartilage; (2.) On the last short ribs; (3.) On the anterior superior spinous process of the ileum on each side; (4.) On the upper margin of the *os pubis*. These three horizontal and five vertical bands map out the abdominal walls into *thirteen* regions, of which five are anterior, four are lateral (two on each side), and four are posterior. They are named as follow:—*Anterior regions* are *epigastric* (No. 4 on the figures); *umbilical* (No. 5); *hypogastric* (No. 6); right and left *inguinal* (No. 11). The *lateral regions* comprehend the right and left *hypochondriac* (No. 9); the right and left iliac (No. 10). The *posterior regions* embrace the *inferior dorsal* on the right and left (No. 15); the right and left *lumbar* (No. 16).

The best idea of the contents of these regions is obtained by defining first the limits of the *liver*. It fills the right *hypochondriac* region (No. 9, figs. 7 and 8), filling up the concavity of the diaphragm; and it is almost completely concealed by the arch of the ribs. A part of the left lobe projects into the *epigastric* region (No. 4, figs. 7 and 8) and *left hypochondriac*. It also projects upwards into the *infra-axillary* region (No. 8, figs. 7 and 8) of the thorax, where it is separated from the thoracic wall by the thin lower margin of the right lung. Its upper margin in this space is on a line nearly with the level of the nipple, about the fifth intercostal space—less frequently beneath the fifth rib. In the perpendicular axillary line its margin is about the *seventh* intercostal space—more seldom under the seventh rib: close to the vertebral column its margin is in the tenth intercostal space—less frequently in the ninth (FRERICHS). At the median line the upper boundary of the liver cannot usually be distinguished from the lower margin of the heart. It is best made out by drawing a straight line from the point of contact of the right margin of the cardiac dulness with the upper boundary of the liver to the apex of the cardiac dulness on the left (CONRADI, FRERICHS, vol. i., p. 30).

Percussion of the liver after a meal is to be avoided; and any obstinate constipation which may be present must be removed before percussion, by means of free purgation, and any accumulations of gas must also be got rid of.

In the epigastric and left hypochondriac regions lies the stomach. The umbilical region is crossed by the transverse colon, passing from right to left a little above the umbilicus.

The convolutions of the jejunum and ileum occupy the umbilical and hypogastric regions. The large intestines surrounding the convolutions of the lesser intestines occupy the iliac and lumbar regions on each side. The kidneys are equally shared between the *infra-scapular* (No. 14, fig. 11) and the inferior dorsal regions (No. 15, fig. 11). The spleen in its greater bulk is in the same region on the left side.

SECTION VIII.—METHODS OF EXPLORING THE ABDOMEN.

These are principally three—namely, *inspection, manual examination* (*palpation*), and *percussion.*

Inspection furnishes information relative to *size, form,* and *movement;* and such information ought always to be acquired when the chest, as well as the abdomen, are both exposed simultaneously in a good light, the patient being protected from cold by a previous regulation of temperature in a room suited for the purpose of such an examination. The eyes of the patient ought to be directed away from the examiner.

Palpation furnishes information relative to position, size, consistence, elasticity, spontaneous movement, or mobility, and the presence of vibrations which may reach the surface. Care should be taken that the hand, when applied, should not be cold.

Percussion furnishes information relative to the comparative solidity of regions, and thereby indicates the kind of organ immediately below the seat of percussion.

The chief objects to be held in view in exploration of the abdomen are the following:—

To ascertain—(1.) Its form and size; (2.) Its degree of tension or solidity; (3.) Its temperature; (4.) Sensibility or tenderness over any part; (5.) The presence or absence of tumor in or amongst the viscera; (6.) The presence or absence of fluids in the peritoneal sac; (7.) The nature and extent of the intestinal contents.

SECTION IX.—DISEASES OF THE STOMACH.

GASTRITIS.

LATIN EQ., *Inflammatio;* FRENCH EQ., *Gastrite;* GERMAN EQ., *Magenentzündung;* ITALIAN EQ., *Gastritide.*

Definition.—*Forms of Inflammation tending to exudations and destruction of parts, or condensations of tissue, especially about the pyloric opening.*

Pathology.—*Idiopathic gastritis* is an exceedingly rare disease. When inflammation of the stomach does occur, it is generally the consequence of direct injury from irritant or corrosive poisons. Dr. Jones has never met with a case of acute idiopathic gastritis. Louis examined five hundred bodies without finding a single instance. Andral, however, relates several cases in his *Clinique Medicale;* and it is stated by Dr. Robert Williams, that during the whole of the Peninsular war, not more than six cases were reported among the troops, although exposed to every species of privation, and addicted as they were to its most efficient exciting cause—the use of spirits in every form in which alcohol could be got to drink.

The difficulty of exciting acute inflammation in the stomach is well shown by experiments upon animals, and by the often long escape of the polyphagist, who swallows knives and watches, and all sorts of heterogeneous things; and of the Indian who passed many times daily a blunt sword into his stomach with impunity, till at last its coats were pierced, and he died. The stomach, also, we find, will bear tea or coffee of an almost boiling temperature, followed perhaps shortly afterwards by a quantity of ice. One of the persons resident at the Eddystone Lighthouse at the time it was burnt, in 1755, swallowed a quantity of molten lead, which accidentally dropped into his mouth when looking from below upwards to observe the progress of the fire. But even after this intensely hot substance had passed into his stomach he lived several days, having been taken to the Plymouth Hospital, where he was attended by Dr. Sprey, who describes the case in the *Transactions* of the Royal Society. His attendants hardly believed his story possible; but on examining him after death, a lump of lead, weighing 7 oz. 5 drs. 8 grs. was taken from the stomach.

The simplest and most frequent form of inflammation of the stomach is that which is brought about by excess in eating or drinking, especially of alcoholic drinks. It is characterized by active congestion and an excessive secretion of mucus in the stomach—a condition which Dr. Jones describes under the name of

Gastric catarrh. Dr. H. Jones thus describes its phenomena:—

> "It occurs under the same influences as catarrh of the air-passages or conjunctivitis, and often either co-exists with these affections or succeeds them. Its anatomical characters are, distension of the capillaries, and abundant secretion of an extremely tenacious, clear, whitish bile or blood-tinged mucus. At the commencement of the disease, or during exacerbations, the hyperæmia is a marked feature; but the presence of abnormal mucus is a more constantly observed and certain character. Congestion of a passive kind powerfully predisposes to this disease, and is commonly conjoined with it. Watery, slightly mucous, feebly acid, or neutral fluids are secreted, and often ejected, constituting pyrosis. It does not tend to the destruction of the glandular tubes" (*Pathological and Clinical Observations respecting the Stomach*, p. 70).

Gastric catarrh may be either *acute* or *chronic.* In the acute form the mucous surface is reddened in spots by a fine injection, its tissue relaxed, and its surface covered by a layer of tough mucus. The following are the conditions under which the lesion is apt to be developed (NIEMEYER):—

(1.) In *fever patients*, the secretions of the stomach being diminished; and hence the diet ought to be adapted to such diminished secretion, otherwise catarrh is apt to occur. (2.) In debilitated and badly nourished persons. (3.) Excess in drinking spirits. (4.) One attack predisposes to others.

The exciting causes are—

(1.) Overloading the stomach with too much food, when symptoms of acute catarrh generally appear on the day following.

(2.) The use of food difficult of digestion.

(3.) The use of substances as food which have already begun to decompose before entering the stomach, such as spoiled meat, new beer, or sour beer.

(4.) Irritation from hot or cold substances, or from alcohol or spices.

(5.) Use of substances which weaken the action of the gastric juice, and retard the movements of the stomach. Alcohol acts injuriously in this way, so does opium, and smoking tobacco before meals.

(6.) The effects of cold.

There is a comparatively rare form of inflammation, described by Dr. Budd, in which coagulable lymph is effused into the submucous areolar coat, and, hardening and contracting, forms a dense gristly mass, binding the mucous membrane to the parts beneath. Round the pyloric orifice such a morbid state often acts as a permanent stricture. It is a condition almost invariably the result of spirit-drinking, and seldom occurs before the age of forty. (*Fibroid degeneration.*)

The organic diseases and functional disorders of the stomach have been mainly elucidated in this country by the researches especially of Drs. Budd, Handfield Jones, Chambers, Leared, and Brinton. Nevertheless, the morbid changes which are known to occur in it are still but very imperfectly connected with the expression of clinical phenomena. "There is no part of the body," says Dr. Chambers, "of which we hear so much from our patients, and are able to communicate so little knowledge in return, as about the stomach; and truly," he also observes, "it is an ill-used viscus—flattered in metaphor and insulted in fact." It has been regarded as a mere bag, a mere mill, or a mere chemical laboratory for the solution of substances: yet still all such similes and metaphors, as Hunter remarked, explain nothing; and he wisely insisted that it is a viscus *sui generis,* with definite functions to perform; in short, a "stomach is a stomach." Its morbid states are similarly peculiar to itself and to its functions.

The morbid states of the stomach, resulting out of forms of inflammation, and ascertained after death to have existed during life, and in some measure to have been expressed by certain symptoms, may be enumerated as follow:—(a.) *Softening of tissue;* (b.) *Glandular degeneration of the proper mucous substance;* (c.) *Congestion.*

(a.) *Softening of the Stomach.*—It was first announced by Hunter, and subsequently confirmed by the experiments of Spallanzani, Wilson Philip, and Carswell, that the stomach under certain conditions as to temperature and properties of the gastric juice, especially at death, and immediately after that event, may be dissolved or digested by the secretions poured forth from its own secreting glands. In certain diseases, also, of a catarrhal kind, it has since been ascertained that potent gastric juice is sometimes secreted by the empty stomach, or that lactic acid, being freely generated from the saccharine principles of food, forms with the mucous membrane an efficient digesting mixture; and not unfrequently a softening of the stomach may be predicted with tolerable certainty by a peculiar train of symptoms, and resulting from the presence of free gastric juice, or of a digesting acid, in the otherwise healthy stomach (BUDD). Dr. John Gairdner, of Edinburgh, however, so long ago as 1824, inculcated a similar doctrine. He observed, in a numerous series of cases of children, that a peculiar action of the gastric and intestinal mucous membrane, analogous to inflammation, weakened the texture of the organ, and rendered it morbidly susceptible to the action of the gastric juice during life (*Edin. Med.-Chir. Trans.*, vol. i. p. 311). It was in opposition to this view that Carswell performed his experiments, an account of which he published in 1838; so that the testimony they undoubtedly afford regarding *post-mortem softening*, as the only kind of softening, must be received with qualifications. The observations of Dr. John Gairdner and Dr. Budd evidently show that the probability of softening may be predicted during life, although Dr. Budd is of opinion that the softening does not take place till after death. A similar opinion is expressed by Andral. The diseases in which it is so apt to occur are those in which there is much cerebral disturbance and increasing debility and emaciation before death, and from such diseases as *typhoid fever*, *cancer of the uterus*, *or peritonitis*,—in infants who die of *tubercular hydrocephalus;* in deaths from exhaustion from inflammatory diseases of the brain, when vomiting is a constant symptom; in persons who die from phthisis and from ulcer of the stomach. The symptoms from which the softening may be predicted are, that when, along with any of these diseases, there is much disorder of the stomach, such as pain and tenderness at the epigastrium, loss of appetite, thirst, frequent vomiting of acid fluids, and nausea, the lesion may be expected to be found after death. In the common form, as Hunter described it, the mucous membrane towards the splenic end is thin, and for the most part stained by hæmatine, very slippery, and appearing as a dark film gliding over the submucous tissue. The tubes appear under the microscope to be a good deal altered, chiefly by solution of their epithelium, while dark grains of melanic matter are deposited between the tubes. The amount of probable knowledge we possess respecting softening of the stomach is thus expressed by Dr. Handfield Jones:—

1. There are two forms of softening: one, the commonest by far, which is simply the result of the action of the acid contents of the stomach upon its own dead tissue; the other, the consequence of a peculiar change taking place in its glandular structure, which generates a powerful acid, dissolving, corroding, or destroying the surrounding tissue; and there can be no doubt that softening of the stomach is always a *post-mortem* appearance (JONES, NIEMEYER, ELSAESSER).

2. The latter form may occur either with an empty or a full state of the blood-vessels of the stomach, the softening part of which will accordingly be either quite pale or of a dark blackish tint.

3. This same form occurs in a great variety of morbid states, which seem to have only this in common,—that they are attended with great depression of the vital powers.

4. It is more common in children than in adults, on account [probably] of the greater delicacy and less resisting power of the system.

(b.) *Glandular Degeneration of the Proper Mucous Substance* is by far the most common of the organic lesions—existing in 72 out of 100 cases examined by Dr. Jones. It is expressed in a variety of forms, the most marked of which are—(1.) Peculiar morbid changes in the tubes, probably analogous to the process which occurs in the tubes of the kidney in Bright's disease, where the molecular contents of enlarged epithelial cells increase, ultimately leading to complete destruction, so that the tubes become filled with the debris of this destruction; (2.) Melanic and fatty deposits in the epithelium; (3.) Interstitial deposit of nuclear and fibroid exudation at the expense of the gland-substance, so that hypertrophy of tissue may exist, along with—(4.) Atrophy of the tubular epithelium of the tubes themselves, and of the solitary glands—a form of cirrhosis generally associated with alcoholism, and described by Dr. Wilson Fox.

There is thus, on the one hand, in some instances, an utter destruction of the tubular glands—actual loss of substance without replacement; on the other hand, there is no actual loss of substance, but a replacement of elements by the deposition of granular matter within the tubes, without diminution or alteration of their form. That these partial degenerations, even when they extend over a considerable portion of the stomach, do not materially interfere with the ultimate digestion of food seems to be established by the cases described by Dr. Jones, deficiency of stomachal digestion being compensated for by increased vigour of intestinal digestion. Such loss of the gastric glands appears thus to have little influence over the vital acts, so that it is rare to find any evidence of the existence of such lesion during life, even when the degeneration is very extensive. I believe, however, from observations upon the intestinal mucous membrane in analogous glandular degeneration, that in cases characteristic of intestinal wasting, associated with anæmic states, as described at p. 681 of this volume, the mucous membrane throughout the whole alimentary tract is similarly affected, and that an examination of the mucous membrane of the lips, and buccal membrane of the mouth, indicates with great probability the change below, just as the condition of the tongue is the index from which we judge generally of the functional state of the alimentary canal. The general symptoms of such cases are undoubtedly anæmia and debility, without any obvious organic cause, often associated with vomiting or nausea in the morning, no desire for food, and a sensation as if it never was effectually swallowed, but stuck at the diaphragmatic entrance of the stomach, causing the peculiar feeling of weight which attends indigestion, and the abundant generation of gaseous fluids. It has been also well remarked by Dr. Chambers that such symptoms indicate a participation of the whole alimentary tract in such lesions, and that where the lesion is merely confined to the stomach, the absence of such symptoms is sufficiently accounted for by the compensation to stomachal digestion afforded by the healthy intestinal tract.

(c.) *Forms of Congestion.*—These may be described as *passive congestions;* or as *active congestions* associated with inflammatory lesions.

The *passive* form of *congestion,* like the passive form of *ascites,* is explained by the physiological phenomenon now recognized as perfectly familiar—namely, that the freedom of the transit of blood through any part is in a great measure dependent upon the healthy performance of its functions, and of the function of those parts immediately associated with it—so that, if function is arrested, the circulating current is arrested also, and the blood reverts, or is thrown back, to those intervening parts in the course of the circulation between the site of obstruction and the force propelling the blood. Thus blood ceasing to flow freely through the lungs reverts, or is thrown back, upon the right side of the heart; blood ceasing to flow freely through the liver is thrown back upon the stomach, upon the one hand, or upon the spleen by the gastro-epiploic vessels, expressed by congestions of these viscera; or, on the other hand, the portal system being obstructed, the blood reverts to the membranes of the intestines, and expresses such an occurrence by ascites, as well as by gastric venous congestion. Any mere mechanical impediment, therefore, which prevents the blood returning from the stomach towards the heart will induce congestion of the stomach.

The immediate effects of such congestion upon the functions of the stomach are sufficiently expressed by symptoms such as *hæmatemesis*, or the vomiting of blood; the secretion of the gastric juice also is diminished, the stomach can digest less food, and requires longer intervals of rest between the meals.

The persistence of such passive forms of congestion further leads to the deposition of melanic matter, to disintegration of the tubular glands of the stomach, and the formation of ulcers in its mucous membrane, just as varicose ulcers are established in the limbs from the passive congestion which attends varicose veins in the lower extremities, by impairing the nutrition of the part, as explained by Simon, Budd, and Jones.

Besides congestion from mere mechanical impediments, there are congestions of the stomach which have been described as *vicarious*—that is, as taking the place of congestions which ought to occur in other parts—such, for example, as when it occurs along with hæmorrhage in women from stoppage of the catamenia; and which has been observed to occur in yellow fever, in malignant cholera, and in typhoid fevers. Congestion also arises during the development of gastritis.

Symptoms.—Abnormal states of the stomach are expressed by—(1.) *Vomiting, associated with lesions of other organs.* (2.) *Deficient secretion of gastric juice.* (3.) *Fermentive processes (alcoholic, butyric, or lactic, tending to the development of entophytes, such as sarcinæ.* (4.) *Indigestion, associated with and depending upon*—(a.) *Morbid states of those viscera which are conjoined with the stomach in the processes of digestion, such as the liver, pancreas, and small intestines;* (b.) *Imperfect action of the kidneys, as in Bright's disease;* (c.) *Defective or diminished morophological changes during the processes of nutrition in the tissues, generally expressed by altered secretions and excretions, as in many constitutional diseases.* (d.) *Indigestion, associated with pyrosis and increased secretion of the juices of the stomach and salivary glands, and with cutaneous disorders, such as urticaria;* (e.) *Indigestion, associated with drunken habits.* The diseases of the stomach with which one or more of these organic or functional states may be associated are,—*Gastric catarrh and gastritis, chronic ulcer, hæmatemesis, perforation, dilatation, stricture, gastric fistula, hernia, cancer, colloid, tumors* (non-malignant), *sarcinæ, injuries, laceration, dyspepsia, gastrodynia, pyrosis.*

The essential juices of the stomach and of the intestines play a most prominent part in determining the nature of the disease; and some of the most important principles of treatment are based upon the physiological relations of those juices, and upon the fact that while the process of digestion of food is only commenced in the stomach, it is completed in the alimentary canal by the influence of the intestinal fluids. There is a *stomachal digestion*, in which the gastric juices, with the saliva and mucus of the stomach, play the most prominent part; and there is an *intestinal digestion*, in which the intestinal juices, composed of the *biliary*, *pancreatic*, and *intestinal secreted fluid*, play the most prominent part. It is also an important fact, especially insisted upon by Dr. Chambers, having been proved by the experiments of Bidder, Schmidt, and Handfield Jones, that the intestinal digestion may be made to do more or less of the work of stomachal digestion, so that the exercise of the function of the stomach may be spared when necessary, and the food encouraged to pass from it into the bowels, to be digested entirely by the intestinal juices.

The reader is referred for information regarding the properties and constituents of the gastric juice to the text-books on physiology; but it is necessary here to advert to the fact, recently established by the experiments of Schmidt and Bidder upon animals, and the observations of Grünewaldt upon an Esthonian peasant who suffered from a stomach fistula, that there is a constant circulation of an immense quantity of fluid through the mucous membrane of the alimentary canal, necessary to the solution and absorption of food from its interior. It is now also well ascertained that from the gastric glands the principal part of the solid animal matter of the gastric juice is derived, and which is the exciting cause of the solution of albuminoid substances in the stomach. The solid matter is regarded as a *gastric ferment*, and has been variously named as such, and also as *rennet*. But water constitutes the chief bulk of the juice; and from the experiments and observations alluded to, it is known to perform a most important function. It is continually poured forth from the surface of the mucous membrane in vast quantities, to the extent of between *a fifth* or a *quarter* of the *weight* of the *body*, and as constantly returns to mix again with the sanguineous fluid. This secretion of fluid is constantly going on from the internal alimentary mucous surface. It mixes with the dissolving food in the alimentary canal, and takes up those particles of it which it is calculated to hold in solution, loaded with which it returns again to the sanguiferous, chyliferous, and lymphatic circulations. Thus a "poor" watery fluid is constantly being sent forth, to return "*laden with wealth*," and so to minister to the nutrition of the body. This has been described as an internal mucous circulation of fluid within the body, and the arrest of this interchange, with the great retention of water, has been shown by Virchow and Parkes to be a constant condition in the febrile state (see vol. 1., p. 263).

The symptoms of *gastric catarrh* usually take the form of what is commonly called a "*disordered stomach*," expressed by headache, especially across the forehead, increased on stooping, and associated then with flashes before the eyes, and a sensation of tightness, as if the head would burst. Nausea and sickness exist, with sensations of heat and of cold, distaste for food, the sight or smell of which is apt to produce sickness, retching, and vomiting. The food already in the stomach undergoes an abnormal decomposition; lactic and butyric and acetic acids are produced, and fetid gases are set free as a result of these fermentive processes. Eructation into the mouth, of sour and rancid matters from the stomach also common. The tongue is generally coated with a white creamy fur; the odour of the breath is offensive, the mouth feels slimy, and the taste is bad.

Gastritis from poisoning causes general depression, so great in some cases as to simulate perforation. The pain generally spreads from the epigastrium over the abdomen, and is accompanied with vomiting of mucus or of bloody mucus, which may be followed by purging of similar evacuations, preceded by severe cholicky pains, and followed by collapse, small pulse, cold skin, and clammy sweat. In such cases there are grounds for suspecting that poison may be the cause of such symptoms (NIEMEYER).

Treatment.—Emetics may be required in cases where the stomach has been overloaded; and it is certain, from the gases and fluids causing prominence over the stomach, that it contains decomposing food. One scruple of *ipecacuanha*, with one grain of *tartarate of antimony*, is the safest and most efficient emetic. When injurious matters have passed into the bowels, causing flatulence and colicky pains, mild laxatives may be required, such as *rhubarb*, or *compound infusion or mixture of senna*, or *fluid magnesia*, in small doses, may be given every hour or two hours, followed by five to ten grain doses of *bicarbonate of soda.*

In cases of *chronic* or *passive* congestion of the stomach, and in *chronic gastritis*, the advantage of *nitrate of silver* has been advocated by the late Drs. James Johnson and Symonds, and more recently by many other physicians.

Dr. Johnson's formula is the following:—

℞ *Argent. Nitrat.*, gr. $\frac{1}{2}$, *Ex. Hyoscayam*, gr. ii. to gr. iv., made into a pill, which may be continued every night, the quantity of the nitrate being gradually increased to two or three grains daily. It may not be continued, however, beyond six or eight weeks.

Dr. Fleming, of Birmingham, advocates its administration in solution.

℞ *Argent. nit. Cystal*, gr. i. to gr. iv., *Aq. distilat. f* ℥ss.; taken at bed-time—the stomach being empty—and repeated every night, or every second, third, or fourth night, according to the severity of the gastric symptoms.

In cases of *acute gastritis*, mercurial purgatives by *calomel* are of great service. Three to five grains may be given to an adult; followed by a dose of *castor oil* or the *compound senna mixture* of the pharmocopœia. Where it is not desirable to act so searchingly on the small intestines, *blue pill* with *compound colocynth* or *rhubarb* pill used in equal parts (*a a* gr. ii.) combined with one grain of *ipecacuanha* powder, is a mild and gentle laxative.

Iced water to drink in small quantities, or small pieces of ice in the mouth, tends to allay thirst, and to appease pain; and the continuous use of hot water fomentations over the region of the stomach, as hot as the patient can bear them, is of great benefit (BRINTON).

CHRONIC ULCER OF THE STOMACH.

LATIN EQ., *Ulcus longum;* FRENCH EQ., *Ulcère chronique;* GERMAN EQ., *Chronisches Geshwür;* ITALIAN EQ., *Ulcera cronica.*

Definition.—*Inflammation of the stomach, ending by loss of substance in the form of an ulcer.*

Pathology.—The observations of Drs. Chambers, Gairdner, Habershon, Jones, and Brinton, have shown this lesion to be by no means uncommon. *Simple, chronic,* and *perforating* ulcers are described. The ulcer is generally solitary and deep, seldom larger than a shilling, circular or oval in shape, its edges clean, sharp, and well defined, as if punched out. It is generally situated along or near the lesser curvature, and usually nearer the pyloric than the cardiac orifice, and more frequently on the posterior than the anterior wall. It is more common in women than in men, nearly in the proportion of two to one. It is especially a disease of middle and advanced life, although Dr. Budd once met with it in a girl of fourteen. Dr. Brinton records two cases under the age of ten years. It is relatively more frequent amongst the poor than amongst the rich; and is especially found in maid-servants between the ages of eighteen and twenty-five.

It tends to prove fatal by—(1.) *Perforation;* (2.) *Hæmorrhage;* (3.) *Exhaustion.*

When the ulcer eats its way through, the aperture gradually narrows, as it reaches the peritoneum, to little more than a point, which corresponds to the centre of the ulcer. The peritoneum inflames at this spot, and sloughs, when perforation is completed by the separation of the slough. An important sanitary effort is here made by the adhesion of the peritoneum surrounding the slough to some opposed surface, such as to the liver, pancreas, colon, or abdominal wall. Fistulous openings may then be established, so that food escapes from the stomach when such passages communicate with the alimentary canal. A most comprehensive record of such cases is published in *The Edinburgh Monthly Journal* for July and August, 1857, by Dr. Charles Murchison, physician to St. Thomas's Hospital. According to Dr. Brinton's observations, about 13·4 *per cent.* of cases of ulceration terminate by perforation; and he considers that the liability to perforation decreases as life advances; and, as Dr. Crisp first showed, during the ages from fourteen to twenty it is most frequent. The patient may survive the first shock of the accident, ultimately to succumb to the combined effects of peritonitis and gastric exhaustion.

Hæmorrhage, while it is one of the most frequent and important symptoms of ulcer, is also a mode of fatal termination. It generally occurs soon after a full meal; and about from 3½ to 5 *per cent.* of the cases prove fatal in this way.

"This long and exhausting malady," says Dr. Brinton, "predisposes the constitution to a variety of other diseases, and renders unusually fatal many of those attacks of illness which, in the course of years, very few persons altogether escape."

Symptoms of Gastric Ulcer.—"The malady is announced by disturbances of gastric digestion: at first by mere uneasiness and pain; then nausea and vomiting, or regurgitation, that expel the food previously taken; or a tasteless or acid watery secretion. At this stage of the disease it is sometimes cut short by the occurrence of perforation, with its sequel of fatal peritonitis. Failing such an accident, the dyspeptic symptoms are next complicated by hæmorrhage from the stomach; sometimes a sudden and dangerous gush, oftener a slow and intermittent drain of blood. The anæmia produced by this hæmorrhage is generally associated with a cachexia which seems to be essentially independent of it; being chiefly the result of the inanition necessarily implied by frequent vomiting of the food, or by large destruction of the gastric mucous membrane, and consequent impairment of its function. In young females another symptom is often present, in the form of more or less complete amenorrhœa, which may be associated with either of these two states of anæmia or cachexia; in other words, may be connected with ulceration, with hæmorrhage, or with both. The gradual acquisition of all these symptoms conducts the disease, in a variable period, to a climax, whence we may next briefly trace it towards its termination. Retaining the liabilities to death by perforation, by hæmorrhage, by vomiting, and by exhaustion, which the above organic results of ulceration severally imply, the lesion often ends by one of these modes of dying, or by two or more of them in combination. In other cases a spontaneous subsidence of these symptoms, in something like the inverse order of their occurrence, announces a recovery; or a similar amendment is only effected by a careful medical treatment, such as quite entitles us to dignify it by the name of a cure" (*Med.-Chir Review,* p. 159, July, 1856).

The character of the pain is peculiar, at first being little more than a feeling of weight or tightness, of a dull character, and continuous. It gradually becomes intensified into a burning or gnawing sensation, which produces a kind of sickening depression. It generally comes on from two to ten minutes after the ingestion of food, and remains during one or two hours, which correspond to the period of gastric digestion, after which it gradually subsides, or, if vomiting empties the stomach, it also invariably ceases. The pain is generally expressed at the centre of the epigastrium, or at the middle line of the belly, immediately below the extremity of the ensiform cartilage, often confined to a mere spot, and rarely to a space more than two inches in diameter. A dorsal pain, first described by Cruveilhier, is also subsequently established, generally in a few weeks or months after the epigastric pain. It is expressed by a gnawing sensation, interscapular, or from the spine of the eighth or ninth dorsal to that of the first or second lumbar vertebra. Pressure in the epigastric region is sometimes unbearable, and, for obvious reasons, must be applied with the utmost care and delicacy.

Vomiting usually occurs when the pain reaches its height; and, completely emptying the stomach, generally affords relief. For further details of symptoms, the reader is referred to the admirable papers of Dr. Brinton, from whom the statements have been condensed.

Treatment.—For obvious reasons ulcers are most difficult to heal, and such cases are to be treated by rest to the stomach, and by dietetic rather than by medicinal means. The patient must eat in small quantities, and especially of milk compounded with arrow-root, macaroni, semolina, sago, tapioca, biscuit powder, Indian meal, or oatmeal gruel. *Iced milk,* combined with one-quarter to one-third of *lime-water,* is particularly recommended by Dr. Chambers; two or three tablespoonfuls of which are to be taken at short intervals, so that about two quarts of milk may be thus used during the day, rendering regular and more bulky meals unnecessary. The lime-water tends to prevent coagulation of the milk, and the milk thereby more readily passes unaltered into the intestines, to be digested by them rather than by the stomach. Dr. Balthazar W. Foster, of Birmingham, has well laid down the details of managing such cases, in "A Lecture on the Treatment of Gastric Ulcer" (*Brit. Med. Journal,* June 3, 1865). The most complete rest possible ought to be given to the affected viscus, by stopping the supply of all nutriment by the mouth, and supporting the patient for several days by nutritive enemata. Perfect quiet in the recumbent posture must be observed, the lips and tongue being moistened from time to time by a little water. The patient may thus be kept for eight or nine days, or even longer, on nutrient enemata alone, when the pain, the irritability of stomach and of the system will cease. The substances Dr. Foster has found most useful for enemata are milk, strong unsalted beef-tea, raw eggs beaten up in milk, occasionally a little brandy, and generally in two enemata daily ten or twenty minims of *tincture of opium.* The enemata should be as small as possible, from two to six ounces only at a time. The interval to the stomach thus gained of complete rest is of the greatest importance towards success in the treatment of gastric ulcer, and, combined with the restricted diet described, will usually effect a cure in three weeks.

Next to dieting, blood-letting by two or three leeches applied to the region of the stomach about twice a week affords obvious benefit, the patient often gaining weight during their use. Blistering applied to the spine is also said to relieve the dorsal pain. Small lumps of ice may be swallowed if the stomach is irritable, and astringent remedies, especially the *salts of metals,* such as a combination of *iron* and *alum,* are of great benefit; and, as a change, *gallic acid, nitric acid,* and bitter barks may be administered. Constipation is to be counteracted by enemata. When hæmorrhage occurs, Dr. Budd recommends that small bits of ice be swallowed, that rest be maintained in the horizontal posture, and that astringent medicines be administered, such as *oil of turpentine, acetate of lead and opium, alum and tannic acid. Oil of turpentine* should be given in doses varying from ten to twenty minims in *cold* water, and repeated more or less frequently according to the urgency of the symptoms. When hæmorrhage is copious, a mixture containing ten minims of *diluted sulphuric acid* and five grains of *gallic acid* in water, taken every two or three hours, is the best remedy (DR. B. FOSTER).

Vomiting may be relieved by *dilute hydrocyanic acid* (five minims), combined with *bicarbonate of soda* (twenty or thirty grains) in solution, repeated every two or three hours.

The *subnitrate of bismuth,* in doses of ten to twenty grains every six or eight hours, alone or combined with five to ten grains of *compound powder of kino,* has a remarkable effect in relieving pain, vomiting, and diarrhœa (BRINTON).

HÆMATEMESIS.

LATIN EQ., *Hæmatemesis;* FRENCH EQ., *Hématémèse;* GERMAN EQ., *Bluterbrechen;* ITALIAN EQ., *Ematemesi.*

Definition.—*A discharge of blood from the stomach.*

Pathology.—On opening the stomach of a patient that has died of hæmatemesis blood may be found in various degrees of consistency, or from a pure liquid black or brown blood to a solid coagulum. Portions of the blood thus extravasated may be also found in the œsophagus and in the intestines. The internal surface of the stomach is almost always coated with a layer of viscid mucus which separates it from the clot. This mucus is necessarily dyed of a red colour. The quantity of blood found is very various. The stomach has been found distended with blood to the utmost, forming a perfect mould of the cavity. In general, the mucous membrane of the stomach is hardly stained with the colouring matter of the blood, but it is congested, and in some few spots ecchymosed—blood being infiltrated into the sub-cellular tissue. But, even after the most abundant hæmorrhage from the gastric mucous membrane, the source of the bleeding may not be found.

The stomach, though generally healthy, is sometimes found diseased, and the hæmatemesis a consequence of ulceration of an artery or vein. Sudden death may thus result from ulceration of the stomach, which had involved several vessels. Cruveilhier gives an instance of ulceration of the coronary arteries; and Goëppert of ulceration of the coronary veins of the stomach. The most frequent cause of ulceration of the blood-vessels of the stomach, however, is cancer.

When hæmatemesis is symptomatic, the lesions found are extremely various. In one case it may result from a scirrhous tumor of the pancreas; in another from an enlarged kidney compressing the aorta; in a third from an aneurism of the cœliac artery, which, obstructing the hepatic and splenic arteries, had caused the greater portion of the blood conveyed by those arteries to pass through the gastric artery, and thus caused congestion of the mucous membrane of the stomach. Frank found in the stomach of a woman, who died of hæmatemesis at Pavia, a clot which weighed five pounds; there was no lesion of the stomach, but the liver was tuberculated and in a state of suppuration. Morgagni gives a case of hæmatemesis, in which the spleen was bigger than the liver, and weighed four pounds and a half, and Barry has given a similar case. Sometimes in symptomatic hæmatemesis the blood thrown up has come from an aneurism of the aorta bursting into the stomach; and Lieutaud gives an instance in which the blood came directly from the liver, which had adhered to the ruptured stomach.

Causes.—Hæmatemesis may arise also from causes which are peculiar to the stomach, as from the effect of vomiting, or from a blow. Another peculiar cause is ulceration of the gastric artery or vein, vessels which sometimes rupture from the effects of cancer or inflammation. Frank speaks of a girl who suffered from hæmatemesis, in consequence of a small bone sticking in the coats of the stomach. In armies on actual service the thirsty soldier sometimes suffers from this affection, in consequence of drinking incautiously water containing leeches.

The following summary of causes of hæmatemesis may be shortly stated:

(1.) Rupture of over filled blood-vessels, without change of texture.

(2.) Venous congestion of gastric mucous membrane.

(3.) Impediments to circulation in the liver.

(4.) Obstruction of the portal veins by blood-clots.

(5.) Pressure of branches of the portal veins from cirrhosis or enlargement of the gall ducts.

(6.) Pigmentary embolism from plugging of the capillary vessels of the liver with masses of pigment.

(7.) Yellow atrophy of the liver destroying the capillaries.

(8.) Rupture of diseased vessels and bursting of varices or aneurisms.

(9.) Hæmorrhagic and scorbutic conditions.

(10.) Injuries to walls of blood-vessels.

Predisposing Causes.—New-born children are sometimes subject to this disease from the day of birth till about twelve days old. Gendrin gives three cases of this kind, although there was nothing unusual in the delivery. Except at this early period hæmatemesis is rare till puberty. Frank, indeed, says he never saw this disease before puberty, nor after sixty. Both sexes are liable to it; but women suffer more frequently than men, and especially those who are either pregnant or labour under amenorrhœa.

Symptoms.—Hæmatemesis may be acute or chronic, the chronic form being usually termed melæna.

The acute form of hæmatemesis may be sudden in its attack, or may be preceded for a few hours by shivering, heat, weight and oppression at the epigastrium, by nausea, headache, and by pains between the shoulders. The buccal and pharyngeal membranes are also said to be sometimes congested, and the gums swollen. Gendrin likewise esteems a swollen state of the liver or spleen lesions sufficient to account for hæmatemesis, having observed those phenomena in five or six cases.

At length the hæmatemesis occurs, and a quantity of blood, black, clotted, and mixed with alimentary matters, is thrown up, sometimes streaming both from the nose and mouth. The symptoms which now follow are proportioned to the quantity of blood lost, and are nearly the same as in hæmoptysis. If the quantity be small, the pain in the epigastrium ceases, and the patient is relieved. If larger, the patient is in some degree relieved, but greatly exhausted; while, if the quantity thrown up, as it often is, be so abundant as to half fill a wash-hand basin or a chamber vessel, the patient becomes pale, a cold perspiration runs down his face, he has an over-whelming sense of sinking, and his pulse becomes frequent and weak. There are instances in which hæmatemesis has proved suddenly fatal.

It is rare that the attack terminates by one vomiting of blood. In the greater number of cases a few hours have scarcely elapsed when the epigastric and dorsal pains are renewed, the thirst and shivering return, and the vomiting recurs, often perhaps four or five times in the space of two or three days; a sensation, as of a burning liquid in the stomach, often precedes these subsequent attacks.

The symptoms which mark the recovery or the death of the patient are the same as those which occur in hæmoptysis.

The bowels, which are generally constipated previously to the hæmorrhage, become spontaneously open shortly after its occurrence. The stools are at first natural, but quickly become black, semi-liquid, very fœtid, and evidently contain blood mixed with bile and fæces. The abdomen is often full of gases and the seat of painful colic.

It is supposed that hæmorrhage from the stomach may take place without vomiting, the blood passing into the duodenum, and being ejected by the intestines.

The *colour* of the blood thrown up varies according to the time it has continued in the stomach. If poured out rapidly and immediately rejected it is often arterial, but accumulated slowly it is of a blackish-brown, and clotted. Sometimes a thin layer of coagulated blood forms, which, when thrown up, has been mistaken for a portion of the mucous membrane of the stomach.

The chronic form of hæmatemesis has been termed *gastro-melœna.* In this form of the disease the blood is not poured out pure, but undergoes some change, so that it resembles chocolate or coffee grounds, and is, in fact, a species of black vomit. This affection usually occurs as the last stage of many diseases, especially if the patient be of a broken and worn-out constitution. The quantity thrown up is often large, amounting to a pint or two in the course of the day, and this may last for several days. When the patient dies, which is usually the case, the stomach is found congested, but without other appreciable lesion. Dr. Baillie mentions having met with a few of these cases with no very urgent symptoms, and which ultimately recovered. In this form of hæmatemesis, also, the melænic matters often pass in the stools.

Diagnosis.—The chief difficulty in the diagnosis of hæmatemesis is to distinguish it from hæmoptysis; but the burning heat of the stomach, the black pitchy stools, the absence of cough, and of all the signs furnished by auscultation, sufficiently distinguish it from hæmoptysis. The colour of the blood from the stomach, likewise, is generally black, dark, clotted, and may be mixed with food, the coagula containing no air, and generally acid in reaction; while that from the lungs is more commonly arterial, bright-red, frothy, and mixed with mucus; not coagulated, and if so, the coagulum generally contains air, and in re-action alkaline. The quantity is also in general greater from the former than the latter viscus, although there are many exceptions to this rule. It should be remembered, also, that blood may pass from the nose into the stomach during sleep, or from the gums or tonsils after lancing. This disease is one of those, also, most easily and most commonly feigned. A microscopic examination will determine whether the matter ejected is blood. The red juice of fruits has been mistaken for blood. The matters thrown up in melæna bear no resemblance to the fluids ejected from the lungs.

Prognosis.—Hæmatemesis is devoid of danger when it is *vicarious* and arises from pregnancy, from amenorrhœa, and from suppressed hæmorrhoids. When, however, it arises from organic disease of the stomach, from disease of the liver, spleen, or heart, it is always of grave import, although perhaps not immediately fatal. When, also, it is the result of the action of a morbid poison, the danger is likewise often imminent. In melæna the case is always dangerous, but some few recover.

Treatment.—The treatment of the acute forms of hæmatemesis is similar in many respects to that of *hæmoptysis.* The great volume of the arteries of the stomach, and their origin almost immediately from the aorta by means of the cœliac artery, are reasons which have been alleged for this affection being but little influenced by general or local bleedings. The vast amount of blood, also, sometimes lost by hæmatemesis renders it necessary to support the patient by acid wines much sooner and to a much greater extent than in hæmoptysis. In melæna the only chance for the patient is a liberal support by wine, diet, and medicines, and by *opiates* to quiet the stomach.

In cases of *hæmatemesis* from *cirrhosis,* or other obstruction to the circulation through the liver, and in the vicarious hæmatemesis of women, *leeches* to the anal region or *os uteri,* are of service. The action of ice, by swallowing small pieces from time to time, is very beneficial, covering at the same time the epigastric region with ice-cold compresses, which require to be renewed frequently.

Solutions of dilute or aromatic *sulphuric acid,* in doses of ten to twenty minims, or of *alum,* are of service. *Alum* may be given in doses of eight to twelve grains, combined with Batley's solution of *opium,* or with *laudanum,* or with the *sulphuric acid.* In *chronic hæmatemesis, gallic acid* is recommended in the following formula by Dr. Brinton:—

℞. *Acid Gallici,* gr. x.; *Acid Sulph. dil.,* ♏x.; *aq. distil., f*℥i.

Solution of *pernitrate of iron,* in doses of thirty to forty minims, may be of use. *Ergot* may also be of service (see page 802, under hæmoptysis).

Tannin is of service, as one of the best astringents in the form of a pill (O. Rees)—three to six grains twice or thrice a day. The patient must maintain absolute rest in bed, and must not rise to stool.

Ipecacuanha in doses to produce nausea has been shown to be of service in arresting hæmorrhage, and restoring heat to patients in the collapse of hæmatemesis (Osborne, Trenor, Graves). The dose may be one to two grains every fifteen or thirteen minutes till nausea is felt, when the remedy is to be discontinued, as vomiting is to be avoided. If stimulants are required, iced champagne may be given; and when the vital powers are much depressed, *hydrochlorate of ammonia,* combined with *hydrochloric acid,* may be given in the following formula:—

℞. *Ammon. Hydrochlor.,* ʒiss.; *Acid Hydrochlor., f*ʒss.; *Decoct. Hordei Co.,* oz. i., of which two or three table-spoonfuls may be taken every second or third hour (Copland).

DYSPEPSIA.

Latin Eq., *Dyspepsia;* French Eq., *Dyspepsie;* German Eq., *Dyspepsie;* Italian Eq., *Dyspepsia.*

Definition.—*Impairment of digestion, arising without perceptible change of structure or lesion of the stomach.*

Pathology.—It is thus regarded as an abnormal functional difficulty; and the impairment may be due—*First,* to what have been called sympathetic relations with other organs, themselves in a morbid state, and which are now explained by the phenomena of "*reflex action.*" Examples of this may be referred to in the nausea and sometimes *vomiting* which attends irritation of the *lung, brain, liver,* or *uterus.*

Second, the digestion may be impaired by a scanty secretion of the gastric juice; and the difficulty of digestion is characterized by slowness and long retention of food by the stomach; prolonged distress after eating, especially of weight and uneasiness at the pit of the stomach; peculiar tendency to decomposition of food in the alimentary canal; the evolution of fetid gases (*pnuematosis*); and the appearance of unaltered ingesta in the stools.

Gastric digestion should occupy, on an average, from two to three hours.

Often an inheritance by birth, the conditions which tend to produce this morbid state are especially, mental over-exertion, prolonged anxiety, especially after meals, gluttony, drunkenness, and sedentary habits, and the consumption of more food than the system requires (LEARED).

Thirdly, the impairment may be due to the abnormal quality of the gastric juice; or from diminished movements of the stomach, so that the ingesta are not sufficiently mixed with the gastric juice (NIEMEYER).

The "*causes, symptoms, and treatment of imperfect digestion,*" from these points of view, are admirably treated of by Dr. Leared.

The symptoms brought about by diminished secretion of the gastric juice are very similar to those of chronic catarrh and chronic gastric ulcer. The appetite is lessened, and is easily satisfied; but however little may be eaten, gas is rapidly generated, and sour, fetid, or rancid eructation of liquid occurs. The patients suffering from flatulence and consequent distension, are disturbed and depressed. Palpitation of the heart is common, as well as irregularity of the pulse, headache, occasional dimness of vision, and attacks of a peculiar form of giddiness described by Trousseau (*vertigo stomicale*), but which Niemeyer believes to be due to psychical causes. The mental depression from dyspepsia is often extremely distressing, varying in degree from slight dejection and ill-humour, or irritable temper, to the extreme of melancholia, or suicidal monomania.

"Half the unhappiness in the world," writes Sidney Smith, "proceeds from little stoppages, from a duct choked up, from food pressing in the wrong place, from a vext duodenum, or an agitated pylorus.

"The deception practised upon human creatures is curious and entertaining. My friend sups late; he eats some strong soup, then a lobster, then a tart, and he dilutes these esculent varieties with wine. The next day I call upon him. He is going to sell his house in London and to retire into the country. He is alarmed for his eldest daughter's health. His expenses are hourly increasing; and nothing but a timely retreat can save him from ruin. All this," says Sidney Smith, "is the lobster; and, when over-excited nature has had time to manage this testaceous incumberance, the daughter recovers; the finances are in good order;

and every rural idea effectually excluded from the mind. In the same manner old friendships are destroyed by toasted cheese; and hard salted meat has led to suicide. Unpleasant feelings of the body produce correspondent sensations in the mind, and a great scene of wretchedness is sketched out by a morsel of indigestible and misguided food. Of such infinite consequence to happiness is it to study the body" (*Memoir of the Rev. Sidney Smith*, by Lady Holland, vol. i., p. 125; London, 1855).

In the impaired digestion from over-secretion of gastric juice or its abnormal qualities, *cardialgia* is the name of the characteristic symptom —namely, pain at the orifice of the stomach. The secretion of a fluid abnormally acid by the stomach, causes a most unpleasant sensation about the cardiac orifice, and hence it is also sometimes termed *heartburn.* This fluid is often regurgitated into the mouth, has a most disagreeable oily acid or rancid taste, and not only sets the teeth on edge, but, expectorated on any carbonated alkali, causes effervescence. By Dr. Prout it is stated to be principally *lactic acid.* The effects of this condition are more or less pain in the stomach, accompanied by distressing flatulence, derangement of the bowels, headache, disturbed sleep with terrifying dreams. The remote effects are—palpitation, gravel or stone, or a gouty or rheumatic state of the constitution, or *uric acid diathesis,* for the urine is loaded with the lithates, and the urine small in quantity. This state of things may be caused by an absorption of the acid, the assimilation in the lacteal system being most imperfect (DR. PROUT).

This condition of gastric derangement most commonly occurs in those that live high, eat largely of rich black meats, and drink largely of malt liquors or champagne, which act as ferments, turn acid, and dispose everything else to undergo the same changes. Some persons, especially those descended from gouty or rheumatic parents, have an idiopathic tendency to this form of impaired digestion, and in these the most opposite substances will produce it, as sub-acid fruits, salt meats, pastry —indeed anything that deranges their enfeebled powers of digestion. Tobacco has a poisonous principle which greatly favours the occurrence of this disease, and many persons suffer severely after smoking a very few cigars, or even one.

Urine in Dyspepsia.—Alterations in the free acidity are the most important signs. But in some dyspeptic cases, especially those attended with torpid digestion or with acid vomiting, it has seemed to Dr. Parkes that the urine is more alkaline than usual; so that there may be abnormal acidity as well as abnormal alkalinity in cases of dyspepsia. When the urine is more acid than usual after food, it is also often scanty, and deposits urates and oxalates of lime—conditions associated chiefly with cardialgia, nausea, and frontal headaches. Chloride of sodium is small in amount when digestion is imperfect; and the more that is in the urine, the more perfectly has digestion been carried on. Oxalate of lime-crystals are common in dyspepsia (PARKES, l. c., p. 333).

Treatment.—Congestion, catarrh, and functional states associated with what are called dyspeptic symptoms, or "imperfect digestion," are the causes of stomach diseases for which the physician is called most frequently to prescribe.

When there is reason to believe *congestion* exists, a sparing and easily digested diet is to be prescribed, and total abstinence from fermented drinks is imperatively demanded; and in cases where there is reason to believe that catarrhal inflammation prevails, the blandest food must be given in very small quantities. In severe cases leeches are to be applied over the region of the stomach, and the patient may sip iced water, or suck small pieces of ice, to relieve the thirst.

In impaired digestion from any cause, a mode of life tending to improve nutrition is to be aimed at.

Dr. Leared justly puts great stress on the necessity of strict attention to diet in all cases of impaired digestion. If fulness and uneasy sensations are experienced after dinner, less food should be taken at that meal, and more at breakfast; the principle being to apportion the amount of food necessary to sustain the body more evenly over the waking hours than is commonly done. The great fault in the dietetic system of this country consists in the fact that most people are supported mainly by dinner. This meal is consequently too large. The quantity taken at dinner should therefore be resolutely diminished till breakfast is appreciated (LEARED, l. c., p. 150).

Special symptoms, common to various morbid states, require special modes of treatment. Excess of acid is best neutralized by *lime-water*, *magnesia*, or *alkaline* remedies, selected according to the state of the patient's bowels; and the gastric fermentation which is apt to be established may be checked by *brandy* and *various aromatic spirits*. *Bicarbonate of potash* and *nitrate of potash*, in the proportion of *eight* parts of the former to *one* part of the latter, is useful in cases of habitual acidity; and all these antacid remedies should be taken about three or four hours after a meal.

Pills containing from a quarter to half a minim of *creosote*, given with each meal, will in general counteract fetid eructations. It checks that fermentation in which acetic and carbonic acids are formed; while *conium* and *belladonna* are the medicines which better than *opium* allay general nervous irritability.

In cases of slow digestion, with deficient secretion of the gastric juice, the rules of treatment are—(1.) To let albuminoid food be as liquid as possible; eggs, therefore, must be eaten when cooked short of coagulation oflthe albumen; (2.) To let the day's allowance of food be taken in smal quantities at regular intervals; (3.) That by the administration of alkalies the food may pass to the intestines, and be digested there, rather than by the stomach. This latter mode of treatment by alkalies, recommended by Chambers, is contrary to that recommended by Dr. Budd. Both are consistent with physiological facts, and the course to be followed must be determined by the nature of the case. Both gave large doses of *bicarbonate of soda*—ʒii. dissolved in a pint of warm water—to counteract the excessive acidity, or to promote the passage of food to the intestines.

The amount and kind of food taken is of great importance to be attended to in cases of slow digestion. If a fair amount of exercise be taken, the following dietary, slightly modified from that proposed by Dr. Leared, will be found appropriate in such cases:—

BREAKFAST, (8 A.M.)

Bread (stale),.......4 oz.	Mutton Chop, or other meat (cooked) free from fat and skin,..3 oz.	Tea, or warm milk and water and sugar, or other beverage,....................¾ pint.

LUNCHEON, (1 P.M.)

Bread (stale), ...2 oz.	No solids, such as Meat or Cheese.	Liquid,.....................¼ pint.

DINNER, (5 or 6 P.M.)

Bread (stale),.......3 oz. Potatoes and other Vegetables,.......4 oz.	Meat (cooked) free from fat and skin, ...4 oz.	Liquid, not more than ½ pint.

TEA or SUPPER (not sooner than three hours after Dinner).

Bread (stale), ...2 oz.	No solids, such as Meat or Cheese.	Tea, or weak brandy and water, or sherry and water, or toast and water, to the extent of ½ pint.

The dietetic treatment is of the utmost importance; and the quantity of wine or other fermented liquor, and also of animal diet, should be thus reduced till the disease subsides and the urine is healthy. Soups, tea and coffee, drank, as they usually are, boiling hot, debilitate the coats of the stomach, and tend consequently to produce this affection, and are abandoned by many persons from their so often exciting cardialgia.

The best bread for the dyspeptic is the unfermented bread, as baked by the process of the late Dr. Dauglish, entirely from wheaten flour of the best quality. It is sometimes known as "*aërated bread*," because carbonic acid gas is substituted for yeast. It is more easily digested than common househeld bread. Pastry and "*sweets*" must be, as a rule, strictly forbidden. They are "Sweet in the mouth, but bitter in the belly."

For excellent advice on this subject, the reader is referred to Dr. Leared's book, p. 160, *et seq.;* and to Dr. T. K. Chambers's *Lectures chiefly Clinical.*

Forms of indigestion marked by excessive acidity and heartburn may be relieved by *bicarbonate of soda,* in doses of fifteen grains, combined with a few grains of nitre, and taken two or three times a day. At the same time, free excretions from the liver and bowels must be sustained by occasional small doses of *blue pill* or *podophyllin,* combined with extract of *colocynth* and of *henbane;* while exercise and diet are duly attended to.

Weakened digestion from over-fatigue may be often restored under the use of *carbonate of ammonia,* conjoined with *compound tincture of gentian,* or with *extract of gentian* in the form of a pill. Extracts of *nux vomica* or *strychnia* are also valuable remedies. *Half a grain of extract of nux vomica, half a grain of sulphate of iron, and four grains of compound colocynth pill,* form a combination which, taken early in the morning, or one hour before dinner, generally induces gentle action of the bowels (LEARED). *Compound rhubarb pill* may be substituted in place of the *compound colocynth pill.*

Another most useful ingredient in a dinner pill I find is *ipecacuanha,* to the extent of one grain, or half a grain in each, in cases of slow or torpid digestion.

Indigestion from habitual drunkenness, or where there is great irritability or sensitiveness of the stomach, is best relieved by the pure bitter infusions, such as *gentian*, *quassia*, *hops*, and *calumba*, singly, or combined in a mixture, so that a dose may be taken two or three times a day, an hour before each meal.

Quassia may generally be taken as a *cold infusion*, and is thus prescribed by Niemeyer:—"In the evening, pour a cupful of cold water over a tea-spoonful of *quassia chips*; by the next morning a bitter infusion will have formed—to be taken fasting;" or water may be poured into a bowl made of *quassia-wood*, and sold for this purpose, and after standing over night in the bowl, is to be taken in the morning. *Hop-bitter* is most agreeably taken in the form of the many bitter ales, such as are brewed by Bass, Allsopps, Ind.-Coope, and the genuine Bavarian beer, brewed all over Germany. Such ales must be got direct from trustworthy brewers as brew from hops, and where no injurious substitute is used instead of hops. For other reasons it is also necessary to obtain the ale direct from the brewery; as, once it passes into other hands, there is no guarantee that some of the numerous processes for "*stretching*," and otherwise increasing the *quantity*, to the disadvantage of the consumer, will not be practised; and which are so well known, that for the purposes of the physician bitter ales, as agents in curing disease, must be prescribed direct from the brewer; just as milk must be prescribed direct from the cow.

The extract of malt, originally prepared by Trommer, as a genuine extract, containing the soluble constituents of the malt and of the bitter of the hop, may be prepared by any pharmaceutical chemist. One hundred parts of it contain the following ingredients (NIEMEYER):—

Grape Sugar, Malt Sugar, Bitter Essence of Hops, and Tannin,	76·	parts.
Albuminous or Protean substances,	7·	,,
Phosphates of Lime and Magnesia,	·82	,,
Alkaline Salts,	·18	,,
Water,	16·	,,

Niemeyer speaks highly of it in the treatment of cases of "*irritable indigestion*," as occasionally having been almost the only nourishment the patients bore.

Small doses of *opium* or of *morphia* in an *ammonia mixture* may also be given at bedtime, so as to secure sleep at night.

In prescribing the mineral acids, the following general rule, stated by Dr. Bence Jones, ought to be kept in mind—namely, that the influence of *sulphuric acid* is astringent, while that of *hydrochloric acid* promotes digestion, and of *nitric acid* secretion.

PYROSIS.

LATIN EQ., *Pyrosis;* FRENCH EQ., *Pyrosis;* GERMAN EQ., *Sodbrennen;* ITALIAN EQ., *Pirosi.*

Definition.—*Paroxysms of pain at the cardiac or œsophageal region of the* *stomach, with occasional erruption of thin watery acid and colourless fluid, but which may be cold and insipid to the taste of the patient.*

Pathology.—*Pyrosis* (πυροω, to burn), is also known by the name of water-brash, *fer chaud.* It is a painful disorder of the stomach, occurring in paroxysms, and which does not cease till the patient vomits up a limpid colourless fluid like water, to the patient's taste cold and insipid, but which sometimes gives an acid and sometimes an alkaline re-action.

This disease is frequently met with in Scotland and in Ireland; and Linnæus says one-half of the inhabitants of Sweden are liable to it. From the large quantities of spirits drank in those countries it has been supposed to be caused by their immoderate use. Dr. Pemberton, however, was convinced, after the minutest investigation, that this opinion was erroneous. "For had the disease arisen from the intemperate use of spirits, we should expect to find it most frequent among men, who are more addicted to immoderate drinking than women. On the contrary, I find," he adds, "that the disorder is more frequent among women than men, in the proportion of five to one. I must remark, moreover, to show how unfounded is the opinion respecting the use of spirituous liquors being the cause of the disease, that the women in the north of Ireland are remarkably temperate in their own country; and again, that the same order of women, when they are brought to this, and contract the pernicious habit of drinking spirits, are free from this complaint." This affection seldom occurs except in those who live upon a low and insufficient diet. It often exists in connection with some derangement of the uterine or nervous system, or with organic disease of the stomach, or pancreas, or liver. It is not uncommon in advanced life.

Symptoms.—The fit of pyrosis usually comes on in the morning and forenoon, when the stomach is empty. The first symptom is a sense of constriction, as if the stomach was drawn towards the back, while others describe it as a severe and often a burning pain. This *gastrodynia* (as in fact it is), the patient finds increased by standing or sitting upright, and therefore he seeks relief by bending his body forward and making pressure on the affected part. The attack lasts from a few minutes to the greater part of an hour, when a clear, limpid, tasteless fluid is vomited up, varying in quantity from an ounce to a pint. As soon as this fluid is rejected the pain ceases, and the paroxysm is at an end. The paroxysm may occur three or four times a day; but when there is only one, it usually comes on before ten o'clock in the morning. In addition to the paroxysm, the patient's appetite is generally impaired; he complains of thirst, his bowels are generally constipated, his surface pale and his body emaciated.

Treatment.—The medical treatment of this affection consists in saline remedies mainly, such as a drachm of the *sulphate of magnesia,* with fifteen minims of the *tinct. hyoscyami,* three times a day. Many other medicines have been recommended, as the *tinct. kino,* by Dr. Pemberton. The *compound tincture of benzoin,* to the extent of one fluid drachm, with mucilage, is most efficacious (BAILLIE, SYMONDS). The *subnitrate of bismuth* is also to be commended. *Sulphurous acid,* in doses ♏ xxx. to one fluid drachm, thrice daily, and shortly before meals, is advocated by Dr. Lawson (*Practitioner,* Sept., 1868). It is especially useful where the *sarcinæ ventriculi* are present in the fluid evacuated. The diet should, if possible, consist of some animal food, and be otherwise nourishing. Oatmeal and brown bread must be avoided.

SECTION VII.—DISEASES OF THE INTESTINES.

ENTERITIS.

LATIN EQ., *Enteritis;* FRENCH EQ., *Entérite;* GERMAN EQ., *Enteritis;* ITALIAN EQ., *Enteritide.*

Definition.—*Inflammation of the small intestines.*

Pathology.—Although *enteritis* is generally described in text-books, as a rule it is a rare disease, and seldom affects the intestine throughout its whole extent. The several parts under which it is anatomically described are variously influenced by local inflammatory processes. The peculiarities of anatomical organization, such as the various forms of minute glandular parts (see vol. i., p. 546), determine in some measure the forms by which these organic lesions are expressed. With some modifications, explained by such peculiarities of structure, the *organic* lesions of the lesser intestinal tract are in many respects precisely similar to those described in the stomach; while the functional disorders are indicated by the various forms in which the intestines express irritation or perverted action, such as by *spasms, colic, flatulence, indigestion, constipation,* and various forms of *fluxes* or *diarrhœa.*

The phenomena of inflammation, when they do occur, generally express themselves in the ileum.

Acute diffuse inflammation is marked by redness, thickening, and impaired cohesion. The redness is of a *deep venous red*, approaching to blackness, either partial or general in extent, and in dotted, arborescent, or striated patches. It is distinguished from mere passive congestion by the increased arterial vascularity of the submucous tissue. The thickening is generally sensible, and often considerable. The impaired cohesion is not so obvious as in the stomach, but the mucous membrane may be removed much more easily than in health, from its attachment to the parts subjacent.

In the chronic forms of diffuse inflammation the colour, thickening, and cohesion of the gut are not greatly changed; but in general the thickness is more considerable, the cohesion of parts, instead of being impaired, is often rendered more tenacious, while the dark venous hue, on subsiding, leaves a greyish or slate-coloured tint, from a deposit of melanic matter in the substance of the membrane.

Inflammation of the mucous membrane of the small intestines may be inferred to exist from the large quantities of fluid often discharged by stool during life, at the same time that the abdomen is the seat of pain and tenderness. After death the fact may be proved by the loose diffluent fæcal matter often found in the small intestines; at the same time the mucous membrane is partially or generally inflamed.

Chronic catarrh of the intestines is the most common of diseases.

It is the result of the following conditions, which are illustrated at great length by Niemeyer, namely:—

(1.) Obstruction to the hepatic circulation, and especially to the escape of the blood from the portal vein.

(2.) Lesions of the respiratory and circulatory organs, which cause obstruction to the emptying of the *vena cava.*

(3.) Disturbance to the external circulation, such as accompanies severe inflammations of the skin, as from burns, or from sudden exposure of the skin to low temperatures; as by travelling in cold weather.

(4.) As a result of peritonitis.

(5.) As a result of mental excitement.

(6.) As the result of local irritations from the use of purgatives; of some kinds of vegetable food; the passage of undigested decomposing substances from the stomach into the intestines; or the retention of fæcal masses.

(7.) A symptom of such lesion as *lardaceous* disease of the intestine; of septicæmia, and of cholera (simple and malignant).

The effusion of coagulable or fibrinous lymph in the small intestines is an extremely rare occurrence. "I have," says Dr. Baillie, "seen in violent inflammation scattered portions of coagulable lympth thrown out upon the surface of the villous membrane. This, however, is very uncommon" (p. 158). Billard has seen it but twice in the intestines of children. Dr. Handfield Jones also notices that the surface is sometimes the seat of an exudation much resembling that of croup; the attacks recurring several times, each presenting a stage of irritation which ends in the formation and throwing off of a false membrane. This membrane sometimes forms a layer of some thickness, extending pretty uniformly over the surface, or appearing in the stools as tubular casts of the intestines, and sometimes it is as thin as a wafer, or consists merely of tattered shreds. In one case mentioned by Dr. Copland there were shreds of dysmenorrhœal false membrane discharged from the uterus, but not at the same time (*Pathological Anatomy*, by Jones and Sieveking, p. 526). The diphtheritic exudations described by Rokitansky are of a similar nature. Pseudo-membranous inflammations of the bowels have also been described by Dr. W. Cumming, of Edinburgh, and Dr. Simpson. It is the rule to find such exudation in cases of *scorbutic dysentery*, when the inflammatory process seems to extend from the great intestine into the small.

The following are the principal results of enteritis:—

(a.) *Softening of tissue* has been described in the small intestines similar to that affecting the stomach; but it occurs much less frequently, and is not expressed by any recognizable clinical phenomena.

(b.) *Glandular lesions and degenerations*, as results of inflammation, are of much more frequent occurrence, if not constant, over limited portions of the intestine. The lesions are peculiar, from the structure of the parts; and the *degenerations*, as they are termed, are analogous to those described by Dr. Jones as occurring in the stomach (see p. 897 and 898).

As in the stomach, so in the intestines there is to be observed in some cases, on the one hand, an entire destruction of the gland-tissue, with actual loss of substance, so that when the mucous membrane is delicately dissected from the muscular parts, it may be viewed as a transparent object with a lens. On the other hand, there may be no actual loss of bulk or of substance, while the structure is nevertheless greatly altered—(1.) By alterations of the contents of the mucous tubes, without change of form; (2.) By an interstitial deposit of fibrinous matter encroaching upon and ultimately obliterating the proper glandular tissue. Thus, externally and in bulk, the parts may seem to be unchanged; but they are found to be materially altered in specific weight, and the range of alteration is considerable.

Thus, through lesions or degenerations of the gland-tissue, *atrophy* of the mucous membrane of the intestines becomes expressed in two ways (as originally expressed by Dr. Bucknill with reference to the nerve-substance of the brain), namely,—(1.) *Positive atrophy,* in which the tissue of the gut wastes, while the glandular texture is altered; (2.) The tissue of the gut may not have wasted; on the contrary, it may have gained in bulk, but the glandular tissue has been changed, or been altogether replaced; while (3.) The two conditions may be co-existent.

The observations which appear to me to prove these statements are, alterations in the specific gravity of the mucous membrane of the intestine, associated with characteristic morbid appearances, as shown by microscopic examination of sections. These combined modes of examination, not only in mucous membrane, but in all parts in which I have applied the test, have yielded one characteristic result—namely, that atrophic states of prolonged duration, which are generally described as *granular degenerations* of minute tissue, are for the most part of comparatively low specific gravity, and with a chemical re-action under the microscope indicative of the presence of fat; while, on the contrary, the acute inflammatory conditions of tissue (where loss of peculiar minute structures, like the glands of the intestines, is due to destruction from exudation and replacement by it) have uniformly high specific gravities. These results are similar to those which Dr. Bucknill has expressed with reference to the brain (*Med.-Chir. Review,* January, 1855, p. 212). A specific gravity of 1·032 to 1·033 of the mucous membrane, when it is free from congestion, may be regarded as associated with the healthy state of the gland-tissue. In conditions of *positive wasting* throughout, it descends to 1·030; while in conditions of *relative wasting,* where bulk is unchanged, but where glandular parts are displaced or destroyed by exudative deposits, the range of specific weight of parts examined has been as high as 1·044, and in the large intestines as high as 1·050. The specific gravity of Peyer's patches I have found to vary from 1·032 to 1·044, and even in patches from the same intestine there is often a considerable latitude in the range of the specific weights of the glands. The atrophic states of these glands are indicated—(1.) By a reticulated condition, void of all glandular elements, and which seems to occur naturally with the advance of years beyond forty; or to occur after extensive *infarction* during the progress of typhoid fever; the elimination of the product thus taking place without ulceration. (2.) By the remains of the cicatrices of ulceration in the form of dark granular deposits; or of a thin, clear, skin-like membrane, of a pale colour, and with a wrinkled contracted border. These morbid changes are most commonly associated with the prolonged continuance of complex morbid processes, as in constitutional diseases, such as those of anæmia, or in organic lesions leading to impairment of the constitution generally. They are also associated and expressed more or less locally amongst the peculiarly local glands or accumulations of glands of the gut, such as the solitary lenticular glands and the patches of Peyer, during the progress of some zymotic diseases, such as in cases of *cholera, dysentery,* and *typhoid fever.*

The definite morbid conditions in which the glandular apparatus may be found are as follow:—(1.) *Intumescence, stuffing,* or *cramming* of the gland, by different kinds of deposit, by debris of normal elements, or of both, and associated with redness and vascular congestion of the submucous tissue—conditions which correspond to the so-called "*infarction*" of the older pathologists; (2.) *Softening, degeneration,* and *elimination* of the abnormal constituents in various ways; (3.) *Ulceration,* sometimes leading to sloughing, of whole patches of glands; (4.) *Collapse of solitary glands,* or otherwise cicatrized remains of ulcerations of solitary and aggregate glands; (5.) *Accumulation of melanic matter,* characteristic of pre-existent and long-continued vascular action, with changes in the tissue from hæmatine (*Pigmentary degeneration;* see vol. i., p. 96); (6.) *Lardaceous disease* (see vol. i., p. 99).

The cramming of the glands, associated with submucous vascularity, is generally due to a milky-like exudation, with the variously metamorphosed epithelial elements. Such exudation may subsequently undergo various kinds of metamorphoses yet to be determined; but generally it may be stated that *corpuscular* and *fibrinous* elements co-exist in the various forms in which the glands are found intumescent.

There can be no doubt, from the observations of Dr. Handfield Jones, as well as from what is consistent with daily observation, that many of these obvious changes go on independently of any expressions of the phenomena of inflammation as commonly understood. But it is now well known also that the undoubted results of the inflammatory process sometimes occur without the manifestation of symptoms, such as *pleuritic effusions,* and which yield to appropriate remedies. In such cases the inflammation has been considered and described as latent. Does it not therefore appear consistent to associate the phenomena of such latent inflammations with the phenomena described by Goodsir and Redfern as the first stage of lesion in the cartilage-cell; with those of the early degeneration in the epithelium of the uriniferous tubes in Bright's disease; with the degeneration of involuntary muscle described by Quain; with the changes described by Virchow in the cornea, by Bucknill and Skae in the brain; with the early changes in the epithelium of the mucous surface of the bronchi, where they lose their cohesion, separate with abnormal rapidity, and ultimately assume forms and characters not to be distinguished from pus; and, lastly, with the phenomena of alteration and degeneration of the minute contents of these glandular parts now described; and consider such changes as expressions of one and the same complex morbid process, and which Virchow has described by the name of *parenchymatous inflammation?* (See vol. i., p. 60.) The ultimate results and further expression of the inflammatory process varies according to the texture of the part and other circumstances; but here we have the initiative of the process similar in all tissues, while its ultimate results are various; and while the complex state known as "fever" has found a constant expression in "the elevation of temperature" may we not be entitled to say that "the no less complex process of inflammation" has a no less constant expression in the diminished cohesion of the minute elements of tissue?

The **Symptoms** of *enteritis* partake more or less of those belonging to *intestinal catarrh*, combined with pain, aggravated by pressure, as a characteristic. Movements from the bowels become more frequent, and diarrhœa is a most constant symptom. Pain may or may not be present. The chief seat of pain is generally about the umbilicus, or the right iliac fossa. The discharges from the bowels relieve for the moment the griping pains. The pulse is excited, and generally full and strong.

The absence of intense pain and tenderness, of vomiting, of constipation, of excessive vomiting and tympanitis, of the small and frequent pulse, all of which are characteristic of peritonitis, is sufficient to distinguish enteritis from that disease.

Acute intestinal catarrh is generally accompanied by fever, as in catarrhal fever from cold.

Treatment.—The treatment of *enteritis* and *intestinal catarrh*, when not arising from a morbid poison, is by leeches to the abdomen, gentle purgative medicines combined with an opiate, fomentations, and purgative or opiated enemata. After the inflammation has subsided, mild tonics, as the *compound tincture of gentian* with *nitro-muriatic acid*, may be substituted, with the prospect of recovering the lost tone of the parts.

When catarrhal diarrhœa exists, if there is reason to believe that much congestion prevails connected with the hepatic region, a cathartic dose of *calomel* followed by *castor oil* may be necessary, If the stools indicate an acid re-action, *magnesia* may be given with advantage. When membranous films or shreds of coagulable lymph are passed, electro-galvanic applications over the abdomen and dorsal spinal region, combined with *creosote* or *tar* in the form of a pill, is quoted by Dr. Wood, on the authority of Drs. Cumming and Simpson, of Edinburgh, as worthy of a trial. Next to those remedies are such as determine towards the skin; and for this purpose combinations of *Dover's* with *James's powder*, or a *solution of tartar emetic* with *laudanum*, are the most useful medicines; and their beneficial action is very much aided by the use of the warm bath, and a flannel roller applied with firmness round the abdomen (CRAIGIE).

Where hyperæmia is excessive, and accompanied by moderate transudation from the intestine, Niemeyer considers that cold external applications are suitable, as well as in the severer forms of catarrhal enteritis, as after extensive burns, accompanied by great pain. The best mode of using such cold appliances is by cloths wrung out of cold water and laid over the abdomen.

In the chronic form of intestinal catarrh, particularly those combined with obstruction, the same authority considers irritating and warm compresses most suitable. Preissnitz's compress is now extensively used for this purpose. A wet towel in folds, covered with a dry one, is laid over the abdomen all night; or it is renewed several times daily.

The greatest care ought to be given to the management of the diet. It ought to be strictly antiphlogistic, consisting entirely of slops and light puddings.

Animal food must be entirely withheld, and only permitted occasionally in the form of soup. The safest diets are those of arrow-root, sago, tapioca, gruel of oatmeal, *sowens*,* barley water, toast water, burnt oatcake water. This last substance, used as a drink, is said to allay irritation and morbid sensibility of the bowels (CRAIGIE). When the abdominal pains and stools subside, and the appetite increases, the diet must be, if possible, still more scrupulously attended to. Oatmeal porridge boiled to the utmost possible degree of pulpiness, and not too consistent, is one of the best of diets, which may be alternated with ground rice, alone or combined with barley flour, all of which may be eaten with diluted milk from the cow, or with milk undiluted of town-fed cows.

TYPHLITIS.

LATIN EQ., *Inflammatio cœci intestinii;* FRENCH EQ., *Typhlite;* GERMAN EQ., *Blinddarmentzündung;* ITALIAN EQ., *Tiflitide.*

Definition.—*Inflammation of the cœcum, which often leads to ulceration of its mucous membrane, and not unfrequently of the entire wall of the bowel.*

Pathology and Symptoms.—Collections of fæces in the cœcum and ascending colon, or of fæcal matter composed of such crude accumulations as the skins or stones of fruit, portions of unripe fruit or uncooked vegetable substances, intestinal concretions, balls of lumbrici may be the first beginnings of cœcal inflammation, preceded by repeated attacks of colic and catarrh, with stomachache and alternate constipation and diarrhœa. When the inflammation is so severe as to involve the entire coats of the bowel with ulceration of the wall of the intestine, the muscular coat becomes paralyzed, and is unable to propel onwards the fæcal contents. Mucus or bloody mucus passes by the rectum, the result of catarrh; but no proper evacuation is obtained. Thus scybalлæ tend to accumulate throughout the saccular pouches of the large intestine; and dilation, with paralysis of the bowel, extends along the gut. In most cases there are severe pains in the right iliac region, and characteristic fæcal tumors. The pain is increased by pressure and motion, and is marked by severe exacerbations, with intervals of comparative ease. The fæcal tumor generally takes the position and assumes the shape of the cœcum and ascending colon, and may extend from the right iliac fossa towards the lower margin of the ribs.

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* "The husk and some adhering starch separated from oats in the manufacture of oatmeal are sold in Scotland, under the inconsistent name of '*seeds*.' These, if infused in hot water, and allowed to become sourish in this state, yield on expression, a mucilaginous liquid, which, on being sufficiently concentrated, forms a firm jelly known by the name of '*sowens*.' Not less than a quart of the *seeds* are to be rubbed for a considerable time with two quarts of hot water, after which the mixture is to be allowed to rest for several days till it become sour. It is then strained through a hair sieve, and the strained fluid left to rest till a white sediment subsides. The supernatant fluid is to be poured off, and the sediment washed with cold water; after which it may be either boiled with fresh water, stirring the whole time it is boiling, or it may be dried and prepared when convenient in the same manner as arrow-root. It may be eaten with wine, milk, or lemon juice and sugar" (Pereira *On Food*, p. 326).

In some cases the inflammation extends to the peritoneum of the adjoining intestine and abdominal wall, as well as to the connective tissue which unites the ascending colon behind to the iliac fascia (*perityphlitis*). There may then be pains in the right thigh, the psoas and iliacus muscles also being infiltrated, they are unable to contract or the patient to raise his thigh.

All these symptoms, although severe, may pass away after several passages of masses of badly smelling fæces; secondary inflammations cease, and the exudations are gradually absorbed, when pain subsides and the tumor disappears.

On the other hand, the disease may be fatal by peritonitis, or by the formation of a fæcal abscess in the right iliac region (NIEMEYER).

Treatment.—A full dose of *castor oil* (℥ss. to ℥i.) is indicated if vomiting does not exist. Drastic purgation is not to be thought of. The use of enemas through long rectum tubes are the most efficient means of relief, which may throw up four or five pints of liquid in a continuous stream, so as to soften, crumble down, and set in motion the fæcal collections. *Salt, castor oil,* or *turpentine,* or *milk* ought to be added to the fluid injected (NIEMEYER).

Leeches may be required if pain on pressure exists in the iliac region.

DYSENTERY.

LATIN EQ., *Dysenteria;* FRENCH EQ., *Dysenterie;* GERMAN EQ., *Ruhr*—Syn., *Dysenterie;* ITALIAN EQ., *Dissenteria.*

Definition.—*An infectious febrile disease, accompanied by tormina, followed by straining, and scanty mucous or bloody stools, which contain little or no fæcal matter. The minute lenticular and tubular glands of the mucous membrane of the large intestines, with the intertubular connective tissue, are the chief seats of the local lesion, which sometimes extends into the small intestine beyond the ileo-colic valve; as in cases in which* scorbutus *is a predisposing cause.*

Historical Notice, Pathology, and Morbid Anatomy.—Dysentery is a disease which varies considerably in different countries and localities; and sometimes in apparent accordance with the exciting cause. Sporadic cases, which now and then occur in our large towns, are not generally so violent, and are less fatal than the epidemic cases, and those which occur in tropical climates. The effects on the constitution are no less varied and severe.

Dysentery has at all times proved one of the most severe scourges of our fleets on foreign stations, of our armies in the field, and during campaigns, even in temperate regions. It is sometimes so prevalent that it exceeds the number of sick from all other diseases put together. It has followed the tracks of all the great armies which have traversed Europe during the Continental wars of the past 200 years. It helped to destroy the British army in Holland in 1748. It decimated the French, Prussian, and Austrian armies in 1792. It was a chief cause of death in the ill-fated Walcheren expedition in 1809. It cut down the garrison of Mantua in 1811 and 1812. Sir James M'Grigor records how fatal the disease was in the Peninsular campaigns; and we know how disastrous it was to our troops during the first winter they passed in the Crimea, in 1854. In the words of Sir Ranald Martin, "It is the disease of the famished garrisons of besieged towns, of barren encampments, and of fleets navigating tropical seas, where fruits and vegetables cannot be procured. During the Peninsular war, the first Burmese war, and the late war with Russia, *dysentery* was one of the most prevalent and fatal diseases which reduced the strength of the armies."

Dr. Clymer also notices, in an article on *chronic camp dysentery*, that the extreme frequency and mortality of this disease in the armies of America during the civil war of 1861 to 1863 gave it interest and importance to the American physician. More than one-fourth of all the cases of disease reported during the first two years of the war was of the several forms of dysentery and diarrhœa. The annual number of cases for the whole army was greater than three-fourths of the mean strength—the ratio of cases being 765 per 1,000 the first year, 852 per 1,000 the second year; the mortality being at the rate of 12·36 per 1,000. Acute dysentery never prevailed as an epidemic in the camps of the American armies during the war.

That it is a dangerous and frequent disease throughout our inter-tropical possessions, the tabular statements furnished by Sir Alexander Tulloch to Sir Ranald Martin, and by Dr. Joseph Ewart, of Calcutta, sufficiently testify.

In England generally, however, *dysentery*, as a cause of death, has been decreasing since 1852, although about 200 years ago it was one of the most prevalent and fatal diseases of London. Yet still, although the disease is less violent and less fatal (for as a cause of *death* it has remarkably diminished during the past ten or twelve years), and although the unfavourable hygienic conditions which were wont to bring about dysentery no longer exist, the active endemic conditions which favour, promote, or are congenial to its development are only dormant, and not eradicated. The disease, therefore, is still sometimes brought about just as in the days of Sydenham or Willis. In no respect, however, do we find that the dysentery of this time differs essentially from the description given by Sydenham more than a hundred and thirty years ago. When we look, therefore, to the history of the disease, and to the nature of its lesions—to its re-appearance from time to time among us, with the same identical characters—there are strong grounds for believing that there is something specific in the nature of the poison which produces dysentery, just as specific as that of *small-pox*, *typhus fever*, *typhoid fever*, *yellow fever*, *scarlatina*, *ague*, or *diphtheria*. But besides the specific identity of the disease, as it now exists, with the disease of former times, there is another point of view from which the history of the pathology of dysentery is especially instructive. It is this:—Like all diseases which have been at the same time epidemic and severe, it has been the subject of discussions as frequent and as varied as its ravages have been severe; and one single description of the disease will not do for a record of the characteristics of all epidemics. Most minute descriptions of the state of the intestines in dysentery have been given by many writers; but, as Dr. Copland justly observes, from his extensive experience, "Dysentery is neither so simple in its nature, nor so unvarying in its seat and form, as some recent writers in this country have stated;" and "that writer will but imperfectly perform his duty who, in giving a history of a most prevalent and dangerous malady, confines himself to the particular form it has assumed during a few seasons, within the single locality, or the small circle of which he is the centre, and argues that it is always as he has observed it."

Dysentery is, moreover, a most formidable disease, on account of its oftentimes insidious nature, from its tendency to recur, and from the after-influences it exerts on particular organs and on the system at large. For these reasons almost all writers on the diseases prevalent in tropical climates place dysentery at the top of the list of severe affections, and refer to it as the cause and origin of many of those chronic and intractable abdominal diseases which so often afflict Europeans resident in tropical climates; and which entails most varied forms of impaired health when they return to European countries.

The prevalence and mortality of dysentery in various countries, and the average rates of sickness and mortality from dysentery and diarrhœa in India, are well shown in the following tables by Sir Alexander Tulloch and Dr. Joseph Ewart:—

Prevalence and Mortality of Dysentery in various Countries, by the late Sir Alexander Tulloch, K.C.B.

STATIONS.	Period of Observation.	Aggregate Strength.	DYSENTERY.		
			Attacked.	Died.	Proportion of Deaths to Admissions.
Windward and Leeward command,..	20 years.	86,661	17,843	1,367	7·7 per cent.
Jamaica,....................	20 ,,	51,567	4,909	184	3·7 ,,
Gibraltar, ...	19 ,,	60,269	2,653	64	2·4 ,,
Malta,......................	20 ,,	40,826	1,401	94	6·6 ,,
Ionian Islands,.........	20 ,,	70,293	3,768	184	4·8 ,,
Bermudas,...............	20 ,,	11,721	1,751	36	2·0 ,,
Nova Scotia and New Brunswick,	20 ,,	46,442	244	18	7·4 ,,
Canada, ...	20 ,,	64,280	735	36	4·8 ,,
Western Africa, ...	18 ,,	1,843	370	55	14·2 ,,
Cape of Good Hope,...	19 ,,	227,111	1,425	44	3·0 ,,
St. Helena,.............	9 ,,	8,973	751	69	9·0 ,,
Mauritius,...............	19 ,,	30,515	5,420	285	5·2 ,,
Ceylon, ...	20 ,,	42,978	9,069	993	11·1 ,,
Tenasserim Provinces,	10 ,,	6,818	1,460	137	9·3 ,,
Madras, ...	5 ,,	31,627	6,639	559	8·3 ,,
Bengal,....................	5 ,,	38,136	5,152	411	8·0 ,,
Bombay,..................	5 ,,	17,612	1,879	151	8·0 ,,

Average Rates of Sickness and Mortality from Dysentery and Diarrhœa among European Troops in India. (Compiled from data contained in Tables XXVI. and XXVII. of *Vital Statistics of European and Native Armies in India*, by Dr. Joseph Ewart, of Calcutta Medical College.)

Presidency.	Periods.	Strength.	Ad-mission.	Deaths.	Percentage of Admissions to Strength.	Percentage of Deaths to Strength.	Percentage of Deaths to Admissions.
I.—From Dysentery alone.							
Bengal, ...	1812 to 1853-4	543.768	100,542	8,873	18·48	1·64	8·82
Bombay,.........	1803-4 to 1853-4	306,978	51,010	4.705	16·61	1·53	9·22
Madras,.........	1829 to 1851-2*	213,587	30,593	2,304	14·32	1·07	7·53
II.—From Diarrhœa.							
Bengal, ...	1812 to 1853-4	543,768	64,823	2,141	11·92	·39	3·30
Bombay,.........	1803-4 to 1853-4	306,978	32,290	551	10·51	·17	1·77
Madras,.........	1829 to 1851-2*	213,587	19.4.8	353	9·11	·16	1·81
III.—From Dysentery and Diarrhœa—as a Class.							
Bengal,.........	1812 to 1853-4	543,768	165,365	11,013	30·41	2·02	6·65
Bombay,.......	1803-4 to 1853-4	306,978	83,300	5,256	27·13	1·71	6·30
Madras,........	1829 to 1851-2*	213,587	50,051	2,657	23·43	1·24	5·30

* Exclusive of 1839, 1840, and 1841.

The morbid anatomy of dysentery has not been described with uniform distinctness, and the anatomical descriptions have, in general, been extremely vague. Medical science has not yet finally settled many points in the pathology of the disease; consequently, the doctrines as to treatment are somewhat uncertain; while the means of prevention are not less imperfectly defined. It has been usual to describe cases of *dysentery* as being either *acute* or *chronic;* but there are also cases belonging to a third class, which may be termed *complex.*

Acute Cases of Dysentery.—In this form the specific lesion in the form of inflammatory action does not confine itself to the tissues of the mucous membrane only. The serous covering of the intestines, or even such solid viscera as the liver, spleen, kidneys, are involved in a disease-process. Ulceration or sloughing of large portions of mucous membrane and exudation go on together, and there may be very little corresponding fever at all commensurate with the severity of the lesions, so that while the disease is acute, it is at the same time, in many instances, of a masked and almost latent nature. Death frequently takes place within the first ten or twelve days in such cases; but the disease may terminate gradually and spontaneously, or as the result of appropriate treatment, by the end of the third or fourth week. On the other hand, the disease may not end so favourably and early; but, evincing a marked and obvious resistance to treatment, may advance unchecked; the morbid changes being slow in progress, often extending over several months, and then the case passes into

Chronic Dysentery—One of the most hopeless and intractable forms of disease which the physician has to treat. Under the influence of the slow morbid changes about to be noticed, the wasting of the tissues of the patient progresses steadily, till a human form, literally reduced to the state of a living skeleton, whose bones are held together by skin and ligament, is all that remains. The skin acquires a dry, bran-like, furfuraceous aspect, and the epithelium desquamates in scales and powdery particles. During the progress of such chronic cases various intercurrent morbid states become developed, not necessarily connected with the primary affection, but forming secondary lesions to the disease, and constituting the third form in which *dysentery* must be studied, namely,—

Complex Cases of Dysentery.—There are various secondary lesions which render cases of dysentery complex, and which are regarded by some as directly connected with the primary affection. There are also secondary lesions connected with antecedent forms of disease, which sustain a renewed impulse to their development by the dysenteric state. These secondary lesions may be shortly stated to consist,—

In (1.) Lesions of the small intestines, and of various solid viscera more or less connected with the dysenteric state; and

In (2.) Lesions which may be referred to the co-existence of certain morbid states of the patient with the dysenteric condition, such, for instance, as the *typhous, scorbutic*, and the *scrofulous* state.

Morbid Anatomy of the Tissues in Acute Dysentery.—The accounts of the morbid anatomy of dysentery are especially confusing; and while the disease has been mainly recognized during life, and defined as a febrile disease accompanied by tormina, and followed by straining and scanty mucous or bloody stools containing little or no fæcal matter, yet the local lesions associated with these conditions have not been clearly defined nor uniformly described.

For example, Chomel and his school considered that the local lesion in dysentery consisted in congestion simply, and tumefaction of the mucous membrane, especially in patches of some extent, so as to form dark-red or purple prominences, from the surface of which the epithelium becomes detached by desquamation. Cruveilhier believed that dysentery was an erythematous inflammation of the large intestine, quickly followed by sphacelus; and he emphatically insists on the point that the follicles and solitary glands have no share in the disease. "It is not," he says, "a follicular inflammation." Rokitansky includes these two forms of lesion as the essential characteristic lesion in dysentery.

The disease, as described by each of these observers, is regarded as a process of rapid, and at first of superficial inflammation, leading inevitably and speedily to mortification of the mucous membrane of the intestine, and unattended by any special disease of the solitary glands. Rokitansky states that, even in the slightest variety of dysentery, the mucous membrane is swoollen and red, and may be removed in the form of a pulp from beneath the furfuraceous and vesicular epithelium. In after stages, and in the severer forms, the mucous membrane becomes gelatinous, and is easily separable, or it passes into a state of sphacelus, black, friable, and offensive. All these observers regard ulceration as having no essential part in the disease-process which constitutes dysentery, and as being of very rare occurrence.

Some of the writers who have described the tropical forms of the disease have been still less distinct as to the details of its morbid anatomy. For example, Twining seems to have followed Chomel in considering the lesion to be simple inflammation of the mucous coat; and Annesley is in a great measure unintelligible as to the points of morbid anatomy which he describes.

It was not till Dr. Parkes published his minute and admirable description of the morbid anatomy of dysentery, as he saw it in India, that we had anything definite on the subject as regards the tropical forms of the disease. He not only showed the very early implication of the glandular apparatus of the great intestine in dysenteric inflammation, but he established the fact, so far as his cases went, that, while ulceration occurs with great rapidity, a case never presents true dysenteric symptoms without ulceration being present. At Moulmein, in India, he investigated, in 1843-44, cases of dysentery in Europeans to the number of fifty, and in Asiatics to the number of twenty. He concluded from these observations that,—(1.) Certain alterations in the glands of the mucous membrane of the large intestine, and sometimes of the ileum, constitute the earliest lesion in dysentery. (2.) That in all cases, when not too far advanced, the mucous membrane presented the appearance of numerous whitish round elevations, of a size varying from a millet-seed to a size so minute that a lens only can show the lesion. These elevations were hard, and being pierced, gave forth a white excretion. Many of these had a black speck in the centre, and were surrounded by a vascular circle. (3.) He noticed that exudation sometimes occurred in points beneath the mucous surface; that such points of exudation had a white appearance, with contents similar to those of the solitary glands. The mucous membrane over these points could be easily rubbed off, leaving an ulcer (Parkes *On the Dysentery and Hepatitis of India*).

The observations of Dr. Parkes were thus opposed to the views just stated, and led to extended investigation, by which such contradictions may be reconciled; and it was determined that differences of climate do not cause any essential difference in the structural changes which accompany dysentery. The observations of Drs. Craigie and Abercrombie in Scotland, in 1837, prove this; and also those of the late Dr. Baly, in 1847, as regards England. Drs. Cheyne, Graves, and Mayne have demonstrated the same fact as to Ireland. By the records of epidemic dysentery at Prague and elsewhere, as described by Dr. Finger and others, the observation holds true as regards the dysentery of Europe generally; and by comparing these records with the well-recorded cases of those who have seen the disease in the tropics, both in civil and in military life, it will be seen that the true dysentery of tropical and temperate climates does not differ *as to its anatomical signs* in any essential particular.

The descriptions of the disease in our own country, as given by Cheyne, Craigie, Abercrombie, and Baly, all agree in recording the inflamed condition of the mucous membrane of the colon, with its small round ulcers, pulpy softening, or sphacelus of some portions, and ulcers of various forms left by the separation of the sloughs, and enlarged firm tubercles, which no doubt were the inflamed solitary glands. Again, Sir John Pringle, M. Broussais, and other historians of dysentery, found the same lesions in the dysentery of the camps in the continental campaigns of Europe; and Broussais expressly states his belief "that the ulcers of the large intestine had their origin in the solitary glands." Thus the "tubercles," the "pustules," and the "small-pox-like elevations" (now known to be lesions of solitary glands) of the mucous membrane have been most minutely described by Hewson, Pringle, and Davis; and the last of these observers describes in graphic language the fatal dysentery of the Walcheren expedition, and shows that its anatomical characters are similar in all respects to the forms of dysentery which have been described in this country.

We have therefore only to compare all these records with the histories of tropical dysentery, as given by Zimmerman, Annesley, Pringle, Copland, Dr. Hunter, Chisholm, Ballingall, Parkes, Morehead, Sir Ranald Martin, Tait, Macpherson, and others, to know,—"whether any peculiar character of the anatomical changes in the large intestine essentially distinguishes the dysentery of intertropical countries from the dysentery of this and of other temperate regions." Dr. Abercrombie admitted identity in the nature of the dysentery; but, that the extent of the intestine affected varies considerably. Dr. Craigie showed that the lesions in dysentery occur in two forms; one continues over the surface, the other limited to the *muciparous follicles*, which become enlarged, indurated, and ulcerated; while, on the other hand, Dr. Baly has shown that all the well-marked varieties of structural change in the large intestine occurring in tropical dysentery are likewise found in fatal cases of the disease occurring in our own country.

Seeing, then, that the descriptions of the morbid anatomy have been so much at variance with each other, several questions suggest themselves, namely,—(1.) Whether distinct epidemics are characterized by distinct local lesions? (2.) Whether two or more distinct diseases have not been confounded under the one name of dysentery? Or, (3.) Whether the various local lesions described by different writers are only so many varieties, forms, or types of the same disease-process—a process modified in particular cases by constitutional peculiarities, or by other circumstances. This latter view is the one most consistent with observation; and it is in accordance with what we know of the history of many of the *miasmatic* diseases, such, for example, as *true yellow fever*, *remittent fever*, *diphtheria*, and the like. There is some evidence, also, to show that there is a lesion of the colon not belonging to true dysentery —a *colonitis* in which the connective tissue of the gut beneath the mucous membrane is implicated in the first instance, rather than the glandular tubes and vesicles. The result is a diffuse gangrenous inflammation of the mucous membrane, the resulting ulcers not differing from the ulcers originating in the glands by any characters at present recognizable (COPLAND, PARKES.)

In this country it is believed that the lesion in dysentery is confined, for the most part, to the colon and rectum; but that in tropical dysentery the whole course of the colon, and sometimes a considerable portion of the small intestines, are implicated. But, except when the case is associated with *scorbutus*, the small intestines are not involved. Lesions so extensive, while they are common in India, are rare in this country; yet they do occur, and are not uncommon in the south of Europe, in Turkey, and the coasts of the Mediterranean. Therefore, as regards the extent of the lesion, there is no constant or distinctive characteristic between tropical dysentery and the dysentery of more temperate climates.

In both regions the anatomical changes comprehend redness of the mucous membrane, preceding further changes; loss of the substance of the mucous glands by pulpy softening of tissue, sloughing, or ulceration; the detachment of diphtheritic casts of the intestine, or sloughs of tissue. The type of the inflammatory lesion in dysentery is essentially diphtheritic (NIEMEYER). The mucous structures affected are infiltrated with a fibrinous exudation, which enters deeply into the tissue, and which eventually dies, with loss of substance of the intestine, which may or may not heal.

In describing the morbid anatomy of dysentery, the reader is referred to the nomenclature of the gland structures, given in a footnote at page 546, vol. i.

The structure of the colon in the healthy state differs in many important particulars from that of the small intestine. It is remarkable for the absence of folds and villi, and for the presence of more or less dilated sacculi, which give form and shape to the excrement. The minute tubular glands are thicker in proportion to their length, compared with those of the small intestine; and the intertubular connective tissue is considerable,—a structure which takes an important share in the lesions of dysentery. These tubular glands are lined by columnar, cylindrical, and transition forms of epithelium; and the solitary lenticular glands are sometimes closed vesicles (ALLEN THOMSON, PARKES, BALY), and sometimes open follicles. When closed, they are not visible; but if distended, they may be seen with a lens; and when open, a dark depressed point marks the separation in the tubular gland structure which leads to the open follicle. The tubular glands radiate round this spot, which corresponds to a depression indicating the empty vesicle below. These solitary vesicles have thick walls, and are said to be more abundant in the cœcum and rectum than in any other part of the great intestine. This statement leads to the question which has been mooted in relation to these solitary gland lesions, namely,—"Are these lesions of the so-called solitary glands really due to the enlargement of previously existing solitary glands or their germs? or, Are they new formations altogether?"

A similar question is at issue regarding the granulations on the eyelids and conjunctivæ, associated with *purulent ophthalmia* and *granular lids* (see p. 440 of this vol.), (STROMEYER, FRANK, MARSTON). In this disease we have *new formations* of vesicular-like granulations, as well as enlarged follicles; but these are more numerous than the glands have ever been seen to exist in the healthy state. Observations somewhat similar have been made regarding the vesicular glands of the stomach (HANDFIELD JONES). It may be, therefore, that not a few of the "tubercle nodules," the "pustules," the "small-pox-like elevations," and what we call solitary or lenticular glands, are in reality new formations altogether, resulting from increased cell-growth, within the meshes of the connective tissue which binds the mucous gland tubes together, and connects them with the submucous muscular layer. In this respect their formation would be analogous to that described by Virchow in connection with tubercle, and not to be distinguished histologically from a newly formed grey tubercle nodule. So independent have these lesions been believed to be by some, that one observer of Indian dysentery (MURRAY) described a "pustular form of dysentery," which he considered to be in all respects analogous to small-pox on the skin, beginning with the formation of an independent papule and the development of a subsequent pustule, as in that disease of the skin. This view of the subject is of some importance in pathology, as it is related to the specific nature of dysentery and the poison cast off from the mucous membrane, by which it is believed that the disease is propagated like typhoid fever and cholera (DR. WILLIAM BUDD).

Seeing, therefore, that the anatomical signs of dysentery are so constant over all the world, it may be asked, How have modern writers given such contradictory accounts of the morbid anatomy of the disease? The best writers have differed on points of observation simply,—(1.) Some deny the necessary occurrence of ulceration; (2.) Some deny any special participation of the lenticular glands; (3.) Some believe that new formations arise, which are similar in appearance to those small glands; (4.) Some, on the other hand, believe the disease, at its commencement, to be always seated in these small glands; (5.) Some regard dysentery as essentially an erythematous inflammation, terminating in gangrene; (6.) Others believe that such gangrene is a very rare variety of the disease.

To explain such discrepancy, it may be said that,—

(1.) Observations have been too limited, and not exact enough, to give an accurate and comprehensive view of the morbid anatomy of the disease; that dysentery, although a simple and uniform disease, so far as its anatomical signs are concerned, is yet liable to constant changes of type, from its remarkable proclivity to complicate prevailing fevers (specific or endemic), as well as other diseases (e. g., *enteric fever, malarious fevers, fever from the poison of animal effluvia, scurvy, syphilis, phthisis, measles, variola,* and the like), and to be itself complicated by them.

(2.) The healthy or normal anatomy and histology of the mucous membranes are only yet beginning to be understood and studied minutely at our Schools of Medicine; and that many hitherto otherwise excellent descriptions of dysentery are deficient in anatomical exactness and precision, especially as to the minute structures implicated.

(3.) The examination of the colon, upon which descriptions have been based, has often been incompletely done, as Annesley very correctly pointed out—many never having been opened.

(4.) The inherent difficulties of the subject itself, such as the impossibility of seeing the state of the diseased membrane till after death.

But, from all the observations that have been made, there can be no doubt that the anatomical signs of true dysentery are primarily derived from inflammation of the solitary lenticular follicles of the large intestine, tending in the first instance to infarction (*i. e.*, intumescence and congestion), and subsequently to ulceration and destruction of the gland tissue. The disease, extending by a similar process, ultimately involves the tubular glands of the general mucous membrane, which tend to soften and to be cast off as an exuvium or slough, exposing the submucous connective tissue or even the muscular coat of the intestine. It is the mucous membrane of the great intestine, and especially of the rectum and lower portion of the colon, which is the seat of these characteristic lesions in dysentery. The exudative process is generally diffuse, involving the whole of the tissues of the mucous membrane in a diphtheritic process of infiltration. The diseased part looks as if it were covered with a bran-like coating, especially over the summits of the folds of mucous membrane, which are deeply reddened by ecchymosis and injection, and infiltrated by the grey-like exudation. This bran-like coating cannot be scraped off without loss of substance; and the submucous connective tissue below is œdematous and swollen. In some cases, however, the exudation is mainly seated in the solitary glands, in the first instance, and neighbouring mucous tubular glands, whence it spreads by infiltration to the neighbouring parts.

In the scorbutic form of dysentery, or in dysentery occurring in persons whose nervous or vital powers are feeble, or below *par*, as in the aged, infirm, or in the paralysis of the insane (conditions in some respects similar in their influence as to *scorbutus*), a diphtheritic exudation covers to a considerable thickness not only the mucous surface of the colon, but (as an almost constant and pathognomonic morbid sign) the same lesion is seen covering the mucous surface of the small intestine, extending upwards from the ileo-colic valve; and, as Dr. Parkes has noticed, this exudation grows or is laid down especially in the course of the blood-vessels ramifying from the mesenteric attachment transversely across the surface of the gut, and occupying especially the prominences of transverse rugæ. This form of dysentery prevailed to a great extent among the soldiers who died at Scutari during that period of the Russian war when scorbutus prevailed. Some of the cases recorded by Dr. Davis in his admirable description of the dysentery so fatal to the British troops in the famous Walcheren expedition were also of this nature. Dr. Finger, of Prague, and Dr. Maine, of Dublin, and Dr. Baly, have recorded similar cases; but one of the best accounts of this form of dysentery is that which Dr. Clouston records as having prevailed in the Cumberland and Westmoreland Asylum, and which he believed to have been caused by the effluvia from a field irrigated with sewage. The regular diet of the asylum inmates consisted of 24 ounces of animal food, 14 pints of milk, 16 ounces of suet dumpling, 7 pints of oatmeal porridge, 78 ounces of bread, and 7 pints of tea per week (*Med. Times*, June 3, 1865). No fresh vegetable diet seems to have been provided for; and diarrhœa does not seem to have been uncommon.

In typical cases of the outbreak described by Dr. Clouston—

"All the abdominal organs would be found healthy until the small intestine was examined. This, too, would be normal up to within five or six feet of the cœcum. The mucous membrane would then begin to appear reddened in small spots or rings round the gut. Six inches farther down the redness would be universal, and the membrane would begin to be thickened and corrugated into folds, like small *valvulæ conniventes.* A few inches farther down, a yellowish dirty-looking deposit would be seen over the mucous membrane in rings, very thin where it began, but gradually becoming thicker and more continuous till near the cœcum it would be one-eighth of an inch in thickness. The swelling of the mucous membrane would also increase downwards, and the folds running across the gut would become more prominent. These, with their coating of deposit, made the inside of the bowel look like a series of thick transverse ridges, covering its entire surface. This deposit, when examined, would be found to be soft on the surface, but getting more firm towards the mucous membrane, with which it incorporated itself so that it could not be scraped off without leaving the fibrous covering of the muscular coat exposed as a highly vascular, raw-looking surface. This deposit, though on the surface a soft lymphy-looking substance, yet towards the mucous membrane it assumed quite the consistence and appearance of a soft fibrous membrane. The cœcum, when examined in such a case, would be found in the same state as the lower part of the small intestine, with two or three ragged ulcerations the size of beans. In the ascending colon the ulcerations became deeper and larger; still the lymphy deposit on the surface of the mucous membrane became thicker and more feculant in colour. Towards the transverse colon the inside of the gut was one mass of large irregular ulcers, with patches on the deposit between them. The colour of the surface was almost black, and this continued down to the very lower part of the rectum. The mesenteric glands opposite the affected parts of the small and large intestine were enlarged and dark-coloured, and on section were soft and pulpy in consistency. Such were the general appearances in a case that had lasted for about a month" (*Med. Times and Gazette*, June 10, 1865, p. 598).

The several lesions which I have been able to distinguish throughout the numerous dissections of cases of dysentery which I have made, may be stated as follow:—

1. Exudation obvious on the surface of the mucous membrane of the rectum and colon; but also extending deeply by infiltration.
2. Exudation not obvious to the unaided eye, but which was seen, in all the cases examined by the microscope, to fill the mucous tubular follicles of the large intestine.
3. Exudation obvious to the eye, and demonstrable by microscopic examination, as being developed in the solitary vesicular or lenticular glands of the large intestine.
4. Changes in the exuded material, which tend first towards its organization, and subsequently to its destruction and removal by ulceration.
5. Softening and ulcerative changes in the tissues of the mucous membrane itself, and in the glands.
6. Similar dysenteric lesions extending into the *small intestines*, as in scorbutic states of the system.

The extent of the exudative process varies much. In some cases a considerable portion of the *colon* and *rectum* only is affected; in other instances not only is the whole of the great gut the seat of some form of the exudative process, but the lower portion of the small intestine also. The most commonly affected portions, however, are the *rectum*, the *sigmoid flexure*, and the *descending colon*. When the *caput cœcum* of the colon is involved, the *vermiform appendix* participates in the process. Creamy-like exudations have been seen to fill its tubular glands, which in some cases were opened up by ulceration.

In the least severe cases an opportunity does not often occur to see the changes in an early stage; but when life is cut short by some other malady, changes of the following nature may be seen:—

The exuded mucinous material, in its more recent state, forms a layer, which varies from a thin but opaque membrane to three or four lines in thickness, of homogeneous substance, tolerably consistent, and capable of being detached and raised in flakes from the subjacent mucous surface. During the earlier stages of the disease the surface of the mucous membrane appears unchanged below, except, perhaps, by the existence of a little increase of vascularity. The colour of the exuded matter may be uniform, or red, white, or pink in patches, and discoloured in some instances by intestinal gases, the biliary secretion, or by the admixture of blood, and the changes consequent thereon. The most common appearance in severe cases is that of a dark olive-green, passing into a bluish-black. The surface of the exudation may be uniform, or the whole aspect may be mammillated, with here and there a *mammillation* projecting greatly above the others in a fungating mass, surrounded by dark fissures in the exudation. These fungating masses are soft towards their centres, with numerous red vascular points here and there on the surface. A section through the mass shows the base thickened and firm.

The dysenteric process, as seen after death, is generally found to have advanced farther in one part of the intestine than in another; usually, it may be stated to have been farther advanced in the *rectum* than in the *descending colon*, and farther in that part than towards the head of the large intestine. In well-marked and extreme cases the entire mucous surface, from the *caput cœcum* to the *rectum*, may be seen to present all the possible stages of the dysenteric process. Three stages can in general be distinguished, namely,—

(1.) Ulceration of the exudation, and mucous membrane more or less advanced towards the *rectal end* of the great intestine.

(2.) Exudation in various forms towards the *middle* of the *colon* upwards from the *rectum*.

(3.) The exudative process visible microscopically in the tubular glands, and sometimes also obvious to unaided vision in the solitary vesicular glands of the great intestine towards the *caput cœcum*.

One of the best descriptions of the morbid anatomy of dysentery in the English language has been given by the late Dr. Baly in his Gulstonian Lectures for 1847. He describes *three* different forms of lesions as seen by him amongst convicts at the Millbank Prison; and these three forms he believed to correspond with three degrees of severity of symptoms during life,—

(1.) He recognized a swollen condition of the solitary glands, forming round prominences on the surface of the mucous membrane, of various sizes. In colour these were pale, or red round the base, and dotted at the summit with a vascular spot. These appearances would occur about the eighteenth or twentieth day. At an earlier period the congestion round the glands would be more intense; while at a later period the summits of the prominences would become disorganized. Minute yellow sloughs subsequently form, which, becoming detached, leave an ulcer previously occupied by the gland. The mucous membrane around participates in the process. It is red, tumid, and covered with an aphthous layer of lymph to the extent of one or two inches around, with three or four solitary glands prominent in the midst. The ulcers which form result from sloughs rather than from ulceration, and the disease still remains *not severe* as regards the amount of tissue involved; but *as to duration,* the illness may be prolonged and tedious; and the solitary gland cavities may enlarge very much—so large as a horse-bean.

(2.) In more severe forms a greater variety and extent of tissue is involved; and especially of the tubular glands. There is great redness, tumefaction, and softening of tissue; and along with the change of colour and of texture the secretions are greatly altered. The clear mucus is highly charged with albumen, and subsequently with blood.*

These changes occupy principally the prominences of the transverse rugæ. The exudation on the free surface in the recent condition may form a thin opaque membrane three or four lines thick. It is homogeneous and of considerable consistence, so that it may be detached, and raised in flakes from the subjacent surface. With the exception of increased vascularity, the subjacent mucous surface appears still unchanged. A microscopic examination of this exudation shows that it varies with the severity of the case to some extent. In mild cases it is simply particles of epithelium mixed with amorphous granules. In more severe forms the exudation consists of fine germs with nuclei, mixed with elongated cell-forms (connective tissue cells); and the examination of carefully prepared sections shows that such exudation mainly commences in the tubular glands, and by proliferation subsequently spreads over the mucous surface as described. In Dr. Clouston's cases examination of the fibrinous layer (over the small intestine), in the fresh state, showed its structure to consist "of nucleated cells like pus-cells, furiform cells with nucleus, and a fibrinous material between them." The blood-vessels underneath the "fibrous layer were enlarged and very tortuous, and on the free surface of the mucous membrane, between the villi, they could be seen torn and open-mouthed" (*Med. Times,* l. c.)

Exuviæ, or casts of the intestine, may be thrown off in large masses or shreds, leaving a raw-looking vascular surface underneath; and in some respects this process and these casts are analogous to similar phenomena in *croup*, *diphtheria*, *dysmenorrhœa*, and to *typhoid fever*. By carefully examining the evacuations, important information may be obtained as to the nature of the process going on in the intestines. Dr. Goodeve, of Calcutta, has made some valuable observations of this kind. He recommends that the evacuations should be washed with water, so as to get rid of the fæcal matters entirely, and to leave the sediment, which is the product of the colonic disease, free from bile, fæcal matter, and offensive smell. This sediment has been found by Dr. Chuckerbutty to consist of ropy, gelatinous, branny, or thready mucus; lymph in shreds or granular masses; pus; fæces; and sloughs. He observed that patches of membrane, half an inch or an inch or more in size, are cast off as sloughs. These exuviæ are thin, membranous, and sometimes infiltrated with pus; or they are thick and of a yellowish-brown colour. It is not till after the *eighth* or *twelfth* day of the disease that such sloughs are cast off. In these respects they may be considered similar to those cast off from Peyer's patches in typhoid fever. After these shreds are cast off the symptoms diminish, and the patient often gets well rapidly.

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* Some account of the chemistry of the stools in dysentery has been given by Oesterlen, who describes an excessive elimination of water and of albumen. It is very desirable that this observation should be verified in India. The stools should be collected free from urine; and the albumen should be estimated separately from any insoluble sediment. If in severe cases two and a half ounces of albumen are passed in twenty-four hours by stool, it is impossible to over-estimate the importance of such an occurrence.

In a series of cases, with remarks illustrative of the "Pathology of Dysentery," Dr. Chuckerbutty describes the following conditions as indicated by the various kinds of *exuviæ* cast off in dysentery and recovered by the washing process of Goodeve:—

Character of Sloughs.	Conditions Denoted.
Ecchymosed,	Abraded or minute ulcers; intestinal apoplexy.
Compact grey or light-yellow,	Acute phlegmonous dysentery.
Thick pus—infiltrated mucus,	Erysipelatous dysentery.
Ragged,	Gangrene.
Dark olive,	Secondary gangrene of mucous coat.
Thin black, plain, or tubular,	Primary gangrene of mucous coat.
Shreddy,	Gangrene in either mucous or cellular coat.
Molecular, or putrilage,	Disintegration of tissue.
Flaky epithelial,	Commencing gangrene of mucous membrane.
Shaggy,	Violent inflammatory action.
Free filamentous (simple),	Primary gangrene in submucous tissue.
" (pus-infiltrated),	Submucous cellulitis.
Ring-shaped,	Ring-shaped ulceration in mucous folds.
Discoid,	Circular ulcers in ecchymosed patches.

During the shedding of the shreds the patients are much griped, and they pass with straining the sanguinolent masses, or slimy mucus in small quantities, and generally without fæcal matter, fifteen or even twenty times a day. Then a period of cure and improvement supervenes, with diminution of the stools or of the fæcal discharges,—not simply by resolution, but a termination by elimination of the specific sloughs or lesions which have formed in the course of the disease. In this respect the phenomena seem analogous to what occurs in typhoid fever. When these membranous flakes are not shed, but retained and ultimately separated in large pieces, there is considerable danger attending the process. Discharges of blood and fatal hæmorrhages may ensue. Morehead records eight cases of this kind in India, and four of them were fatal, of whom one died from hæmorrhage. To account for this hæmorrhage, it has been observed that changes go on between the intertubular connective tissue and the substance of this exudation, which tend to its organization or supply with blood, and subsequently to its destruction, separation, or removal by ulceration. In vertical sections, down to and through the mucous membrane, I have seen fine blood-vessels, in loops and bulbous cœcal ends, shooting upwards beyond the mucous surface into the exudation; and when such exudation was forcibly removed from off the mucous surface, the membrane on which it lay was found to be highly vascular, and numerous minute ruptured vessels showed their torn mouths, by minute points of exuding blood. Dr. Morehead records a similar observation as regards the connection of the exudation with the subjacent mucous tissue, "through the medium of what appeared to be small capillary vessels, the mucous membrane beneath being vascular" (*Researches on Disease in India*, p. 241). In a case that proved fatal at the end of six weeks, and in which the gut was so friable that almost the least current of water tore it up, Dr. Clouston noticed that "in the rectum blood-clots projected from the open mouths of arteries."

When such a state of vascular action in the mucous membrane and exudation has existed for a lengthened period, the tissue of the gut becomes greatly thickened, and at the same time less coherent. These thickened portions grow luxuriantly, just as isolated patches in a field of green corn grow more luxuriantly than others, being supplied with a greater amount of nutritive material, as the thickened patches of mucous membrane are supplied with more enlarged, more tortuous, and more extensively distributed loops of blood-vessels. These thickened masses of dysenteric exudation continue to fungate and grow from a hardened base, from which the numerous blood-vessels pass into the growing masses. In this way the warty condition of the mucous membrane in chronic cases undoubtedly results. The new material evinces a disposition to contract. The calibre or bore of the gut gradually diminishes, and its texture becomes so brittle that slight force in pulling up a piece of such intestine out of its place will readily cause it to *break* asunder.

As to ulceration, it may be readily understood, with such varied lesions, that the formation of ulcers does not take place in any uniform mode; and the following statement is given as a summary of the processes from which ulceration may proceed:—

(1.) It may occur after intumescence, softening, and simple ulceration of one or of several lenticular solitary glands.

(2.) After intumescence, softening, and sphacelus of many solitary lenticular glands and the intervening tissue in one mass.

(3.) After softening of the tubular structure and the detachment of sloughs, ulceration follows the intumescence and proliferation of growths from the tubes which cover the surface as a "croupous," "catarrhal," or "diphtheritic" exudation, and to which the name of "aphthous erosions" has been applied.

(4.) After submucous inflammation and new growths, with fibrinous and mucinous effusion.

(5.) After intertubular inflammation, and after inflammation surrounding the base of inflamed glands.

(6.) After the formation of submucous abscess.

(7.) By changes of an ulcerative nature, commencing in the vascular exudation itself, as in scorbutic cases.

The *circular* ulcers, for the most part, originate in the solitary glands (PARKES, BALY), or in circular patches of tubes (MOREHEAD), similarly to the stomach ulceration, as described by Drs. Handfield Jones and Brinton; or such circular ulcers may result from both, as when a solitary gland is destroyed it carries with its destruction some of the adjacent tubes. In such cases the colon presents prominent little masses, about the size of a pea, which burst readily on pressure, and give forth fluid contents like pus. Such abscesses may open spontaneously upon the mucous surface through the short canal leading from the vesicular gland (now an abscess), imbedded in the submucous tissue, and between the tubular glands. They undermine the tubular gland substance, and carry off shreds or patches of the surrounding tissue. They may thus be seen in all stages, and sometimes almost symmetrically arranged in a double row along the colon (BLEEKER, MOREHEAD). Many of these little abscess cavities are also formed below patches of thick exudation (HASPEL).

The *transverse ulcers* are due to the transverse arrangement of folds, on which the exudation and textures ulcerate, as already described; and I have known the transverse *rupturing* of very thick exudation mistaken at *post-mortem* examinations for ulceration, on seeing the raw vascular surface of the tissue exposed below at the bottom of the rent.

Microscopically the exudation in its most recent condition may be seen to be composed of fine germs and nuclei, with elongated nuclear cells. It appears to be chiefly exuded into the follicular and tubular apparatus of the mucous membrane, and gradually accumulating there, is pushed upwards to the mucous surface, which it finally overspreads as a whitish coat, coherent and uniform, susceptible of vascular organization, and tending to ulcerate.

Morbid Anatomy of the Tissues in Chronic Dysentery.—In the true chronic form of dysentery the exudation already noticed undergoes various changes. It may be thrown off from the mucous surface altogether, leaving that surface bare and raw-looking, as if ulcerated; but a close inspection will show that the surface is entire and highly vascular. If it is not thrown off, it may undergo a considerable amount of organization; after which it appears that a process of ulceration may be established upon its surface, just as in any other soft tissue. This ulcerative process may extend through the whole exudation, even to the surface of the mucous membrane, which it may penetrate also, and involve the tissues of the intestine in the ulcerative process close to the peritoneal coat. Perforation of the peritoneum is by no means uncommon.

In the chronic forms of dysentery there is a very constant morbid change to be observed, consisting in the deposit of black granular matter on some parts of the mucous membrane. It may be regarded as the result of excessive vascular action, and of subsequent changes in the extravasated blood, elements which mark the site of the melanic spot (pigmentary degeneration; see p. 96).

The sigmoid flexure of the colon is perhaps the most frequently and the most extensively diseased, and the lesion is most expressed towards the rectum. In very severe cases the exudation extends over the whole extent of the mucous surface of the *colon,* which appears covered with black, grumous, carbonized-looking masses, even to the upper part. Ulceration is most frequently seen in the *sigmoid flexure,* destroying at once the exudation and the mucous membrane, so as to expose the muscular tissue of the gut, which is red and irritable. An *appearance* of ulceration often extends in lines across the gut, so as to embrace its whole calibre in some parts. This is sometimes, however, only an appearance of ulceration, caused by the separation of the exudation when it is thick, exposing the highly vascular mucous surface below, which looks raw and ulcerated. When the gut is opened in the usual way after death, and extended on a flat surface, the change from the hitherto curved condition of the intestine is so great as to cause rupture or separation between masses of exudation, especially in places where it is thick; thus giving rise to the appearance noticed, and which has sometimes been described as ulceration. In long-continued chronic cases the rectum is generally studded over with punched-out-looking ulcers, with bloodless bases and thin anæmic edges; and the melanotic deposit, already noticed, is here seen in the greatest abundance. Evidence of healed ulcers, with partially renewed mucous tissue covering them, are not uncommon in this locality, their place being indicated by the amount of black matter. The gland tissue, however, is not reproduced in the cicatrix substance.

Morbid Anatomy of the Tissues in Complex Cases of Dysentery.—In the class of dysenteric cases which may be called *complex* there are a variety of lesions, the pathological significance of which as to extent, form, origin, and locality, renders the cases of dysentery in which they are found of a very complex kind. The lesions which chiefly tend to render cases of dysentery complex, are,—

(1.) Extension of the dysenteric process over the mucous membrane of the small intestine.

(2.) Deposits and ulcerations in the glands of Peyer, as well as in the general tubular structure near the ileo-cœcal valve.

(3.) Atrophy of the glandular parts of the mucous membrane of the alimentary canal.

(4.) Secondary lesions of serous membranes.

(5.) Secondary lesions of solid viscera in the cavities of the *abdomen* and *thorax.*

(6.) Secondary lesions due to the syphilitic, scorbutic, typhus, or tuberculous states, or to the influence of malaria.

In some rapid and acute cases of *dysentery* (five out of twenty-eight cases, as observed by Dr. Baly) it has been noticed that the process by which the *dysenteric lesions* were developed in the large intestine extended beyond the ileo-cœcal valve, and brought about an action in the small intestine similar to that in the colon. As much as the lower two-thirds of the ileum have been involved in this process, while the upper portion has been found intensely congested. In one case of *dysentery*, Dr. Cheyne says he found an exudation of lymph extending nearly over the whole of the jejunum. If the stomach participates in the disease, the mucous membrane may be merely diffusely inflamed, or of a red or violet colour, its surface granulated, and its texture broken by the slightest touch. More commonly the colour of the mucous membrane is natural, but on its surface a number of ecchymosed spots, or small ulcers, are seen, with edges so sharp, clean, and perpendicular, that they appear as if made with a punch. In other cases the tubular glands, as well as the solitary and aggregate glands of Peyer, have shown various stages of morbid action. The absorbent mesenteric glands are rarely affected (BALY); but, except in cases of secondary hepatic abscess, they were found enlarged and inflamed in all cases of Indian dysentery (PARKES), and also in the dysentery associated with *scorbutus*.

By far the most common condition, however, in chronic cases of *dysentery* especially, is that which is due to atrophy of the mucous membrane. As an atrophic change, it may be ascribed to the general wasting (marasmic) processes which take place to a great extent throughout the system in cases of *chronic dysentery*. In this complex state the mucous membrane of the *small intestine* appears pale, thin, and worn,—a condition which pervades the greater part of the alimentary canal, and which is especially made manifest in the living as well as in the dead by the condition of the mucous membrane of the mouth. On turning down the lips, the mucous glands are seen distinctly projecting through the thin pale labial and buccal mucous membrane. When such cases are examined after death, the structure of the solitary glands and of Peyer's patches are found to be degenerated and wasted; no gland-cells are to be seen, and their place is supplied by fibroid tissue, with some vascular injection round the reticulated spaces. In other instances a deposit of black pigment surrounds the locality of the glands, which indicates the long-continued process of vascular action previous to their atrophy. Associated with this general atrophic state, some gland-patches may be observed in an apparently opposite state—that is, distended, and sometimes engorged; but, on examination, their contents appear to be undergoing a molecular, melanotic, and generally fatty degeneration, probably preparatory to complete evacuation and destruction of the gland-element. These two apparently opposite conditions, co-existing in the same cases, appear to indicate that the one condition is but the antecedent of the other; and that the atrophy and degeneration is the last result of a series of morbid processes commencing in the engorged gland-cavities.

In parts of the mucous tissue which exhibited the opposite conditions of extreme hypertrophy and extreme atrophy, the specific gravity of the former indicated 1·046, while the thin and wasted part of the intestine indicated a specific gravity of 1·036 to 1·030.

There is now abundance of evidence to show that in some endemic cases, or in epidemics of *dysentery* in some places, there is a tendency to the secondary affections of organs or parts, during or subsequent to the development of the dysenteric process. Some look upon these secondary processes in relation to the dysentery as in the relation of effect following a cause; or that there is an immediate and direct connection between the primary dysenteric process and the secondary lesion. Such a relationship has not been shown to exist in all cases; and it is more probable that the *dysenteric* process, when it operates on the system during a protracted period, predisposes, as many other morbid states do, to the development of secondary local lesions in distant parts.

The arachnoid, the pleuræ, the pericardium, and the peritoneum have each and all of them in some instances been the seat of opacities or of fluid exudations in dysenteric cases.

Of morbid states of the solid viscera, associated with dysentery, by far the most frequent complication is that with the *kidney* and the *liver.* With regard to the kidneys, their relation to the bowel affection is as yet obscure; but in mild cases, proceeding to a favourable termination, there is no albumen and no casts in the urine. When, on the other hand, the dysentery is severe, it continues some time before exudation appears in the urine, and then its occurrence is preceded and attended by putridity of the copious stools, by *status nervosus,* collapse, and paralytic phenomena. If the renal affection occurs early, so much more severe is the case, and death usually speedily ensues. The kidneys after death are seen to be highly congested, the tubes loaded with exudation, cells, and detritus (Zimmerman, *Syden. Society Year-Book* for 1861).

The association of hepatic disease with *dysentery* would seem to be most frequent in the climate of the East Indies, and in such climates as have a similar influence (MARTIN). In the Bombay Army, out of thirty fatal cases of *dysentery,* twelve were attended with hepatic abscess (MOREHEAD). Dr. Macpherson, Sir James M'Grigor, Dr. Parkes, and Mr. Henry Marshall, gave similar statistical results of their experience at Calcutta, Moulmein, and Ceylon. The French surgeons in the province of Oran, in Algeria, state that hepatitis and consequent abscess were frequently coincident with dysentery. Dr. Parkes observes, that if the functional morbid state of the liver is to be judged of by chemical analysis of the secretion of that viscus, the liver is found to be diseased, more or less, in every case of dysentery. Dr. W. J. Moore, Assistant-Surgeon of the Bombay Army, has collected a valuable series of statistics, which sets in a stronger light than hitherto the relation of hepatic *lesions* to dysentery. The records of five independent observers show *lesions* of the liver (not being abscesses) in about 57 *per cent.* of the cases; while the number of cases in which *abscess* was observed averages about 18 *per cent.* (Grant's *Annals of Military and Naval Surgery,* vol. i., p. 227). The tendency to hepatic complication was found in Algeria to increase with age, and with the length of service in that country. It appears, however, that hepatic *abscess* is but rarely associated with dysentery in natives of those warm climates; and amongst British subjects in their native climate it seems equally rare. In the Millbank Prison, "out of many hundred cases, not one has been complicated with hepatic *abscess.*" It does not appear, however, that the influence of the climate of the East alone on Europeans tends to the hepatic complication, for "in the Peninsular Army, under the Duke of Wellington, the spleen, the liver, and the mesentery were generally found diseased in cases of dysentery; so were these viscera in the epidemic dysentery of Ireland" (MARTIN). In the dysentery of the allied armies in the hospitals of Scutari and the Crimea during the late Russian war, hepatic abscess was of rare occurrence. Dr. Budd attempts to explain how hepatic abscess is a *consequence* of dysentery through the vitiation of the portal blood from the morbid intestines. But the evidence tends rather to show that the hepatic lesion and the dysentery are each excited by the same cause. If Dr. Budd's theory were correct, we ought to have liver abscess a common occurrence after ulcerations of typhoid fever, and after those of tuberculous lesions of the intestines; but we do not find that such a lesion of the liver is usual in such cases.

Regarding hepatic complication in dysentery, the following conclusions may be stated:—

(1.) That dysentery, in a great number of cases, more than a half, commences and runs its course complicated by obvious functional hepatic disease.

(2.) That the hepatic disorder and the dysentery acknowledge a common cause and disease-process.

(3.) That about 18 *per cent.* of the fatal cases of *dysentery* are complicated with hepatic abscess; and about 57 *per cent.* with hepatic lesions.

(4.) That in a few of these cases ulceration of the intestine may be the primary disease, and the source of the hepatic abscess by the phenomena of thrombosis and embolism in connection with the pelvic veins and veins of the mesocolon.

The occurrence of hepatic abscess with dysentery has been generally viewed as a result of phlebitis; but Dr. Parkes, after the most careful observation of such cases, never found the slightest trace of inflammation in the small veins of the intestine, while no direct proof has been advanced of the mediation of the portal blood in the process; and in conclusion, writes Dr. Henoch, "I believe we must give the preference to that view which regards the two diseased processes, dysentery and abscess of the liver, as running their course together, dependent upon one and the same cause; in favour of which view is the circumstance, that in hot climates abscess of the liver very frequently occurs associated with remittent fevers, or consecutive to them, without dissection exhibiting any ulceration of the mucous membrane of the intestine." (*Brit. and For. Med.-Chir. Review*, July, 1854; see also Dr. Morehead, in *British Medical Journal*, March 20, 1869.) The comparative frequency of the occurrence of hepatic abscesses may be seen from the following statement:—

In *Calcutta* General Hospital they occur at the rate of 13·1 *per cent.* (MACPHERSON); in the Medical College Hospital, at the rate of 25·9 *per cent.*; in *Bombay* General Hospital, at the rate of 40 *per cent.* (MOREHEAD); and in *Madras* Presidency, at the rate of 50·97 (ANNESLEY), 19·35 (PARKES), 17·9 (INNES, at Secunderabad); MACNAMARA, in Madras, 50·9 *per cent.*; French surgeons in Algeria, 12·7 *per cent.*; EYRE, of the Madras Fusileers, 22·8; WARRING, in various localities not stated, 23·5 *per cent.*; STOVELL, in European General Hospital, Bombay, 19·3 *per* *cent.;* Leith, in Bombay, 8·5 and 15·2 *per cent.;* and Marshall, in Ceylon, 28·8 *per cent.*

Too much attention and importance seem to have been put upon abscess of the liver *per se,* irrespective of other obviously morbid conditions of that organ—*e. g.*, impaired function, congestion, enlargement. To regard secondary hepatic abscess as due to absorption of pus or other morbific matter from ulcerating mucous membrane, or to a true phlebitis, is to take too narrow a view of the relation of liver disease to dysentery; for, if we are to judge by the condition of the bile alone, the liver is diseased (in function, at least) in every case of dysentery (Parkes). The contrast of the results given in the above table, with the result of the cases seen in colder climates, is indeed remarkable. Baly's experience yielded him no abscesses of the liver. Finger, of Prague, dissected 231 cases of dysentery between 1846 and 1848, and found no abscess of the liver. Broussais records seventeen dissections of dysentery in the camp during 1805 and 1806, and no abscesses of the liver. Rokitansky has never found the liver visibly diseased in cases of dysentery; and in China, where dysentery, as a rule, is very fatal to Europeans, the rarity of hepatic abscess is said to have been remarkable (Dr. Wilson in *Records of Hospital Ship* "Minden").

On the whole, it will be seen that the association of dysentery with hepatic abscess is not equally frequent in all countries, nor in all epidemics. It seems to have been most frequent in the climate of the East Indies, and in the Bombay army especially (Morehead, Parkes). There are some epidemics in Europe in which the hepatic lesion has been observed; *e. g.*, in Dublin, 1818, it was observed in four out of thirty cases (Cheyne). It would therefore appear that the *poison* which causes dysentery has at some times and places the power of establishing hepatic complication so severe as to lead to abscess; at other times and places it seems to be less virulent.

The spleen and pancreas are sometimes also found diseased; and Mr. Twining notices the former as one of the most fatal complications of dysentery in the East Indies. These viscera are found either enlarged and softened, or enlarged and indurated, the spleen being sometimes the seat of abscess.

Of thoracic viscera, the lungs have sometimes exhibited a great tendency to secondary morbid processes in dysenteric cases. This was especially the case in the dysentery of the allied armies during the late Russian war, where otherwise pulmonic lesions were rare.

The pulmonic lesions associated with the dysenteric process were as follows:—(1.) More or less extensive lesion of the bronchial membrane, the finer ramifications of the tubes being filled with frothy mucus and pus-like exudation, and associated with extensive vesicular bronchitis: there were well-marked spots of lobular pneumonia. (2.) Exudations into the pulmonary parenchyma, chiefly in the form of isolated deposits of considerable density, disseminated through the substance of the lungs. These masses passed into a purulent condition, and microscopically they were composed of broken-up cells, granular matter, and pus-elements.

The last class of conditions which render cases of dysentery complex is the alliance of other disease-processes with dysentery. Such cases are generally of a very protracted duration; and the associated morbid lesions are not only complex from the number of morbid processes developed and the organs attacked, but they are complex from the variety of kind, degree, and extent of the co-existent affections. Many disease-processes may be observed to co-exist in one patient; and such multiplicity of disease-processes tends greatly to multiply the number of the anatomical local lesions, and thereby still more to complicate the case.

Dysentery "is found to complicate readily in all climates with the prevailing fevers." Within the tropics it is frequently associated with *remittent* and *intermittent fevers;* in the geographical region of *typhus fever* it is a most frequent complication, under various circumstances, and becomes capable of propagation from person to person; and lastly, it is also occasionally complicated with *scurvy.* When dysentery follows upon, or is associated with, intermittent fever, the spleen will frequently become enlarged, indicated in the outset by general anæmia, or splenic cachexia, with a low asthenic type of dysentery.

The scorbutic complication is developed in cases of *dysentery* when the supply of food has been deficient in fresh vegetables, or when it consists in whole or in the greater part of salted meat. Sir Gilbert Blane asserts that the complication has been known to arise among prisoners of war, living entirely on fresh (animal) diet.

"The most terrible instance of suffering from this cause," writes Sir Ranald Martin, "was that of the European portion of the force employed in Ava during the first Burmese war, where they were for six and a half months fed on salt rations, and where 48 *per cent.* perished within ten months, principally by dysentery with the scorbutic state." Such disasters have since been equalled, if not surpassed, by the sufferings of our troops in the camp before Sebastopol during the winters of 1854-55, under the influence of exposure, fatigue, and continued rations of salt meat and green coffee.

The following is the account which Dr. Clymer has given of the morbid anatomy of the lesions seen in the chronic camp dysentery of the American armies during the war of 1861 to 1863. The account seems to bring the cases under this part of the subject,—namely, "*Complex cases of Dysentery.*"

He arranges the anatomical character of the lesions under the four following groups:—

"(1.) Follicular lesions of the colon, presenting all the transition forms of simple enlargement of the solitary follicles, rupture of the same, and the formation of rounded or oval ulcers, extending nearly or quite to the muscular coat, looking as if they had been cut out with a punch, on a greyish or yellowish-grey base, and sometimes filled with mucus, at other times pus. The enlarged follicles are occasionally the seat of pigment-deposits; and in some cases an areola of pigment, deposited in and among the glands of Lieberkühn, surrounds the enlarged and solitary black follicles. This was found generally in patients who had died of some other disease, as intercurrent camp fever, &c. The colon is usually more or less thickened, even to the amount of a quarter of an inch. Its texture, when cut into, is frequently tough and lardaceous, and often softened. The colour of the mucous membrane of the colon is seldom natural, being ash or slate-coloured, or greenish-red, reddish-brown, or reddish-black; at times there are patches of congestion.

"(2.) The extension and agglomeration of the follicular ulcers, by burrowing in the submucous connective tissue, destroy large portions of the mucous membrane by vast erosions. The mucous layer, containing the glands of Lieberkühn, undermined by the extension of the ulcer, not unfrequently hangs in shreds like a fringe from its edge, the undermined portion being occasionally destroyed by ulceration, but more frequently by sloughing. In such cases the mucous membrane is generally of a slate, dark-red, brownish, or greenish-brown colour; the base of the ulcers is yellowish or yellowish-brown, often with brown or blackish sloughs adhereing to their surface or edges. This group represents a more advanced stage of the disease.

"(3.) In addition to the lesions of the first and second groups, the surface of the large intestine is more or less coated with a yellowish, or greenish-yellow, or brownish-yellow croupous, pseudo-plastic, caco-plastic, or false membrane, similar to the membrane formed in the air-passages in diphtheria, and which sometimes extends to the small intestines, and is generally found in those who have died during the supervention of acute dysenteric symptoms. Examination by the microscope of properly prepared sections shows it to be composed of innumerable round cells (lymph-cells, pus-cells), held together by an adhesive granular matrix, more or less resembling coagulated fibrine. The origin of this membrane may be traced to a rapid multiplication of epithelial cells and superficial connective tissue corpuscles of the diseased mucous membrane.

"(4.) Two forms of ulceration are observed in the colon: in the first, the process begins in the closed follicles; in the second, in the intestinal epithelium or the glandular layer. The closed follicles enlarge by multiplication of their cellular elements till they project as little tumors above the surface. The tumor, having enlarged to a certain extent, ruptures; its cellular elements escape, and a minute ulcer is formed. The cells or corpuscles of the connective tissue surrounding the enlarged follicle now multiply, and the ulcer spreads by the superficial cells floating off into the intestinal cavity, while a new base is continually formed by the multiplication of the subjacent cells. The second form of ulceration begins by an abrasion or denudation of epithelium at some point which does not correspond to the position of a solitary follicle. The follicles of Lieberkühn are next destroyed, and the ulcer spreads in the connective tissue by the process just described. Such ulcers are rarer than those of the first kind, and probably are always secondary to them."

Metastatic abscesses, resembling those in pyæmia after gunshot injuries, are sometimes found, generally in the liver, but also in the lungs and spleen. In seventeen cases Professor Alonzo Clark found the kidneys more or less diseased; their weight was somewhat increased, and the cortical portion abnormally light and granular. Microscopical examination showed the tubuli uriniferi filled with granules and detached epithelium (Woodward, *American Medical Times*, Feb., 1863).

There is still another light in which the pathology of this disease requires to be studied—namely, in the

Types and Forms of Dysentery.—These have been variously described as—

(1.) *The purely inflammatory, acute, hyper-acute,* or *sthenic* form. In this form, while the phenomena indicate acute and severe inflammatory action, there is no tendency to the great depression of the nervous, circulatory, and muscular functions, which gives a marked character to some of the other types of the disease, such as—

(2.) The asthenic forms.

In the *asthenic* forms, besides the depression of the functions just noticed, there is much greater tendency in these forms to spread by infection, or under an epidemic influence. These asthenic forms are sometimes described as *adynamic, typhoid, malignant, bilious, intermittent,* or *remittent,* according as certain phenomena prevail characteristic of these states.

Symptoms of Dysentery.—An ordinary attack generally commences with *diarrhœa;* but in twelve or twenty-four hours disagreeable feelings begin to attend the frequent loose discharges from the bowels. These are irregular pains, commonly called "gripes," along the course of the large intestine, and sometimes described as "shooting," or "cutting." Technically, such symptoms are known as *tormina.* They are momentarily relieved by discharges from the bowels. But after a short time a sense of heat ascends from the rectum, and pain extends to the epigastrium till the whole abdomen is painful. There is a frequently returning inclination to go to stool: the griping and straining continue without the patient being able to pass anything more than a little bloody mucus. These symptoms are generally aggravated during the night and early morning, and they leave behind them the exhausting sensation that there has always remained in the bowel something which has yet to be discharged. This feeling is technically called *tenesmus,* and ultimately becomes the most striking feature in the case. The acute pain in the abdomen, although it may extend to the iliac regions or flanks, generally concentrates itself at last about the rectum.

The discharges from the bowels are at first scanty, consisting of mucus and blood, or bloody slime, as it is sometimes called. As the disease progresses, the evacuations become more copious, tinged with bile, and carrying off shreds of the exudation thrown out on the mucous surface of the intestine. Hardened balls of fæces, called *scybalæ,* are also sometimes discharged: these, however, are seldom seen in tropical dysentery; and if much feculent matter pass, there is always considerable relief. When the disease is fully established, the discharges exhale an odour different from the smell of fæces, and which is almost peculiar to dysentery, and very offensive. It is important to observe the character of the discharges, and especially as to the relative amount of blood, mucus, and shreds of exuviæ. If the disease advances, besides the constitutional symptoms becoming aggravated, more blood and mucus appear in the discharges from the intestines, together with shreds or large sloughs of exudation, which are often described as pieces of mucous membrane. In very acute cases, going on rapidly to an unfavourable termination, a great change often takes place in the nature of the stools, which become suddenly copious, serous, of a reddish-brown colour with black spots, attended with a putrid offensive odour which pervades the whole house. In the acute dysentery of Lower Bengal the patient is not unfrequently carried off by copious discharges of blood (W. C. MACLEAN). The shreds, however, are not mucous membrane, but, like the dysmenorrhœal membrane which forms on the internal surface of the uterus, the dysenteric slough varies in consistence, thickness, and strength. It may be washed perfectly white in water, and its minute histology shows no character of a mucous membrane. The hardened balls of fæces are much more rarely seen than they have been described to be. When the skin is dry, and of a pungent heat, the tongue furred, and the thirst urgent, the urine scanty and high-coloured, and the pulse increasing in frequency—these are symptoms of increasing danger in dysentery. Throughout the disease there is febrile distress, the nights are passed without sleep, or when it is obtained it is in short periods, dreamy and disturbed; and when the patient awakes he is unrefreshed and his spirits low and desponding. In the majority of cases the disease takes a favourable turn between the sixth and tenth days; the symptoms are then mitigated, the pain ceases, the number of stools diminish, and the flow of urine is restored. On the contrary, if it terminates fatally in this stage, hiccough, vomiting, a small and rapid pulse, and pale sharp features, denote the approach of death. The intellect, however, is perfect, and the patient, often deploring the fate which he sees inevitably to await him, dies after a short agony. In the dysentery described by Dr. Clouston some of the patients had ordinary diarrhœa (diarrhœa of irritation?), from periods varying from two or three hours up to twenty-four hours, before blood appeared in the stools. In some cases there was great pain in the abdomen for twenty-four hours before the diarrhœa set in (evidence of irritation?). In other cases there was scarcely any pain at any period of the disease.

Dr. Clouston distinguished two classes of cases. "In the first the patient had two or three loose stools, or perhaps had no ordinary stools at all, but at once began to pass glairy mucus mixed with blood, in small quantities at a time, from the bowel. He had no pain, no fever, no want of appetite, and he refused to believe he was ill. This would continue for a day or two, and then the blood would increase in quantity, and the stools would become more frequent. Pain would begin to be felt in the region of the rectum, and the pulse would mount up by ten or twelve beats. For days the patient would be at stool every hour or two, and of course would become weaker. His tongue was then seen to be coated with a dirty yellowish-white fur; but the appetite for such forms of nourishment as milk, strong beef-tea, calves' foot jelly made with wine, was still good. Solid food was not relished. The stools would then be seen to be coated with a semi-fibrinous semi-purulent-looking membrane. The tongue would then become clean, and glazed, and beefsteaky; the evacuations became feculent, mixed with pus, the latter element becoming gradually less as the patient advanced in his slow convalescence.

"In the second class of cases the patient had from the first great pain in the abdomen, of a griping kind, a hot skin, and a pulse over 100; the dejections were copious, and frequent, and watery, while they were largely mixed with blood. In many cases there was sickness; in all, loss of appetite. After some days the tongue and mouth would become dry, and parched, and black; the features pinched; the pulse small and quick; and death soon ensued. In some cases the stools would after a time become membranous and shreddy, and then purulent, till the patient was more gradually weakened and exhausted. One such case lived six weeks, another two months. In one only of this class of cases (out of seventeen) did the patient recover.

"All the cases had the following features in common:—Bloody stools at first, tending to become purulent; intense fetor of the evacuations during the whole of the disease; no scybala, and great thirst" (*Med. Times and Gazette*, June 10, 1865). These were cases of dysentery caused by the poison of animal effluvia from decomposing human excreta (undiluted) acting on constitutions in which vegetable diet seems to have been deficient (scorbutic), and in whom the nervous power was below *par*.

If the disease proves fatal in the chronic form, the patient generally becomes rapidly altered and prostrated by his sufferings, is strikingly emaciated, and often earnestly prays to be relieved from a life disgusting to himself and entirely despaired of by others. Death begins at the heart.

The patient, on the other hand, may in a few rare instances recover; the local symptoms gradually yield, till his health and strength are ultimately restored in a moderate degree. Convalescence is slow, rarely complete; and there is perhaps no disease which makes so persistent and pernicious an impression on the human constitution as dysentery.

Causes and Modes of Propagation.—It may be stated, as a general proposition, that there is no country where paludal fever exists in which dysentery is not an endemic and prevailing disease. In the East and West Indies, in China, the Ionian Islands, Gibraltar, Malta, the Canadas, Holland, the coasts of Africa, as well as in many different parts of France, of the Peninsula, of the continent of America, and of the eastern parts of Great Britain, the prevalence of intermittent or remittent fevers and of dysentery is notorious.

This connection is so intimate that a given number of persons being exposed to the action of paludal miasmata—as, for example, a boat's crew sent ashore in a tropical climate, the probabilities are that of the men returning on board part will be seized with dysentery and part with intermittent fever.

Paludal fever and dysentery, moreover, are not only conjoined in locality, but they often co-exist, precede, or follow each other in the same individual, so that the fever frequently ends in dysentery, and the dysentery in remittent fever. This proof of the common nature of these diseases is corroborated by every writer of any celebrity, and more especially by those who have detailed the diseases of our armies. But dysentery also prevails where there is no other evidence of the presence of malaria. Nevertheless, the evidence in favour of malaria being the common, though probably not the sole cause of dysentery, appears to be much the stronger. It seems also determined that dysentery prevails generally in the inverse ratio of the intensity of paludal fever. In Jamaica, for example, when the white troops suffered in the large proportion of 91 *per cent.* annually from paludal fevers, the cases of dysentery were to those of fever as one to nine; while in the Madras Presidency, when the troops suffered from fever in the much less ratio of only 30·25 *per cent.* annually, the cases of dysentery were to those of fever as forty-seven of the former to thirty of the latter. It appears that dysentery is less common in the hotter than in the colder months, or arises under circumstances less favourable to vegetable decomposition. Thus in India and China it is from the middle of November to the latter end of February, or when remittent fever changes into intermittent, that dysentery greatly prevails. It seems to be recognized, by those most competent to judge, that there is a directly exciting action of malaria quite apart from that indirect action of undermining the general health, the importance of which as a predisposing cause cannot be over-estimated (Grant, W. C. Maclean).

Our knowledge of the predisposing causes is derived from what principally occurs in the military and naval service; and from the sufferings of the troops we learn that exposure to the night air, to wet, or to fatigue, together with the intemperance and often improper diet incident to the life of a soldier, especially on active service in the field, have at all times been found to be conditions powerfully predisposing to dysentery.

The effects of *salt* diet in the production of dysentery being less known than the other predisposing causes, it may be as well to state that, by an experience of twenty years in the West Indies, it has been determined that in the Windward and Leeward Command, when the rations issued to the troops consisted of salt provisions five days in the week, the mortality from diseases of the stomach and bowels among the officers was as two to four *per cent.*, while that among the soldiers was as 20·7, or a tenfold ratio. On the contrary, in Jamaica, when salt provisions were issued to the troops only two days in the week, the mortality from the same diseases approximated so nearly between these two ranks as to be almost an equality. And corresponding facts to these have been observed in Gibraltar, on the coast of Africa, and at St. Helena. The Sierra Leone Commissioners on the western coast of Africa, who investigated this subject on the spot, were of opinion that the large proportion of salt rations mainly contributed to the sickness and mortality from diseases of the stomach and bowels in the form of dysentery and diarrhœa; and the following statement, given by the late Sir Alexander Tulloch in his Statistical Reports (page 11) on the sanitary condition of the troops in the West Command, shows the marked reduction which took place in the deaths from this class of diseases subsequent to the introduction of fresh meat diet; the mortality being reduced to a *tenth* part of its former ratio:—

In the navy the same effects of ill-regulated diet have been observed, and the good effects of a change. "In 1797," says Dr. Wilson, "the victualling (of the navy) was changed, greatly improved, and consequently

Previous to Alterations in Rations.						Subsequent to Alterations in Rations.					
Year.	Mean Strength.	Dysentery and Diarrhœa chiefly.		Ratio per 1,000 of Mean Strength.		Year.	Mean Strength.	Dysentery and Diarrhœa chiefly		Ratio per 1,000 of Mean Strength.	
		Admitted.	Died	Admitted.	Died.			Admitted.	Died.	Admitted.	Died
1825	571	235	32	411	56	1828	232	139	1	600	$5\frac{1}{10}$
1826	471	256	26	543	56	1829	114	50	—	439	
1827	345	209	13	606	38	1830 to 1836	42	22	1	524	
Total,..	1387	700	71	Average. 505	Aver. 51	Total,..	388	211	2	Average. 543	Aver. $5\frac{1}{10}$

immediate to the change the health of the seamen improved strikingly. *Scurvy, typhoid fever, dysentery*, and *ulcer*, which up to the period of the change had produced great havoc, became comparatively rare in occurrence and light in impression," and, it may now be added, are hardly known except by name.*

An insufficient diet was the main predisposing cause of the dysentery which prevailed in London at the Penitentiary, Millbank, shortly after its completion. This prison is built on a marsh below the level of the Thames at high-water, the river being banked out by a narrow causeway. As long as the prisoners were allowed a full and ample diet they appear to have resisted the action of the paludal poison and to have enjoyed good health. No sooner, however, was the quantity and quality of their dietary lowered than dysentery of a very fatal character broke out, and made it necessary to clear that establishment for a time of all its inmates. In the dysentery so well described by Dr. Clouston (already frequently referred to), the predisposing cause seems traceable to a diet deficient in fresh vegetable food, in constitutions of feeble nervous power, and aged, weak, and paralyzed persons, exposed to the poison of animal effluvia, in the form of decomposing and undiluted human excreta, as an exciting cause of the disease. The morbid appearances were characteristic of the scorbutic form of dysentery; and the premonitory symptoms were similar to those described by Dr. Barker (and referred to at p. 577, vol. i.) as peculiar to poisoning by sewage gases. Such cases were not contagious.

From the MS. notes of Dr. Alexander Grant regarding dysentery in India, to which I am kindly permitted to refer, I find that he, too, is of opinion that sewage miasm (animal effluvia poison?) may be a direct exciting cause of diarrhœa and of dysentery, constituting one form of the disease.

In cachectic states, whether from malaria, syphilis, scorbutus, the exhaustive effects of heat long continued have a markedly predisposing influence in favouring the production of dysentery; and in the treatment of the disease the existence of any such cachexia must be inquired into, and if possible counteracted.

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* As Dr. Christison justly observes, the salt meat of military and naval rations is not the same as the salt meat of civil life. The former is highly salted, in order to keep for two or more years in every climate. Its nutritive value is thus greatly overrated, and its nutritive constituents are still further diminished by the process of washing out in water before it can be eaten. Thus, besides the irritant effects of the salt diet in producing dysentery, another element exists as a cause of disease—namely, the insufficient nutrition which the salt ration diet is able to impart.

There are few facts to enable us to determine the proportions in which the different ages suffer from dysentery, but the returns of the troops from the Mauritius show that the mortality from this disease falls principally on soldiers advanced in life (TULLOCH).

FORCES IN THE MAURITIUS.	AGE.			
	18 to 24.	25 to 33.	33 to 40.	40 to 50.
Aggregate strength of seven years,	3,892	5,361	1,215	300
Died of Dysentery,	26	63	24	8
Ratio per 1,000 of mean strength,	6·7	11·8	19·7	36·6

Besides unwholesome solid food, water of an impure kind and from an impure source favours the development of dysentery. Drained from swamps, and used for drinking and cooking purposes, as it was on the Chinese coasts, it exerted a marked injurious influence both in exciting and in maintaining the disease. In connection with impure water, the reader is requested to refer to what has been said under the head of "Parasitic Diseases;" and especially under "*Distoma Hæmatobium.*"

Many other predisposing causes favour the development or propagation of the disease, especially amongst soldiers in active service—namely, long marches in hot weather, bivouacking at night in the open air (often extremely cold both absolutely and relatively to the day), want of sufficient clothes and bedding may be mentioned as the chief.

The causes of the chronic camp dysentery, as it prevailed in the United States armies during the war of 1861 to 1863, are stated by Dr. Clymer to be the long-continued co-operative action of the following agencies:—

Scorbutic taint from defective diet; paludal toxæmia; filth and overcrowding of camps and barracks; excessive and prolonged heat, and physical fatigue during the prolonged and active campaigns.

It does not seem to be so clearly understood as it ought to be, that dysentery is contagious, or rather that it is capable of being propagated from person to person. Being a frequent complication or concomitant of contagious fevers, it has been believed to inherit similar contagious properties. In the severe form of dysentery, for which the old Infantry Barracks of Secunderabad, in the Deccan, have long been notorious, it has been observed that men labouring under other diseases, who happened to be exposed to the putrid effluvia of the excretions of dysenteric patients, were often severely affected by the disease (W. C. MACLEAN). There is, therefore, good reason to believe that the exuviæ of dysenteric patients, as passed by stool, may, like those of typhoid fever, propagate the disease; and the observations of Budd and Goodeve give support to this view. Niemeyer also entertains this belief.

Prognosis.—The prognosis in dysentery depends much on the country in which the disease occurs, and on the combination of circumstances predisposing to the disease, not less than on the form or type which the disease may assume. In hot climates it is calculated that the deaths vary from one in nine to one in twenty; and on actual service the chances of recovery are much diminished. In all returns, however, the total deaths recorded give a faint idea and inaccurate representation of the real mortality resulting from dysentery. If it were possible to trace out the men who were invalided from the army and navy services from the effects of this disease, it would be found that the mortality is very much greater than is represented by tabular returns. It is a malady which, once fairly engrafted on the system, never leaves it till life itself becomes extinct (BRYSON, and others). It is sometimes also insidious in its mode of attack and progress; and there is such a desire, on the part of soldiers especially, to avoid the restraints of hospitals, that the disease is sometimes beyond the power of medicine before coming under treatment especially in tropical commands (TULLOCH). In the cases described by Dr. Clouston as those of the second class (see page 947 *ante*), the chances of recovery are hopeless from the first, and all complications with scorbutus are very unfavourable. There may be diseases of a more rapidly fatal character, but there are few which entail so great an amount of suffering. When once the disease has passed into the chronic form, it slowly but not the less surely continues, by a most loathsome process, to exhaust the vital energies, until death relieves the patient of an existence rendered almost intolerable by pain, debility, and the offensive nature of the discharges (BRYSON).

Diagnosis.—It is difficult, perhaps impossible, in the first stage, to distinguish dysentery from diarrhœa; but the blood, the number of the stools, and small quantity of fæcal matter passed will, in times when dysentery is prevalent, indicate the true nature of the disease.

Treatment.—After what has been written regarding the nature and the causes of dysentery, it is the obvious duty of the physician to direct his attention, in the first instance, to the *prevention* of the disease. He must inquire especially as to the conditions of the diet, that it be sufficient as to its animal and vegetable elements, and of good quality. Next, he ought to insure the means of detecting the disease early—for time is of the greatest importance in its cure and prognosis—and especially by removing the patient, if possible, from the sphere of action of any of those predisposing or exciting causes as have been noticed; and also to see that his surroundings are free of all those circumstances which co-operate in aggravating the disease, the chief of which are over-crowding, bad ventilation, bad food, exposure, intemperance. With regard to medicinal agents:—"He who would treat this disease with success," writes Sir Ranald Martin, "while he shuns exclusive means, must assign to each remedy its proper value. Blood-letting, sudorifics, and purgatives constitute the most universal remedies, and in simple uncomplicated dysenteries they will prove all-sufficient. But when the abdomen is tumid, and there is pain in the liver, or in any other region, while the nature of the discharges indicates advancing inflammation, *calomel*, conjoined with sudorifics, and repeated to meet the occasion, will powerfully aid the curative effect through its influence on the depurative functions—on the circulation, by unloading, jointly with purgatives, the gorged vessels of the abdominal organs—on the blood and on secretions generally—and on the very sudorific function which we wish to excite. While *calomel* is a most powerful agent when used judiciously as an aid to blood-letting, pushing it to the extent of ptyalism is by no means to be recommended; nor should mercury in any shape be used in *adynamic* forms of the disease, in *scorbutic dysentery*, nor in poisoning by *animal effluvia*, in the *splenic cachexia*, nor in states of *anœmia*, for in all these conditions of the system its actions are most injurious."

Ipecacuanha (*radix ante dysenterica*) was, and still is, much in vogue as a remedy in the treatment of dysentery; but although highly useful in some conditions it is not to be regarded as a specific in all forms of the disease. It is more effectual in the acute than in the chronic forms. Piso, in 1658, and Helvetius gave it alone in large doses, which were followed by speedy cures—a method again revived and recommended by Mr. Docker and my colleague, Professor Maclean. A marked diminution in the rate of mortality from the disease has attended this revival of the treatment by *ipecacuanha*, which Mr. Twining gave in eight-grain doses with *extract of gentian*, twice or thrice daily.

The large dose method is to give as early in the disease as possible grs. xxv. to grs. xxx. of *ipecacuanha* in as small a quantity of fluid as possible. A preliminary dose of opium is often of service in enabling the stomach to retain the *ipecacuanha*. If it can be swallowed in the form of a *bolus*, by wrapping it in soluble tissue paper, so much the better. The patient should then remain perfectly still in bed, and abstain from fluid for at least three hours. If thirst is urgent it may be appeased by sucking small bits of ice, or taking not more than a tea-spoonful of iced water. In from eight to ten hours, from 10 to 15 grains may be again administered, with the same precautions as before. The beneficial results are manifested by the tormina and tenesmus subsiding, the motions becoming feculent, blood and slime disappearing; and often, after profuse perspiration, the patient falls into a tranquil sleep and awakes refreshed. The *ipecacuanha* may require to be continued in diminished doses for several days, with sufficient intervals between each dose to admit of food being also taken; and for several nights after the stools appear normal, grs. x. to xii. of *ipecacuanha* should still be given at bed-time. Astringents in any shape during the acute stage are not only useless but dangerous (MACLEAN, DOCKER, WARING).

Blood-letting has now been totally superseded and rendered unnecessary by the use of *ipecacuanha*.

Ergot has been used in an enema to the extent of 12 or 15 grains in some bland fluid; or in 6-grain doses by the stomach, in cases of epidemic dysentery, with the beneficial result of reducing the quantity of blood in the stools (Dr. Gros in *Practitioner*, Nov., 1868).

Opium is more valuable in the chronic than in the acute form of dysentery. It may be given in large doses combined with *acetate of lead* (gr. iii. to gr. iv.) in each dose, *nitrate of silver* or *sulphate of copper;* and in enemas it tends to relieve *tromina* and *tenesmus*. The following is a good formula for such an enema:

℞ *Plumb. acet.*, gr. x; *Acid. acet. dil.*, ♏x; *Morphiæ acet.*, gr. ¼. *aq. tepid.*, fl. oz. iv. (Waring).

In mild cases, but where the pains were excruciating and attended with tenesmus, the warm bath generally gives instantaneous relief in cases following chills, if adopted sufficiently early.

Dr. Maclean directs that it be brought to the bedside, to be kept at a high temperature (not under 99° or 100° Fahr.), and the patient to remain in it till he feels faint. He is then to be carefully and quickly dried, put to bed, and have gr. xv. to xx. of *ipecacuanha.*

Leeches to the number of six to twelve applied round the verge of the anus often affords sensible relief to the tormina and tenesmus, by unloading the portal and hæmorrhoidal veins (Waring).

In subacute and chronic dysentery, "no remedy," writes Dr. Waring, "has proved more useful than *nitrate of silver,* in doses of half a grain to one and a half grains daily, reduced to fine powder, and conjoined with Dover's powder in the form of a pill. It has also been extensively used in the form of enema, as follows:—First throw up into the transverse colon, by means of a flexible stomach-pump tube, introduced to the extent of six or seven inches, *very cautiously and gently,* enemata of warm water, or milk and water, to the extent of three, four, or six pints, so as to bring away any fæcal accumulations. Then follow up this practice by the injection of two and a half to three pints of distilled water, holding in solution gr. xv. of *nitrate of silver*" (Hare, Waring).

In chronic cases a combination of *sulphate of copper* and of *opium* is often highly serviceable (J. Brown, Raleigh, Waring), in the following formula:—

℞ *Cupri sulph.*, gr. ¼ to ½; *Pulv. opii.*, gr. ½. *Make a pill or powder, of which three are to be taken daily.*

Dr. Waring has seen much benefit from this formula; but recommends the substitution of five grains of *Dover's powder* for the *opium.*

Solution of the pernitrate of iron is highly commended by Professor Maclean. It is astringent and tonic in doses of x.♏ to xv.♏ diluted in water. It may also be used as an injection.

Dover's powder.—Pulv. ipecacuanha composita is of great service in full doses of gr. x. to gr. xv. at bed-time, followed up by a five-grain pill of the same, taken every four or six hours for two or three days, or till relief is obtained. In smaller doses, frequently repeated, it may be combined advantageously with *nitrate of silver* (Waring).

Nux vomica, combined with *opium* and *iron,* may be of use (Graves). In *malarial* dysentery, full doses of *quinine* (not less than twenty grains) ought to be given in acute cases before giving *ipecacuanha,* and it should be continued till there is evidence of *cinchonism.* The two drugs should then be given in alternate doses till the characteristic good results of each are produced (Maclean).

The bark of the root of *calotropis gigantea* (or *mudar*) has been recently used in India, and is said to be an excellent substitute for *ipecacuanha.* It is used in doses of a scruple to a drachm, is a reliable cholagogue, and sedative to the muscular fibres of the intestine, rapidly allaying pain, tenesmus, and irritation (Durant, *Ind. Med. Jour.*, May, 1866).

In general the dysenteric patient is not admitted into the hospitals of our large towns until the disease has passed into the second stage, and there is no class of disease which then offers so few chances of recovery. On the continent the neutral salts and mild purgative medicines are highly spoken of; but it is difficult to understand how these substances, having no specific power over the disease, can be beneficial in a highly ulcerated state of the intestine. Of all purgatives, however, two ounces of an infusion of *ipecacuanha* (in the proportion of one drachm to a pound of boiling water), combined with five to ten drops of the *tincture of opium*, and given every six or eight hours, appears to be the best; but the disease, though mitigated, is seldom cured by these means. Vegetable tonics, containing *tannin*, as *kino*, *hœmatoxylon*, or *catechu*, however prepared or combined, give temporary relief, but are ultimately inefficient.

Dr. Bryson writes that he has seen all the astringents, both mineral and vegetable, mercury both internally and externally, with many other medicines, tried without any benefit; but there were some means which were useful in relieving the more urgent and distressing symptoms. Amongst these he mentions *a well-regulated farinaceous diet*, *opium*, *suppositories*, *anodynes*, *astringent injections*, in combination with *opium*, *cascarilla*, *resinous astringents*, and the application of *leeches to the rectum* when tenesmus was distressing, or over the course of the colon when there was deep-seated pain. An injection of warm starch (two ounces) with *laudanum* in it, will often give great relief. As much nourishment should be given in a liquid form as the patient can be got to take. Milk boiled with flour should be taken as often as possible, night and day. It should be taken cold, even with ice, and in small quantities at a time; and small pieces of ice not only allays sickness and nausea, but seems to soothe the irritability of the intestines. Strong beef-tea or Liebeg's extract of flesh are most useful.

The value of a change of climate, as a curative measure, is forcibly illustrated by Dr. Bryson. He says that the crews of vessels improved in health almost immediately after quitting the station where dysentery prevailed.

Where sewage is applied to the soil by surface irrigation, it ought to be diluted largely with water, and deodorized by *carbolic acid.*

My friend and colleague, Professor Maclean, writes me in the following words on the treatment of dysentery, the result of his extensive experience in India and China:—"The first thing to bear in mind in the treatment of tropical dysentery is, that the appearance of strength in the patient, given by the acuteness of the symptoms, is delusive. Under the use of strong antiphlogistic treatment the strength of the patient is apt to fail suddenly; and this is often the case even when the treatment has been more conservative in its character. It was once the custom in India to deplete freely in this disease, either by a general bleeding or by the repeated application of leeches; but the most judicious and successful practitioners in India rarely bleed now, *even in the most sthenic forms of the disease,* and confine the use of leeches within the narrowest limits.

"*Mercury.*—It is certain, too, that mercury is yearly less and less used in India than it was; and there is much evidence to show that a corresponding reduction in the mortality of the disease has been the result. The objections to its use are numerous,—it entails great suffering on the patient, if pushed to pytalism, aggravating his miseries, and too often permanently injuring his constitution; it has no specific action on the disease, and its cholagogue effects can be attained by remedies which are not open to such objections as can be brought against mercury. (With regard to its cholagogue effects, grave doubts are thrown by the experiments of Dr. George Scott, referred to at the footnote of page 165 by the author). In sloughing dysentery it is followed by the worst results; and the observations of clinical observers in India have shown that individuals under the influence of mercury are not only not exempt from attacks of the disease, but are peculiarly prone to be affected by it. This is the case in a very marked degree in Asiatics (MOREHEAD and MACLEAN).

"*Ipecacuanha.*—This remedy has long been used in South America in the cure of dysentery,—whence, indeed, it came. It was much used in India until the mercurial notions of James Johnston prevailed. It was again used by Dr. Twining, of Bengal, by whom it was strongly recommended, and also by Dr. Mortimer, of Madras. Twining combined it with blue pill and gentian, and used it chiefly in small and oft-repeated doses. In South America the practice has always been to administer an infusion of the bruised root,—ij, being infused over night in ℥iv. of water, and given early in the morning. In Peru it is given in doses of ʒss. to ʒi. of the powdered root in a little syrup and water. This practice of giving ipecacuanha in large doses has lately been revived in India with encouraging success, and, I believe, the greatest number of cures. It appears to act on the portal capillaries, and on those of the mucous membrane of the bowels, and to determine powerfully to the skin. *It is usually given in doses of half a drachm or a drachm, either in pills or bolus, or suspended in mucilage, according to the fancy of the patient. It is advisable to give an opiate half an hour before, and to withhold all drink for some hours.* Unless there be hepatic complication, it seldom happens that much vomiting is caused by these large doses; on the contrary, they are often tolerated when smaller doses are rejected. The dose should be repeated in about six hours. A sufficient interval should be allowed to intervene between the doses of ipecacuanha to admit of the patient being sustained by nourishment adapted to the stage of the disease. I need hardly add a caution not to press the remedy too far.

"Dr. Cornish, of the Madras army, has shown from official documents that the mortality from acute dysentery in Southern India under mercurial treatment was 7·1 *per cent.* Since the general introduction of ipecacuanha in full doses it has fallen to 1·3. Dr. Ewart, of Bengal, has shown that equally good results have followed the same system in that Presidency. During the forty-two years from 1812 to 1853–54 the mortality among European troops in the Bengal Presidency amounted to 88·2 in the thousand. But during 1860, when large doses of ipecacuanha were administered, the mortality was only 28·87 in the thousand.

"Great credit is due to Mr. Docker, of the 7th Royal Fusiliers, for recalling practitioners to the use of this invaluable remedy (*Vide* Grant's *Annals of Military and Naval Surgery*, vol. i.)

"In no disease is early treatment more necessary than in dysentery, and I believe that, if conducted as above, except in the malignant and 'putrid' forms, we may look for good results in a large proportion of cases. Turpentine epithems and fomentations should be diligently used, and the patient's strength should be supported by nourishment of a bland kind, suited, in degrees of nutritive value, to the stage of the disease.

"In the scorbutic form we have a valuable remedy in the *Bael fruit*, when procurable. This fruit contains a large quantity of *tannin*, with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal; and in the scorbutic form I have seen it successful when all other measures have failed. (See note *On Bael Fruit*, by Dr. Grant, in *Indian Annals*)."

HÆMORRHAGE FROM THE INTESTINES.

LATIN EQ., *Hæmorrhagia;* FRENCH EQ., *Hémorrhagie;* GERMAN EQ., *Blutung*—Syn., *Hæmorrhagie;* ITALIAN EQ., *Emorragia.*

Definition.—*Loss of blood from some portion of the mucous membrane of the alimentary canal below the stomach. It may have its seat in the small intestines, or in the large, or in both, but probably never flows from the whole length of the canal.*

Pathology and Morbid Anatomy.—If the intestinal hæmorrhage be considerable, the mucous membrane is generally blanched and colourless; but when more moderate, the surface of effusion may be determined by the mucous membrane being congested, and perhaps infiltrated at the affected portion; but as the hæmorrhages from the upper part of the bowel are generally capillary, the exact source or point of lesion can rarely be discovered after death. Sometimes there is ecchymosis, a considerable extent of the mucous membrane being suffused with blood. The mesenteric vessels are also found gorged. When the hæmorrhage takes place from ulcers, blood-coagula generally adhere to the edges of the ulcers which have bled, and the surface of the ulcer seems suffused with blood. The blood may be fluid, but generally it is loosely coagulated, of a chocolate brown colour, or of a dark tarry colour and consistence.

Varicosities of the rectum, sometimes termed *blind piles*, are also sources of intestinal hæmorrhage; and the term hæmorrhoids is applied to certain bleeding tumors which form round the edge of the anus, and also at the lower portions of the rectum as far down as, but still above, the internal sphincter. The latter are called *internal* piles, and the former *external;* but the general name of *hæmorrhoid* may be taken to signify "venous dilatations and hæmorrhages occurring in or from the rectum" (NIEMEYER).

The *internal tumors* or piles form between the inner sphincter and external edge of the rectum; they consist of a number of small soft hemispherical tumors of four to five lines in diameter, of a violet tint, and formed by venous dilatations in a diffuse thick bluish net, from which single *varices* at last appear, which may completely surround the anus. At first the varices are small, but with a broad base appearing and disappearing at intervals; and at length they may attain the size of a cherry, or even larger. They are apt to be pressed through the anus, every time the bowels are evacuated, and drawing the mucous membrane after them it eventually forms an elongated pedicle for them, so that they remain outside the anus. Once formed they never disappear. These tumors may rupture and much blood escape from them; when their walls are bluish, thin, and delicate, they also often inflame and become indurated with thick walls; or they ulcerate or form small abscesses, which, should they burst and cicatrize, are thus radically cured. But the inflammation thus excited may extend to the veins which form the venous plexus of the lower part of the rectum, and these vessels, especially those that are varicose, often become impervious and obliterated from the formation of *plugs* or *thorombs* in them, or of *phlebolitheo.* The mucous membrane of the rectum covering these tumors is, by the succession of inflammation and of sanguineous infiltration into these tumors, at length rendered so vascular as to bleed on the slightest friction.

The *external piles* are formed by the action which the sphincter exerts over the tumors thus formed at the edge of the anus, so that at each act of defæcation they become compressed at its orifice, are pressed outwards, and are thus progressively elongated. They at length hang pendulous external to the anus, and in time become pediculated, hard, and fibrous at their insertion.

When hæmorrhoids are complicated with *prolapsus ani,* the fibres of the *sphincter* and of the *elevator ani* muscles often become atrophied, wasted, and their action impaired.

In the early stages of this affection the blood or lymph effused may be absorbed, and the disease entirely subside. At a more advanced stage some become indurated and of little sensibility, while others again are soft, bleed profusely, are intensely painful, and sometimes rupture or ulerate (hæmorrhoidal ulcers). The mucous membrane, which is greatly vascular, is sometimes swollen, sometimes fissured or ulcerated, and these fissures sometimes penetrate so deep as to occasion rectal fistula.

Cause.—The same causes which produce *hæmatemesis* will tend to bring about *intestinal* hæmorrhages, and especially excessive congestion of the portal circulation from diseases of the liver, spleen, or heart, but especially *cirrhosis* of the liver. Intestinal hæmorrhages are also apt to occur from *lardaceous disease of blood-vessels* and ulcers, such as the *tuberculous, typhoid,* or *dysenteric.* Hæmorrhages also occur in the course of some specific diseases, such as *yellow fever* and *scorbutus;* also from the effects of *sulphureted hydrogen gas*—a cause of epidemic hæmorrhage from the bowels among the workmen in the mines of Anzin.

Children are very liable to slight hæmorrhage from the bowels while teething, and at other periods of infancy. It is most common, however, in the adult.

Hæmorrhoids are a frequent source of loss of blood. They are caused by everything that produces plethora of the abdominal vessels.

Persons, therefore, who indulge largely in hot tea or coffee, or who drink to excess of fermented liquors of any kind, or who habitually consume more food than is required for the support of the body, are liable to this affection. It is remarked also that hæmorrhoids often affect those who ride much on horseback, and likewise pregnant women.

In a very few instances hæmorrhoids have been met with in children of six and seven years of age, but twenty to fifty-five is the more common period of life when they occur. Both sexes appear equally subject to them.

The causes of escape of blood from the hæmorrhoidal veins are summed up by Niemeyer under the following heads:—

(1.) Collection of fæces in the rectum (constipation), tumors in the pelvis, or a gravid uterus (pregnancy).

(2.) Congestion from cirrhosis of the liver obstructing the portal vein, or over distension of it, especially from excessive use of fermented drinks and alcohol.

(3.) Lung diseases, where the capillaries are compressed and atrophied, and also heart affections, tend to impede the flow of blood through the hæmorrhoidal vessels.

Symptoms.—Hæmorrhage from the intestines may assume one of two forms, or that in which the blood poured out is pure, and that in which the blood, acted upon by the capillaries, is poured out black, pitchy, tarry-like, and grumous, when the disease is termed *melœna.*

The attack of intestinal hæmorrhage may be sudden or preceded by a series of preliminary symptoms, as pain in the back and loins as low down as the sacrum, and even descending down to the thighs. The patient also may suffer from colic pains, from flatulence, loss of appetite, and other symptoms of indigestion, while the bowels also may be either constipated or open.

Hæmorrhage from the large intestine is not uncommon, and is far from being attended with those grave consequences attached to that from the small intestines, although the quantity discharged is often great. Hæmorrhages from the small intestines are always symptomatic of severe diseases. From the large intestine the hæmorrhage is often periodical, and a great relief to persons subject to headache. It also frequently accompanies the hæmorhoidal flux. There are instances, however, in which the quantity passed is so great that the patient falls into a state of complete anæmia. In consequence of the descent of the gut, a patient may pass about half a pint of blood every other day for many weeks together, till he not only becomes sallow and dropsical, but is unable to move from bed. From the generally innocent tendency of hæmorrhage from these parts, it is of course intended to exclude those cases in which it proceeds from *diseased heart,* from *dysentery,* from *scurvy,* or from *enteric fever,* as well also as from organic disease of the intestine itself.

Hæmorrhoidal tumors produce many unpleasant symptoms, the least of which are the sensation of a foreign body just within the anus, and pain on the patient passing a stool, which is generally hard, constipated, and tinged with blood. Sometimes the hæmorrhoids are so numerous as to fill up the rectum, and should they descend so as to be grasped by the sphincter the pain is often exquisite, and the patient obliged to return the part with his finger, otherwise it becomes strangulated. When the tumors have become large and tense, pain is constant; and the patient can neither walk, lie, sit, nor stand; and even a pultaceous stool causes much pain on passage, so that the patient refrains as long as possible from going to stool.

When inflammation attacks the hæmorrhoidal tumors the pain is often so severe as to extend to the perinæum and testicles in the male, and to the vagina, uterus, and bladder in the female. These pains are much augmented on every movement, even by lifting up the leg, turning in bed, by sneezing or by coughing. In the worst cases every attempt at defæcation is distressing, and dreaded by the patient. Even sleep is at last almost lost, and a grave dysuria often still further adds to the torment of the sufferer.

The quantity of blood lost is sometimes trifling, but in severe cases it often amounts to many ounces daily. In the former instance the patient suffers little except from the local irritation; but in the latter he loses flesh, becomes exceedingly nervous, and often sinks into a state of melancholy which renders life a burthen.

Diagnosis.—The only disease with which intestinal hæmorrhage can be confounded is the lesion of *hæmorrhoids,* and their diagnosis is rendered certain by an examination, except in cases where they are excessively high in the gut.

Prognosis.—Hæmorrhage from the small intestines, although not necessarily fatal, is always an unfavourable symptom. Hæmorrhage from the large intestines, if idiopathic, is of less moment; but if it be symptomatic, and results from *disease of the heart or spleen,* from *dysentery,* or from *organic disease of the intestine itself,* the prognosis is grave in proportion to the intractable nature of the primary affection.

Piles have seldom any dangerous tendency unless they cause *fistula* or *phlebitis.* When operated on, however, more especially internal piles (as by ligature), they have been known to produce accidents, such as *septicæmia* or *phlebitis,* which have terminated fatally.

Treatment.—*Ergot* seems a useful remedy. (See under *Hæmoptysis.*) Dr. Macgregor, of Glasgow, records a case of *persistent periodical hæmorrhage from the bowels* which yielded to *ergot* when all other remedies had failed (*Glas. Med. Journal,* June, 1867). In hæmorrhage from the bowels in enteric fever, the *tincture of the perchloride of iron* is often useful, and if associated with much arterial action, it may be combined with *digitalis,* as in the following formula:—

℞ *Tinct. ferri perchlorid,* ♏xxx.; *Tinct. digitalis,* ♏xv.; *Aq. Menth. pip.,* ℥iss., repeated every four hours (Waring).

In the event of failure, the *iron tincture* may be tried as an enema, combined with opium, in the following formula:—

℞ *Liq. ferri. perchloridi,* ♏xv.; *Morphiæ hydrochlor.,* gr. ½.; *Aq. tepid,* ℥iv.; *misce* (Dr. John Harley).

In cases where there is a tendency to syncope from intestinal hæmorrhage, *oil of turpentine* (♏x. to ♏xv.) every half hour or hour has proved an effectual remedy (DR. JOHN HARLEY).

The medical treatment of hæmorrhoids consists in the application of a few leeches to the margin of the anus, and in the administration of the *bitartrate of potash*, combined with *sulphur*, in the proportion of one part *sulphur* to two of *cream of tartar*, a teaspoonful being taken three or four times a day, so as to keep the bowels gently relaxed. *Sulphur* has been much insisted on in these cases.

Stringent rules ought to to be laid down as to diet, which should be light, and the patient limited to French and Rhenish wines. Ablution with cold water should be practised morning and evening; and some persons are sufficiently sthenic even to bear an injection of cold water, but much caution is necessary in the application of this remedy.

OBSTRUCTION OF THE INTESTINES.

LATIN EQ., *Obstructio;* FRENCH EQ., *Obstruction;* GERMAN EQ., *Verstopfung;* ITALIAN EQ., *Ostruzion.*

Definition.—*An obstacle or impediment which obstructs the passage of contents through the bowel.*

Pathology.—A typical case of intestinal obstruction is thus related by the late Dr. Brinton, whose monograph ought to be read by every student. He did more in his too short life to elucidate the pathology of such cases than any one who has written on this subject:—

"A person, perhaps hitherto healthy, experiences a sudden constipation, attended with disproportionate uneasiness, or flatulence, soon merging into pain and distension of the belly, with violent rolling movement of the intestines. The distension increasing, nausea and vomiting supervene; and, gradually becoming more frequent, end by rejecting not merely any casual alimentary contents of the stomach, or the greenish, bilious, alkaline fluid commonly thrown up when this organ is unoccupied by food, but a fluid of greater opacity, colour, and consistence, with a distinctly fæcal odour. A further aggravation of these symptoms now conducts the malady to its termination. This, if fatal, is usually preceded locally by signs of paralysis, inflammation, or even rupture of the distended bowel, and constitutionally by exhaustion or collapse replacing a febrile re-action. In other cases, the obstacle being removed by nature or art (if by the former, rarely before life is in extreme danger), the symptoms subside with comparative celerity. The pain, distension, and vomiting cease; the bowels are relieved by copious stools; and the patient (if not placed in further peril by any of those conditions incidental or consecutive to obstruction just hinted at) is rapidly restored to comparative health" (*Intestinal Obstruction*, p. 6).

The most remarkable and most characteristic symptom of intestinal obstruction is the fæcal vomiting, explained by a doctrine that remained almost unquestioned since the time of Galen, until the observations and experiments of Dr. Brinton exposed the error, and established the pathology of intestinal obstruction on a rational basis. When, about twenty years ago, he exposed the error of the doctrine, then and even now entertained, it was supposed that fæcal vomiting was effected by an *anti*-peristaltic movement of the intestinal canal; that, at a certain stage of obstruction, the natural peristaltic action of the bowel above the occluded point was reversed: so that instead of proceeding towards the anus, as heretofore, it took the contrary direction,—thus impelling the intestinal contents in a similarly retrograde course, so as to return them to the stomach, whence they were vomited.

Dr. Brinton showed by abundant proof, experimental and incidental, that the notion of *anti*-peristalsis was contradicted by direct observation; and a careful study of the phenomena of intestinal obstruction, as witnessed in the human subject, and as artificially produced in experiments on animals, led him to the following theory:—

"The movement proper to the healthy intestine is a circular constriction or peristalsis which, travelling slowly and intermittently down its muscular wall, propels its contents in a direction from the stomach to the anus. And when any part of the intestine has its cavity obliterated by an immovable mechanical obstacle, its contents, propelled by such a peristalsis, are stopped at the obstructed point. Here they gradually accumulate, so as first to fill and then to distend a variable length of the canal with a more or less liquid mass. But a peristalsis engaging the wall of a closed tube filled with liquid, and falling short of obliterating its calibre, sets up two currents in that liquid;—one at the surface or periphery of the tube, having the direction of the peristalsis itself, and one in its centre or axis, having precisely the reverse course. Those particles of the liquid which are in contact with the inner surface of the tube are propelled onwards by the muscular contraction of its wall. And this propulsion is necessarily accompanied by a backward current in those particles which occupy the axis or centre of the canal."

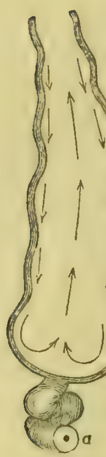

Fig. 56.

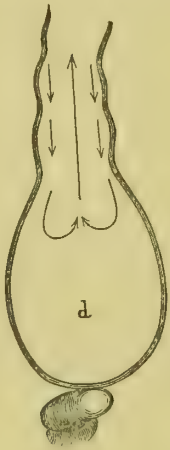

Fig. 57.*

Fæcal vomiting is thus shown to result from the reflux of the intestinal peristalsis—a backward current in the liquids occupying the centre of the tube.

The ordinary course of intestinal obstruction from Dr. Brinton's point of view divides itself into two stages. In the first stage the healthy actions of the bowel are continued; in the second stage they

Figs. 56 and 57 : Diagram to illustrate the peristalsis of an obstructed bowel (after Dr. Brinton).—Fig. 56. Stage of moderate distension, with forward and backward currents, as indicated by the arrows traversing the whole tube above the obstacle; (*a.*) Contracted segment of intestine below the obstacle. Fig. 57. Stage of extreme distension, in which (*d.*) the dilated and paralyzed segment above the obstacle is scarcely engaged by either of these currents.

are arrested or utterly and permanently annihilated by paralysis, enteritis, or peritonitis.

In the first stage, abnormal *distension* of the intestine can generally be felt through the yielding wall of the belly—as a condition of fæcal vomiting—from the very commencement, and continuing a persistent physical sign through all the stages of the obstruction. Experience led Dr. Brinton to assert "that the accumulation of intestinal contents immediately above the obstructed point may sometimes be detected as a slight fulness to palpation, and a much more definite dulness to percussion, where many of the other indications of obstruction are scarcely perceptible, or even absent." The movements of the obstructed intestine may thus be traced rising visibly against the walls of the belly covering the obstructed tube, "in coils that may be fancifully compared to those of a writhing serpent," until the paralysis and collapse usher in the fatal issue.

The character of the pain in obstruction is variable. It is sometimes sudden and violent, and often rises to great intensity in a very short time. It is distinct from the burning pain of peritonitis. It is usually intense in intus-susception, and in the impaction of gall-stones; somewhat less marked in the obstruction produced by twisting of the bowel, or by bands and adhesions; scanty in the obstruction of stricture; and almost absent in the obstruction caused by the impaction of fæces in the large intestine (BRINTON).

Exclusive of hernia, Dr. Brinton estimated, from an analysis of 12,000 necropsies, that obstructions of the intestine cause about 1 in every 280 deaths from all diseases indifferently; and the chief varieties of obstructions have to each other the following proportionate frequency: —Intus-susceptions or invaginations, 43 *per cent.;* obstructions by bands, adhesions, diverticula, or peritoneum, external to the bowel, 31½ *per cent.;* strictures (including a few tumors) involving the intestinal wall, 17½ *per cent.;* torsion of the bowel on its axis, 8 *per cent.*

The forms of intestinal obstruction are mainly as follow:—

(a.) Intus-susception, of which the varieties are,—*Ileo-cæcal,* 56 *per cent.; Iliac,* 28 *per cent.; Jejunal,* 6 *per cent.; Colic,* 12 *per cent.*

(b.) *Obstructions due to bands, adhesions, diverticula, gallstones, lesions,* such as rupture of *mesentery,* and other peritoneal lesions. The small intestine is the seat of the obstacle in 94·53 cases *per cent.*

(c.) *Obstruction due to strictures, tumors, or twistings of the bowel and mesentery* give about 87·36 *per cent.* of cases involving the large intestine.

Intus-susception is the accidental insertion or protrusion of an upper into a lower segment of intestine. It occurs more frequently in infancy and childhood than at any other period of life. Of twenty-five cases observed or collected by Rilliet, *seven* occurred in children of six months or under; *six* during the first year of life; *seven* between five and ten years of age; *five* between ten and under fifteen years of age. Dr. Brinton's experience goes to show that half the ileo-cæcal intus-susceptions are infants under seven years of age; many but a few months old; and he gives the average ages of the ileo-cæcal, jejunal, and colic respectively as 18·57, 34·6, and 31·4 years.

Invaginations of the small intestines are so frequently found after death, in comparatively young and well nourished subjects, that it is generally believed they are formed with great facility, and that they often occur during life, giving rise to temporary bowel derangement; but that they also soon become disentangled again by the normal peristaltic movements. Of 300 children examined by Louis at the Salpetrière Hospital, and who died there, the greater number had two, three, or more *volvuli* without any inflammation of the parts; and there were no circumstances in their history during life which led to the suspicion that these children suffered from intus-susception, (*Mem. de l'Acad.*, vol. iv.) Dr. Baillie, also, in his great work *On Morbid Anatomy*, says, "In opening bodies, particularly of infants, an intus-susception is not unfrequently found which had been attended by no mischief; the parts appear perfectly free from inflammation, and they would probably have been easily disentangled from each other by their natural peristaltic motion." Dr. Macintosh states that he scarcely ever opened a child without finding partial invagination of the small intestines (*Practice of Physic*, vol. i., p. 256). Dr. Hodgkin, in his valuable work *On the Morbid Anatomy of the Mucous Membranes*, and Rokitansky in his *Pathological Anatomy*, each make mention of the frequent occurrence of invaginations in the bodies of adults as well as of children, and they consider them to be produced in the majority of instances during the last moments of life—in the death struggle, or in the *rigor mortis* of the dead intestine. I have frequently observed such invaginations in *post-mortem* examinations, not associated with symptoms during life, and easily reduced by traction; but they have been generally in cases where the irritability of the bowel had been greatly increased during life by excessive diarrhœa, with or without ulcerations of the intestines. Such invaginations were invariably in the small intestines.

Cases of intus-susception *in the adult* are rare—so rare, indeed, that in the extensive experience of one of the largest civil hospitals in London (Guy's), Dr. Wilks records that "he has never seen but one case of intus-susception *in an adult*, and in this case the obstruction was never complete, and death did not occur for some weeks" (*Pathological Anatomy*, p. 292). In the *Transactions of the Pathological Society of London*, extending over the first fifteen years of its existence, there are only seven cases of intus-susception *in the adult* on record—no two of which occurred in the individual experience of any one man. The ages of these seven cases are respectively as follows—namely, eighteen, twenty-five, thirty-two, thirty-four, forty-one, and two at forty-four years of age. In one case the symptoms continued for three months, and at last ended in recovery after the passage of a portion of ileum (*containing a polypoid tumor*) by the rectum. In another case the symptoms continued during four months, and terminated fatally by exhaustion. In my own experience I have never made a *post-mortem* examination of a case of intus-susception *in an adult*, nor have I ever seen a case of intus-susception *in an adult* during life. In the Museum of the Army Medical Department at Netley there are preparations showing the lesions and morbid relations of the parts preserved, from at least eight cases occurring in soldiers at ages varying from twenty to forty-two years of age.

A case recorded by Dr. Todd, in the *Army Reports* for 1864, p. 532, is of great interest, inasmuch as the dissection of the parts, made by me at Netley, shows that the intus-susception was associated with a large polypus growing from the mucous surface of the small intestine. The history of the case further shows that it was preceded and accompanied by intense and severe diarrhœa; and in the course of examination of the parts sent to Netley, my then coadjutor, Dr. Davidson, discovered that the mucous membrane of the *caput cœcum* was infested by the minute parasite known as the *trichocephalus dispar.* This parasite is a very minute round worm, with its head-end of hair-like fineness, usually firmly fixed to the mucous membrane of the intestines, while the rest of the body is generally coiled upon itself and hidden amongst the mucous secretion of the gut. The natural history of this entozoön shows that it has oftentimes been associated with severe epidemics of diarrhœa. Indeed, its discovery more than 100 years ago (1760–61) was made during the prevalence of a severe epidemic of diarrhœa (*morbus mucosus*) amongst the soldiers of the French army, associated with the presence of this parasite in the *caput cœcum* of those who died of the disease.

Five cases of intus-susception are recorded in the *Transactions of the Pathological Society* which are associated with polypoid tumors of the intestine at or near the site of lesion. The latter case referred to in these *Transactions* terminated favourably after the passage of the invaginated portion of gut (containing the polypus), the symptoms having continued for three months.

The *volvulus,* or portion of the gut where the obstruction exists, consists—(1.) Of the external portion formed by that part of the bowel into which the other has slipped; (2.) Of the middle; and (3.) Of the internal part, composed of the reflection of the invaginated portions. As it is not always easy to follow the anatomical relations of the several layers of structures composing an intus-susception, it may be of use to give a diagramatic outline of the relation of the serous and mucous coats of the intestine in such lesions; because it is of practical importance to remember that although the parts are greatly displaced, yet the anatomical relations of the serous and mucous surfaces of the intestine are never altered. Textures of the same anatomical character are always in contact one with another, and the channel of the gut along its mucous surface is always open. That such is the case may be understood by taking the leg of a long stocking from which the toe-end has been cut off, so that the stocking may be converted into a continuous tube open at both ends. If one portion of the stocking be then drawn into the other, a correct imitation of the relation of surfaces in an *intus-susception* will be obtained.

In the diagram (fig. 58) the tube, *a b,* may be traced to be continuous, as indicated by the arrows. The dotted line is meant to correspond to the *serous* surface, and the thick dark line to represent the *mucous* surface of an intestine comprehending an *intus-susception.* From the outer to the *innermost* surface at the site of lesion, on cutting through one layer, *the first* of the enclosing gut, a MUCOUS SURFACE is reached, which has a *cul de sac* reflection at *c.* Thus, the MUCOUS surfaces of the including and the included portions of intestine are in constant apposition, rubbing against each other. On cutting through the *second* layer of the *intus-susception*, a SEROUS SURFACE is reached which has a *cul de sac* reflection at *d*. Thus, the SEROUS surfaces of the includ*ing* and the includ*ed* portions of intestine are in constant apposition, rubbing against each other. At *e* the mucous canal is always more or less open in cases

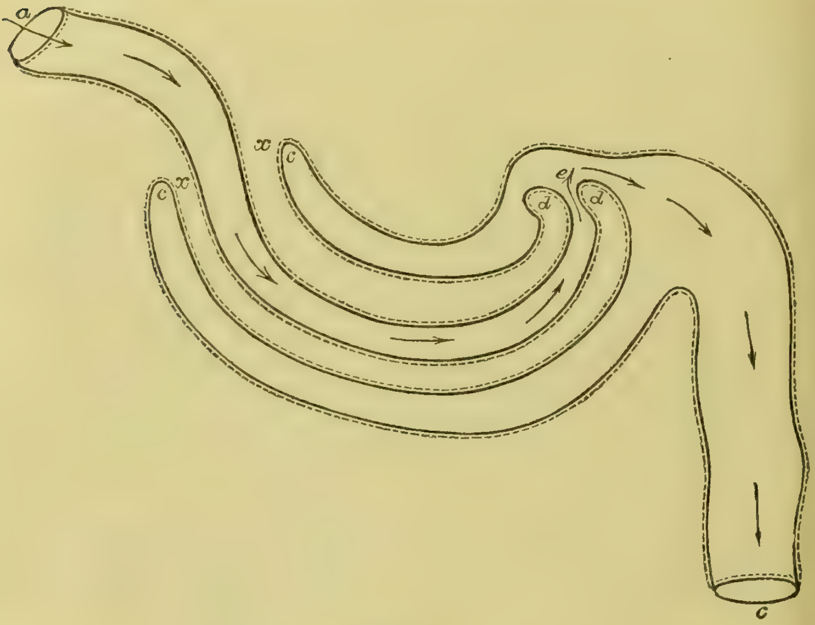

Fig. 58.

of simple *intus-susception;* but the orifice is invariably turned or curved to one side, and may be so firmly applied against the mucous surface of the including intestine (by the dragging of the mesentery which has been included) that the orifice may be closed, like a valve, by simple apposition and compression. In consequence of the lateral attachment of the mesentery to a line along the serous surface of the intestine, a portion of mesentery equivalent to the extent of the serous surfaces in apposition is also dragged into the containing gut, and exercises a most important influence upon the nature of the lesions. In consequence of the one-sided attachment of the mesentery, and the dragging of its parts, the included gut necessarily takes the form of a curve. It thus appears highly corrugated over its mucous surface, dragged to one side and curved upon itself, as it lies exposed on cutting up the outermost layer of intestine. The orifice of the contained or invaginated intestine is thus turned upwards, and is not to be found at what appears to be the lowermost part of the extreme end of the included portion of bowel. This great dragging of the mesentery necessarily also obstructs the flow of blood in the mesenteric vessels, and leads to the gradual effusion of blood between its layers. This effusion is seen after death in the form of compact indurated masses of a dark colour. Blood is also gradually effused from the mucous surface of the gut, which becomes gradually strangulated; and, combined with other symptoms, this persistent effusion of blood is almost pathognomonic of incarceration of a bowel by intus-susception.

In some cases the pressure of the inclosed bowel on the containing gut, and the dragging of the included mesentery, are so intense that the invaginated portion has actually effected an opening by ulceration through the inclosing bowel, and so projected into the cavity of the peritoneum before death took place. In such cases the fatal result was generally by peritonitis. Four specimens in the museum of the Army Medical Department illustrate this fact in the pathology of intus-susception. One has no history; another is quoted as an example of the bad effects of purgation; the third is from a woman aged twenty-four, who had been ill ten days, when an extensive opening occurred through the inclosing bowel, and peritonitis of a severe form speedily proved fatal. A fourth occurred in a soldier aged twenty-two. In this specimen a very large mass of gut is involved, and it is very significant of the injurious influence of purgation in such cases. Although this soldier is recorded to have had persistent diarrhœa, flatulence, bloody stools, and other symptoms of intus-susception, he was nevertheless alleged to be a malingerer. He was treated with purgatives, and lived long enough for the end of the included gut to wear a hole, by pressure and rubbing, through the substance of the containing bowel. No one can doubt but that such a lesion would be greatly aggravated by purgative remedies.

In all cases of intus-susception, whether occuring in children or in adults, the administration of purgative medicines tends to aggravate the lesion and the symptoms. Accordingly the rule of practice is absolute —namely, "to withhold all purgative medicines from the commencement in cases of intus-susception." The bowel being incarcerated, the stimulus of purgation proceeding from above downwards is quite unable to undo the incarceration of an intus-susception. A purgative, therefore, acts injuriously as a stimulus which cannot be obeyed; and the obvious tendency of a purgative is to increase the peristaltic action of the bowels, and therefore to increase still more the invagination. That such is the case will be readily understood by a physiological consideration of the phenomena of intus-susception; for although it is not always easy to account for the first beginning of an invagination, yet physiology enables us to understand how, an invagination once begun, the lesion tends to increase—(1.) From the peristaltic action of the bowel, greatly stimulated and increased by irritation of every kind, so long as tonic irritability continues; (2.) From the spasmodic action of the part of the gut *above* the invagination preventing spontaneous return; (3.) From the invagination being thus completed, it continues permanent, tenesmus occurs, and thus the violent and repeated contractions of the abdominal muscles tend still more to maintain and increase the lesion. The constant motion and pressure of parts one upon another in some cases is so great that the end of the invagination has been known to penetrate through the walls of the inclosing bowel, so as to appear in the cavity of the peritoneum.

The injurious tendency of purgative medicines will be also still more apparent if a lesson is taken from the teachings of morbid anatomy. *Post-mortem* examination, combined with a study of the phenomena of intus-susception during life, shows that the increase of the lesion takes place mainly at the expense of the *external containing* portion of the bowel; and therefore, also, it can readily be understood how some fixed point in the bowel is the first starting-point of an invagination. Most frequently (56 *per cent.*, BRINTON) it is the ileum and cæcum which pass into the colon, then the colon passes into itself, so that the *appendix vermiformis cæci* becomes included. Two orifices then exist at the extreme end of the invagination:—one is that of the lesser bowel, the ileo-cæcal valve, the other is the entrance into the *appendix cæci.* Two preparations in the Museum of the Army Medical Department show this arrangement of the parts composing the lesion in cases of invagination. In 32 *per cent.* (BRINTON) the small intestine forms all the layers. In 12 *per cent.* (BRINTON) the colon is exclusively involved; and the rectum scarcely ever forms more than the outer layer.

Causes.—In all the dissections of invagination whose history I have examined, they have either been associated with the diarrhœa of irritation (as from worms, undigested masses of food); or with cerebral lesions (as in the cases of children in whom invaginations are very common); or with ulcers of the intestines, or polypoid growths.

In giving a summary of cases, Dr. Peacock finds that while in some cases no cause could be assigned for the affection, in others the disease appeared to have been excited by accidents, taking injudiciously large meals or improper food, by the irritation of drastic purgatives, or the presence of worms. Of the cases which he analyzed, "in one instance the disease followed a kick, in another the carrying of a heavy weight, and in a third the taking of a large meal; in one an active purgative had been taken a few days previously, and in one there were worms in the bowels." In the case he particularly records to the Society, "the predisposing cause might possibly be the small polypus which was found attached to the mucous membrane, near what appeared to be the upper end of the invaginated portion" (*Trans. Path. Soc.*, vol. xv., p. 117).

As to how the lesion first commences some notion may be obtained, and the physiology of the process may to some extent be comprehended, by experiments on the intestines of animals while under chloroform, or just after having been killed. If a portion of the small intestine be pinched with a pair of forceps, active circular contraction and constriction of the gut immediately commences at the site of irritation. This constriction continues for some time, and is transmitted, or advances onwards, under the influence of the usual peristaltic action of the intestines. Wave upon wave of constrictions may be made in this way to follow each other in succession, so long as the vital irritability of the intestine continues. If the advance of the constriction onwards is impeded by any cause, such for instance as an undigested mass of food, a scybalous portion of fæces, a foreign body, or a polypoid growth, or even another constriction, and if the onward motion of the bowel fails to dislodge the obstruction, a partial invagination very readily occurs; but where the obstruction is necessarily localized (as from ulcers, polypoid growths, or fixed parasites) permanent invagination commencing in the vicinity of such local lesions is more readily induced. The mere weight of a polypoid growth would necessarily favour the occurrence of invagination by dragging down the bowel to which it is attached, and so inverting its coats. In 5 *per cent.* (BRINTON) the intus-susception is so caused; and often situated above the ileo-colic valve, the polypus having made its way through it, the spasmodic contraction of the valve would effectually prevent the spontaneous return of the invagination.

Another efficient and increasing cause of obstruction exists in the included mesentery. By its inclusion it causes such a drag upon the included portion that the mucous surface of the bowel is not only greatly corrugated, but by apposition against the containing bowel the narrow canal through the invagination becomes completely closed. The anatomy of the parts at once shows how this is effected. The mesentery being attached only to one side of the gut, it drags the intestine to that side; and the greater the amount of bowel invaginated, the greater will be the curvature of the included portion upon the mesentery as an axis; and the greater and earlier will be the complete obstruction.

Thus, when a case of intus-susception is examined after death, the invaginated portion of bowel is always seen in the form of a curve lying within the including intestine. The extreme end of the invaginated portion thus comes to be so turned upon itself that the canal by apposition is completely shut up. The blood-vessels of the impacted mesentery also undergo great and increasing congestion; and as strangulation and obstruction become complete, indurated masses of blood may be found effused within the folds of the mesentery. As strangulation increases, blood exudes from the mucous surface of the intestine, so that small flocculi of blood, as well as fluid blood mixed with mucus and free from fæcal matter, continue to pass *per rectum* as long as the canal remains open; and such symptoms (hæmorrhoids excluded) are justly regarded as pathognomonic of incarceration of the bowel from a simple intus-susception not yet completely closed.

Symptoms of Intus-susception.—The symptoms indicate obstruction and inflammation. *In the child* they mainly consist of restlessness, sudden fits of crying, and straining as if at stool; a discharge of mucus, more or less mixed with blood and free from fæcal matter, sickness, and anxiety of countenance. These phenomena are generally, but not invariably, preceded by a sudden and violent action of the bowels. A physical examination of the belly may disclose a tumor or swelling of the intestine; and sometimes, as in a case described by my friend and neighbour, Dr. Orsborne, of Bitterne, to the Medical Society of Southampton, the invaginated part could be reached with the finger introduced into the rectum.

In the adult the symptoms and phenomena in cases of complete strangulation of the gut by strictures, like internal strangulation from bands of lymph, or twisting of the gut round such constrictions, are sudden; and if the stricture be not relieved, the case proves fatal about the fifth or sixth day. On the other hand, in cases of intus-susception the symptoms of the incarceration are by no means sudden nor rapid in their progress; or, rather, they are comparatively slower in their development and progress to a fatal issue than cases of complete obstruction by strangulation are.

In cases of intus-susception the impediment from the first is partial, and in some cases the obstruction is never complete, although the symptoms may extend over many days, or weeks, or even months. One case is on record in the *Transactions of the Pathological Society* in which the symptoms of incarceration were present during four months, and although adhesions had formed between the serous coats of the invagination, yet obstruction of the intestinal canal was never complete.

A summary of the prominent phenomena of intus-susception may be stated as follows, showing the comparative slowness and incompleteness of the obstruction, as well as bringing forward those phenomena which (when weighed with other symptoms) may be regarded as pathognomonic of intus-susception. Diarrhœa early is generally the first indication of illness. In connection with this diarrhœa and its prevalence, the existence of parasites must not be overlooked, inasmuch as their existence in one patient renders it probable that they may exist also in others as a cause of the prevalence of diarrhœa in an epidemic form. Collapse, nausea, and diarrhœa, are the next symptoms to supervene, followed by tenesmus, or a feeling of fulness in the rectum; and after going two or three times to stool, *blood only* is observed *to pass*, and similar discharges continue. An enormous discharge of fæces may sometimes take place after an enema, but generally accompanied by *a large flow of blood.* So long as the patient lives *he continues to pass blood at stool,* and, on occasions, scybala. Small portions of hardened fæces may continue to be got rid of, but every effort is attended by *the usual flow of blood.* Purgative remedies aggravate the symptoms. Death is comparatively slow; and hence the extensive coagula which are found in the cavities of the heart.

Diagnosis is therefore mainly differential between this and other forms of alvine obstruction. The colic is extremely severe, and the marked remissions of pain are followed by exacerbations, which increase in violence with each repetition. Physical diagnosis ought to be had recourse to in every case. From hernia, cases of intus-susception are to be distinguished by the absence of the hernial tumor at the respective abdominal apertures where hernia is usual; but a tumor within the abdomen is a physical sign of the greatest value; and is not often absent, though easily overlooked, as it is often of small size (BRINTON). The condition of the rectum ought always to be ascertained by a digital examination. The vomiting is less urgent in proportion as the obstruction is lower down in the bowel; if the duodenum is involved, vomiting is almost incessant. Dr. Orsborne, of Bitterne, lately brought me the stomach and intestines of a child that died of this affection; and the relations of the parts involved were peculiar in this respect, that in the reflection of the colon, which contained the volvulus, the mesentery of the duodenum was dragged in, and along with it the adjacent wall of that intestine was also taken in. The parts are preserved in the museum at Netley.

It is necessary to distinguish between ileo-cœcal, or the colic invagination, and that of the small intestine. The former is distinguished from the latter by—

> "(1.) The prominence of tenesmus, which is rarely present in any marked degree where the small intestine only is implicated; (2.) The greater size and fixation, as well as the different site of the tumor, which, if large, generally proceeds towards the left side of the hypogastric or left iliac region; (3.) The subordinate share taken by hæmorrhage, which, instead of copious bleeding by stool and vomit, is often little more than a scanty admixture, scarcely sufficient to tinge the mucus passed from the bowels, with violent and frequent straining by the patient; (4.) The still more subordinate share taken by obstruction, which not only seems to be often anticipated by death, as regards any complete symptoms of its presence, but to be really absent, owing to the patulous state of the axis of the invagination; (5.) The presence of the end of the invagination in the rectum" (BRINTON).

Obstruction of the small intestine is chiefly characterized by—(1.) The umbilical seat of the pain, which is also more early and severe; (2.) Vomiting is more early, severe, and frequent: obstruction is much more rapid, constant, and complete in the small than in the large intestine.

Prognosis is not always hopeless, but is neverthless most grave. The cases which recover are almost invariably chronic or protracted ones. In the fifteenth volume of the *Transactions of the Pathological Society of London*, Dr. Peacock gives an account of the passage of a large piece of bowel by the rectum in a case of invagination, followed by recovery; and in that paper he refers to eighty-eight cases of intus-susception in which portions of bowel are reported to have passed by the rectum. Those which end by expulsion of the *volvulus* have a duration of from twice to thrice as long as that of the fatal cases; and it is not till the second, third, or even sixth week that the remission of symptoms occurs which announces the relief of the obstruction, and which often precedes by a day or two the first healthy alvine evacuations.

It is to be remembered that distension of the involved bowel measures not only the danger but the rapidity of the fatal issue.

The cases of most serious import are those which, commencing in the small intestine, involve the ileo-colic valve; and the majority of fatal cases are those in which the cæcum and ascending colon have swallowed up, as it were, a large portion of the small intestine. The danger may not be at first appreciated, because the obstruction is never complete in the first instance, as we know from the nature of the lesion as well as from the accurate history of cases. The bowel at first is merely incarcerated by the invagination, and it is not until the middle and internal portions with their contained mesentery become compressed, constricted, and ultimately strangulated, that complete obstruction ensues. Till this occurs, the continuous expulsion of bloody mucus from the central tube of the inversion goes on. Inflammation and sloughing thus commence as the parts become subjected to more and more increasing pressure.

Two forms of inflammation prevail—namely, one serous, between the opposed peritoneal surfaces, and commencing at the angle of reflection of the middle on the external layer (*x*, *x*, *x*, in the diagram, p. 964), at the part where the one portion slips into the other. It is here that the peritoneal surfaces of the invagination commence to adhere; and up to the period of adhesion the ancient remedy, inculcated by Hippocrates, of injecting air into the great gut by a long tube, introduced *per rectum*, has effected the greatest number of cures—forcing, by gentle, persistent, and equal pressure, the invagination backwards, and so causing it to be undone. The other form of inflammation takes place between the two opposed mucous surfaces (*c*, *c*, in the diagram). At this angle of reflection an abundant white leucorrhœal-like secretion (corpuscular elements of inflammation with mucin) commences very early to be discharged by the rubbing of the opposed surfaces, and eventually the inflammation (mucous and serous) may be so destructive that ulceration and sloughing of the whole invagination may be the consequence. In this way continuity of the canal may be restored—the invagination having passed, *per rectum*, as a slough. Dr. Brinton believes this favourable termination occurs in not less than one in every two or three cases of intus-susception. On an average, separation of the slough is not complete before the eighth day, and the liberated bowel is rarely expelled *per anum* before the tenth or twelfth day, and is sometimes only prevented by the death of the patient on the fifth or sixth day. Of such cases eighty-eight are now on record—an analysis of which, by Dr. Peacock, has been already referred to (*Path. Soc. Trans.*, vol. xv., p. 114).

Treatment.—The following are the details of treatment for cases of intus-susception advocated by the late Dr. Brinton. The chief indications and treatment are—(1.) *To prevent distension*, by reducing, in every possible way, the quantity of food and drink, restricting the latter to small but frequent sips (preferably through a long straw or tube) of cool iced liquids. Food is to be given, as strong beef-tea, soup, or milk, with equal frequency and caution. Small doses of alcohol (as brandy with water, or soda-water) is to be alternated. But if any repugnance exists to food or drink, or if vomiting is excited by these articles, the amount given must be reduced. Water, milk, and gruel are to be given freely in often-repeated enemata.

(2.) *To assuage pain and to mitigate excessive peristalsis* suggest the same kind of remedy—namely, *opium*, which is to be given continuously and alone in the solid form—preferably as an *extract*. The practical limit of the dose is indicated by the comparative arrest of pain, the approach of narcotism, and decided contraction of the pupil.

Belladonna is only of use as a remedy to diminish the straining peristalsis. It may be given *combined with opium*, as *two* parts of *extract of opium* to one, two-thirds, or even one-half part of *extract of belladonna* in a pill.

Enemata are useful as a mechanical aid to removing obstruction. They may gradually distend the bowel at the site of obstruction, so as to effect such a change in its position and arrangement as may release the impacted portion. The administration of enemata is only safe and efficient if undertaken by a person of competent skill. The quantity of fluid must be injected little by little, and must be retained as long as possible; and the patient must resolve to tolerate some pain in reaching that climax of distension at which only enemata are calculated to relieve obstruction.

In cases where the intus-susception is in the large intestine, inflation of the bowel with air, as originally suggested by Hippocrates two thousand years ago, in his third book (Περι Παθων), has of late years been revived and adopted, first in America, and subsequently in this country (Gorham, in *Guy's Hosp. Reports*, and *Med.-Chir. Trans.*, vol. ix.) This Hippocratic remedy has undoubtedly been more successful than any other. Of twenty-eight cases, the details of which were collected by Dr. Orsborne, there were only *seven* recoveries; and three of these were effected by inflation of the colon with air. Dr. Murphy and Mr. Erichson have borne strong testimony to its value in discussions on the subject; and the former had recourse to the operation on one of his own children with success (MS. notes of Dr. Orsborne). For its success, Dr. Orsborne is of opinion that the remedy should be employed at an early period, before there has been time for adhesion between the contiguous surface of the volvulus. My friend Dr. David Greig, of Dundee, has had recourse to this method of treatment in numerous instances with perfect success, and has published an account of his experience in the *Edin. Monthly Med. Journal* for October, 1864. By means of the ordinary elastic enema tube, fitted to the pipe of a small pair of bellows, he was able to pass a considerable quantity of air into the rectum, continuing the process till the belly showed signs of considerable distension, and even till uneasiness prevailed. Its beneficial action is indicated by the relief of the urgent symptoms, such as straining and vomiting; and gradually a fæcal evacuation is obtained from the bowels. At the same time warm fomentations are to be applied to the belly. The use of large enemata, with manipulation, has also been recommended. A long stomach-tube is to be passed as high up the colon as it will go, and the anus being firmly compressed round it, warm water is to be slowly injected, so as to distend the bowel as much as possible. When the fluid is allowed to come away, the abdomen should be pressed upon with the hands, so as to move about the coils of intestine (TANNER).

The earlier any of these remedies are had recourse to, the greater will be the chance of recovery; and the patient may be put under the influence of chloroform to facilitate the manipulations. When all remedies fail, gastrotomy may be thought of, and its chances of success considered. The operation is advocated by Benjamin Philips, and in some cases it may be justifiable (see his paper in *Med.-Chir. Trans.*, vol. xxxi.) The only cases for which it seems suitable are in obstructions from bands, diverticula, and the like lesions affecting the small intestine. The object of the operation is to divide the cord-like cause of strangulation (BRINTON).

DIARRHŒA.

LATIN EQ., *Alvus soluta;* FRENCH EQ., *Diarrhée;* GERMAN EQ., *Durchfall*—SYN., *Diarrhöe;* ITALIAN EQ., *Diarrea.*

Definition.—*A frequent discharge of loose or fluid alvine evacuations, without tormina or tenesmus.*

Pathology.—This affection is rather a consequence or a symptom of certain pathological states than of itself a disease, and, therefore, it is difficult to give it its true place in Nosology. In many cases it is no doubt a natural effort to get rid of some irritating material which has been passed onwards from the stomach in such a form that it cannot be made available for digestion and nutrition. The diarrhœa or bowel flux in such cases is an effort of nature to wash the irritant away. On the other hand, the excessive secretion is sometimes due to local lesions of the mucous membrane, which can be demonstrated after death—*e. g.*, *Catarrhal inflammation, follicular disease, lardaceous disease.* The irritant material passed through the stomach and these local lesions are causes of the increased flux or diarrhœa. It is also sometimes a consequence of sudden mental impressions, as of fear or unpleasant news; also as a consequence of exposure to cold. It is a characteristic symptom of *malignant cholera,* concurrent with whose presence it has often been observed that there is a wide-spread prevalence of diarrhœa.

The majority of cases of diarrhœa, however, are not to be regarded as *miasmatic* in any sense. They are to be regarded here as local diseases from irritation of the bowel or from local lesions; whereas *summer cholera* (already considered) is different from a mere bowel flux, and is regarded as a disease affecting the individual in his entirety; and the diarrhœa which is associated with cholera must be classed as *choleraic diarrhœa,* and regarded as subordinated to *malignant cholera,* and part of the phenomena of that disease (*Brit. and Foreign Med.-Chir. Rev.*, Oct., 1869, p. 361).

Thus there are many agents, both of a moral and physical nature, that act thus upon the human body, causing diarrhœa; and as there are also many known morbid poisons which bring about this state, it merits some notice in the class of diseases now under consideration. It is often a morbid action of function, rather than any disease of structure, being unassociated with any definite specific lesion of vital parts. It may be regarded generally as the immediate result of unwholesome diet, excess in food or drink, cold, wet, fatigue, and exposure, and various functional derangements of the biliary and gastro-intestinal apparatus.

Cases of diarrhœa with collapse have been recently described by Drs. L. Buhl, E. v. Wahl, v. Recklinghausen, Zalesky, and Waldeyer, which they have associated with the presence of a fungus development in the stomach and intestines, under the name of *mycosis intestinalis.*

The prominent symptoms were vomiting and diarrhœa occurring suddenly during apparently robust health, and quickly followed by lividity of hands and face, collapse, and death in from two to six days. The mucous membrane of the pharynx, larynx, and trachea they describe as excessively red. The lungs are filled with blood, and of a dark colour. Œdema of the areolar mediastinal and sub-peritoneal tissue existed, and also of the neck and larynx. Right cavities of the heart filled with blood. The peritoneal cavity contained much turbid fluid; and the connective tissue along the backbone, progressing downwards and forwards to the sides of the abdomen, was characterized by blood-injection, strongly ecchymosed, and œdematous in a high degree. The wall of the stomach and the whole intestinal canal were similarly injected with venous blood and œdematous. The mesenteric glands were enlarged, or their position indicated by an ecchymosis spreading around them and between the layers of the mesentery. The lymphatic glands of the thoracic and abdominal cavities were also swollen and thick, being permeated by hæmorrhagic infarction. Furunculoid hæmorrhagic patches —roundish (three-fourths of an inch in diameter)—of increased thickness and prominence existed upon the mucous surface of the stomach, having an umbilicated depression, and a feebly yellow-coloured centre resembling a flat scab or commencing ulcer; another patch existed on the small curvature of stomach, near the pylorus (one inch and a half long and an inch broad), with puffy margin and uneven depression. Similar patches, to the number of fifty-nine or sixty, were found from the pylorus to the ascending colon—the smaller patches in the small intestine resembling œdematous flabby swellings, slightly injected.

The lesions, as regards their seat and distribution, were different from those of any known disease.

Very thin microscopic sections of the affected parts, proceeding gradually from the innermost surface of the mucous membrane towards the peritoneal coat, showed now and then black particles of granular pigment; and to the *villi* of the affected parts there adhered groups of zooglœa, the large ones generally having the central position, with smaller groups extending peripherically towards the healthy tissue. These molecular corpuscles were mostly imbedded in gelatinous connective tissue. They were of oval shape, which could only be recognized by powers magnifying 800 to 1,000 diameters. Buds could also be distinguished (but only with the very highest powers) at the sides of some, as in yeast-cells.

The entire substance of the *villi* was permeated with these corpuscular groups, and rarely could *mycelium* be noticed outside the groups. Buhl believes that these corpuscles belong to the group of fungus structures known as *schizo-mycetes* (see page 209 to 218, vol. i.) The submucous tissue was, however, traversed by *mycelium-fibres* of considerable length, but without producing any change in the elements of the tissue itself, except that a considerable number of pus-cells (colourless blood-cells?) were infiltrated into the tissue, and the blood-vessels contained less coloured than colourless blood corpuscles. Buhl considers that the fungus formation had grown from the surface of the mucous membrane towards the interior layer, the thick felty mass of *mycelium-fibres* penetrating the submucous tissue. The fungus fibres (hyphens) were also found penetrating the mesenteric and portal vein, floating among the blood corpuscles. The blood of the whole body contained abundantly isolated small bodies (*conidia*), and the number of colourless blood corpuscles was evidently increased (*lecuocythœmia*). For one *white corpuscle,* Buhl recognized 50 *red ones,* and 350 *conidia.* The mesenteric gland and the lymphatic glands, already noticed, were so much infiltrated by the *mycelia* as to cast the structural gland part into the back-ground.

The presence of isolated *conidia* in the general blood, and of *mycelia* in the portal vein, proves that they passed from the mucous membrane through the veinous radicles and lymphatic vessels. They were found in varicose lymphatic vessels of the serous membrane of the stomach.

The œdema, the hæmorrhage, and the exudation into the abdominal cavity, and the diarrhœa are regarded by Buhl as due to the mycelia entering into the mesenteric veins, and into the lymphatic vessels and glands.

Dr. Waldeyer recognizes in this form of diarrhœa evidence of embolic lesions resulting from the enormous quantity of very minute *zooglœa*, especially in connection with the vessels of the skin, heart, liver, intestine, kidneys, and lymphatic glands. The smaller vessels of these were blocked up by the fungoid elements, and, from the plugging up, led to hæmorrhagic infiltration of the parts, to which the ecchymoses are referable. Finally, he regards the disease and the diarrhœa as a form and result of *malignant pustule* (MILZBRAN) transferred from animals to man. One of his patients was a cattle-feeder; and the swelling of the lymphatic glands and spleen, with the carbunculous affections of the mucous membrane of the stomach and bowels, are constant symptoms of affected cattle, and the enteric fever of horses and cattle plague, in whose blood "*bacteria*" are said to have been found (*Zeitschrift fur Biologie*, vol. vi., 1870; Virchow, *Archiv.*, vol. xxx., p. 366; 52, 1871, part ii., p. 541; and *Handbuch der speciellen Pathologie*, vol. ii., p. 387).

Symptoms and Forms of Diarrhœa.—Nosologists have generally divided the disease into varieties founded on the different states of the discharges; but as these do not depend upon definite pathological states, the classification may, at first sight, appear to be of little use. Nevertheless, the state of the discharges furnishes important indications in the treatment of diarrhœa. The most common appearances are due to the predominance of fluid feculent matter, or to bile, mucus, serum, chyle, or where undigested masses of food pass unchanged, giving rise to what is termed a "lientery." But the discharges are more often of a mixed kind, made up of several of those states.

The idiopathic forms of diarrhœa which require notice are,—(1.) *Diarrhœa of irritation;* (2.) *Congestion, or inflammatory diarrhœa;* (3.) *Diarrhœa with discharges of unaltered ingesta* (*lientery*).

(1.) *Diarrhœa of Irritation.*—This form comprises most of the cases denominated *feculent* by authors. It is induced by stimulating or irritating substances received into the stomach, excesses in eating or drinking, or even by a small quantity of unwholesome food, or by poisoning from animal effluvia and from certain mineral poisons, or what constitutionally disagrees with the patient. In infants it is often brought on by unwholesome conditions of the milk, such as the persistence of *colostrum* in it. Nausea, with severe griping pains before each evacuation, a foul, loaded tongue, copious feculent stools, watery, mucous, or bilious, and becoming frothy, are the phenomena of this form of diarrhœa.

(2.) *Diarrhœa from Increased Vascular Action.*—This variety is caused by whatever induces a greater flow of blood to the intestinal mucous surface, and at the same time lessens or obstructs the cutaneous elimination of fluids; the application of cold to the cutaneous or pulmonary mucous surfaces, or to both at once; cold acid drinks, or ices taken when the body is overheated; suppression of perspiration or of accustomed discharges; checked menstruation or lochial discharge.

The evacuations are watery or serous, with mixed feculent matter, and exhibit every shade, from a dark brown, greenish-brown, to a pale greyish or whitish colour; and they contain, in some cases, pieces of thick gelatinous mucus, or thin, glairy, and stringy mucus. In other instances, whitish albuminous flocculi are abundant in the stools; and in a few instances large membranous or albuminous shreds or flakes present a mould of the internal surface of the gut.

There seems to be conclusive evidence to show that much of the intestinal catarrh described by the common name of diarrhœa is associated with an *erythematous* congestion of the mucous surface of the lesser intestine, extending over a considerable extent, and rarely attended by increased arterial vascularity of the submucous tissue. When the symptoms of such congestive states are manifest during the progress of other diseases which terminate fatally, there may frequently be observed, besides the congested state of the mucous membrane, a marked increase of vascularity in other parts, such as the gastro-splenic omentum, mesentery, and glands, or infarction of the gastric glands, associated with congestion of the stomach generally. These phenomena for the most part are associated with a congested state of the hepatic system; and, occurring in a person otherwise in good health, give rise to symptoms which have been considered as a disease, and variously named *enteria, enteritis, erythemoidea, diarrhœa mucosa,* seu *catarrhosa* vel *catarrhale.* During the autumn and winter months in this country it is common to meet with such cases of disordered bowels in adults; and in children at any season, characterized by frequent fluid alvine discharges, and associated with extensive superficial irritation of the mucous surface. When the irritation predominates towards the upper part of the intestine—in the duodenum, for instance—the symptoms are an inclination to sickness, speedily followed by copious feculent discharges; the surface is easily affected by cold, and the individual may even shiver. There is also thirst, and a feeling of internal heat over the epigastric region. The functions of the liver are manifestly disordered at an early period, as expressed by the dull yellowish colour of the conjunctiva, and sallow darkness of the complexion, especially round the eyes. The tongue is generally moist, but viscid, clammy and furred. The appetite is completely lost, in the first instance. The skin is dry, and the palms of the hands and soles of the feet become unpleasantly hot and burning. The bowels generally become distended with flatus; and there is an uncomfortable sensation of distension, incapacity to expel the air, and occasional griping of the bowels, which are constantly producing a rumbling noise. The stools are at first large, feculent, and consistent, but subsequently they become watery, and even mixed with blood; then *tenesmus,* or a tendency to strain at stool, comes on and increases. Undigested articles of food are also passed—the characteristic symptom of *lientery* or *diarrhœa crapulosa* of the older authors. The belly is not painful when pressed, as in peritonitis or acute inflammation of the bowel, but there is often a deep-seated sense of uneasiness. This state soon terminates; in general favourably. It is more especially brought on by exposure to great changes of temperature in humid and moist weather, by wet feet, damp beds or clothing, and improper dieting (after over-feeding, especially in summer weather) at irregular times; certain articles of food, imperfectly fermented malt liquors, acid wines, and sour unripe fruits, drastic purgatives, and various mineral poisons.

A diarrhœa of whitish stools is often extremely persistent, and may reduce the patient to the very verge of death. It may supervene after injuries which may induce cerebral concussion; or it may come on after dysenteries have been cured. There may be no fever, the appetite may be good, and digestion may seem tolerable, but emaciation and weakness become daily more and more marked. At first the motions only increase to two, in place of the usual single daily one. Afterwards the calls to stool increase, so that, during the twenty-four hours, there may be eight or ten in the day. The desire to evacuate the rectum becomes sudden; and the stools are apt to pass involuntarily, preceded by little or no premonitory sensations, and consisting merely of two or three table-spoonfuls of muco-gelatinous matter, resembling thick milk or puriform fluid, or like a jelly. A similar state of mucous membrane lining the small gut gives rise to a white secretion from its surface: so that white milky stools are observed to flow—*Diarrhœa alba* of Hilary —a form of bowel disease which is sometimes epidemic in Barbadoes.

In addition to the symptoms noticed in the former variety, there is, in this form of diarrhœa, a dry, harsh skin, with increased temperature of the trunk, a flatulent state of the bowels, a small, frequent, constricted, but soft pulse, a furred or loaded tongue towards its root, with red edges and point, and scanty, high-coloured urine. In infants this variety is known as the "watery gripes," and often precedes fatal exhaustion in them.

(3.) *Diarrhœa with Discharges of Unaltered Ingesta.*—This is essentially an atonic form of diarrhœa, and very different from the last variety. It corresponds to the "*diarrhœa lienterica*" of the older authors. The most marked and characteristic phenomena which attend the disease are due to the almost total suspension of the digestive, assimilative, and absorbent functions, the *egesta* often differing but little in appearance from the *ingesta.* Such a form of diarrhœa occurs more frequently in children before the period of the second dentition than at later periods. It is frequently the consequence of previous inflammatory irritation of the alimentary mucous surface and disease of the mesenteric glands. It seems as if, in this variety, the stomach and intestines had lost their true tone or vital energy, as well as the mucous membrane of the alimentary canal; and it no doubt results, in the first instance, from impaired digestion. This was a frequent form of diarrhœa amongst the soldiers in the Crimea, as observed by Dr. Lyons; and the soldiers themselves observed it, and were in the habit of saying, "It is of no use eating, as our food passes through us in the same state as it goes in." The appetite is usually voracious; and when this form of diarrhœa continues long, the debility becomes extreme; and when death takes place, it is from stupor and exhaustion.

In a practical point of view, these are the principal varieties of diarrhœa which require to be distinguished apart from that of *summer cholera* and *malignant cholera* and *choleraic diarrhœa*; and the diagnosis of the form of diarrhœa symptomatic of the invasion of other diseases are noticed under the special diseases of which they form a part.

Treatment.—For practical purposes, the treatment of these three forms of diarrhœa may be founded on the following indications—namely, *first*, that in which the *tongue is clean*, the pulse quiet, and all constitutional re-action absent; and, *second*, that in which the *tongue is white* and coated, the pulse accelerated, some fever present, and the pain or soreness constant and increased by pressure. The stools in either case may be black, green, white, or mixed with blood indifferently.

When the *tongue is clean*, if the disease be quite incipient, the usual practice is to give one dose, consisting of an opiate, combined with a gentle cathartic. The form may be *one grain of opium*, combined with a *drachm of compound rhubarb powder*, or combined with *three to five grains of calomel*. To remove any offending matter that may be present, their action may be aided by *castor oil*, or a *saline cathartic*, such as a *seidlitz powder* or *compound senna mixture*. Sometimes it may be advisable to omit the opium, and to combine antacid remedies with the laxative, as in the following prescriptions:—

℞. Sodæ Bi-carbonatis, Hydrargyri cum cretâ, ā ā gr. ii.—ad gr. v.; magnesiæ Carbonatis, gr. iii.—ad gr. vi.; Pulv. Rhei, gr. v.—ad gr. viii.; *misce.*

Or:—

℞. Sodæ Bi-carbonatis; Pulv. Rhei; Pulv. Calumbæ, ā ā gr. iv.—ad gr. vi.; *misce.*

The administration of such a powder may be repeated at intervals—twice or thrice a day; and *ipecacuanha* in small doses (a quarter or a sixth of a grain) may be sometimes advantageously combined with each dose. These medicines having produced their intended effect, others more distinctly astringent may be administered if the diarrhœa persists. In many cases a drachm of *syrup of poppies* after each stool is sufficient. In severe forms of the disease a scruple to half a drachm of the *compound chalk powder*, in some aromatic, such as peppermint or cinnamon water, every four or six hours, is an excellent remedy; and these medicines may be used whether blood be or be not in the stools. If the opiate and aromatics contained in the above medicine should prove insufficient, it may be necessary to add to each dose some of the class of pure astringents, as a drachm of the *tincture of kino*, or of *catechu*, or *hæmatoxylon*, or of *iron*.

The following formula, prescribed by John Wiblin, Esq., F.R.C.S., for the men working in Southampton Docks, and known as "Diarrhœa Mixture," has been found of much service:—

℞. Conf. Aromat., ʒiij.; Sodæ Carb. ʒiss. (Bi-carb.); Tinct. Opii., ℥ss.; Æther Chloric, ʒiij.; ob. Caryoph., ♏. xl.; Mucilag. Acaciæ, ℥vi. Aquæ Destil, ad. ℥xxiv. *misce.* Two table-spoonfuls for a dose. This quantity may be given every two or three hours; or every hour; or every half hour, should the purging continue.

Absolute rest in the recumbent posture must be maintained, and warmth applied to the surface of the abdomen; and bland denulcent food such as arrow-root with beef-tea or gruel may be taken.

There are cases of diarrhœa with a *clean tongue*, which will not yield to *laxative remedies*, nor to *opiates, astringents*, or *stimulants*, either singly or combined, and which probably depend on a want of tone in the intestine; and in these cases *five grains of salicine* every four or six hours have often stopped a diarrhœa that appeared fast hurrying the patient to his grave. *Tincture of the sesquichloride of iron* is similarly useful, in doses of five to ten minims.

In the diarrhœa of whitish stools, with frequent calls and sudden desire to evacuate the rectum, and when muco-gelatinous matter like a jelly is passed, no remedy is of so much service as the *extract of nux vomica*, to the extent of *a fourth to a half grain dose;* or *strychnia* to the extent of *one-twelfth* of a grain, in a pill twice or thrice a day, with the *sulphate of iron* and *extract of calumba*. Dr. Maclean gave *tincture of the pernitrate of iron*, gr. x. every half hour, in this form of diarrhœa, with great benefit; and it is in diarrhœa of this kind that iron is of so much service. My attention has been called to a new preparation of iron by Mr. W. A. Moss (dispenser of medicines of the Army Hospital Corps at Dublin), which seems to possess some desirable properties, especially as to solubility and freedom from the inky astringent taste of preparations of iron. Its preparation is given in the note below.*

Black or dark stools *(melæna)* are not so much due to bile *(atrabilis* of Abernethy) as that such stools, resembling pitch, are principally composed of morbid or impaired secretions from the intestines (HOFFMAN, HOME, GRAVES). In such cases the discharge of the black matter is followed by a feeling of relief to the system generally. In cases of true *melæna*, where the dark colour is due to blood, great debility and sometimes fainting may follow the evacuations. Stimulating and tonic remedies, such as turpentine, are of benefit (GRAVES).

When diarrhœa is accompanied by a *white furred tongue*, together with pain and soreness, it is necessary to give opiates, combined with some mild purgative. Thus, *half a drachm to a drachm of Epsom salts* with a *drachm of the syrup of poppies;* or *fifteen minims of the tincture of hyoscyamus;* or, in severe cases, with *three* to *five minims of tincture of opium*, every *four* or *six* hours, are remedies on which, as a general principle, we may very confidently rely. In other cases, *rhubarb, castor oil*, or any other *mild purgative*, may be substituted for the Epsom salts. In cases of diarrhœa accompanied by vomiting, *a drachm of syrup*

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* *Ferri Ammonia-Phosphas.*—Heat common phosphate of soda to redness. Take of the pyrophosphate of soda so obtained ʒij. Dissolve in one pint of warm water. Then take of protosulphate of iron ʒiv. Dissolve in twelve ounces of water. Mix the solutions, collect, wash, and dry the precipitate at a gentle heat over a water-bath. Take of this precipitate ʒj., Liq. Ammonia P. L. ʒiss., water q. s. Dilute the Liq. Ammonia with an equal volume of water, and rub up with the phosphate of iron in a mortar until the latter is dissolved. Then dilute to ʒviij. Filter the solution and evaporate at a heat not exceeding 120° Fahr., over a water-bath and proceed as for the other scale preparations of iron. The dose is one fluid drachm.

of poppies alone, repeated every half hour, or every hour, for two or three times, may quiet the stomach, and enable it to bear the other remedies; or soda-water, or the effervescing draught, with a table-spoonful of brandy, with or without a few minims of tincture of opium, often remain on the stomach when everything else is rejected.

Most practitioners lay great stress on the colour of the stools, and the necessity of correcting the supposed morbid states of the liver; but the various colours of the stools are in many instances caused rather by morbid secretions from the surface of the mucous membrane of the intestines than by any defective state of the bile in the gall-bladder; and the conclusion from this consideration is, that, in simple diarrhœa, mercury (which is so often given in a routine way) in any form is either unnecessary or injurious in the majority of cases, except as a purgative. It is, however, sometimes necessary, and more especially in children under four years of age.

One general rule may be acted on in the cure of diarrhœa, which is, that in the adult, whatever be the form of the diarrhœa, if the stools be dark at first, and then become light-coloured, purgative medicines are no longer beneficial, and in no instance ought they to be continued longer than is sufficient to remove any irritative substance accumulated in the alimentary canal.

Sulphuric acid, in doses of the officinal diluted drug, of twenty to thirty drops, with water simply, or combined with the compound tincture of gentian, has been found a useful remedy. The sulphuric acid may be alternated with the nitro-muriatic acid, and prescribed in a similar manner.

The dietetic treatment should be limited to slops, puddings, and white fish boiled, and the drink to weak *brandy and water,* which acts locally as an astringent, and generally as a diffusible stimulus.

LARDACEOUS DISEASE OF THE INTESTINES.*

Latin Eq., *Morbus Lardaceus;* French Eq., *Lardacée;* German Eq., *Speckige oder wachsartige Degeneration;* Italian Eq., *Lardacea.*

Definition.—*A disease in which the blood-vessels of the villi, and of the mucous glands especially, become altered and added to by an albuminoid material, ultimately infiltrating the tissues and the glands, tending to hæmorrhages, diarrhœa, and ulcerations.*

Pathology.—Next to the spleen, liver, and kidney, lardaceous disease of the intestines comes next in frequency. In them it especially affects the arterial capillaries of the villi and the surrounding networks of the mucous and submucous tissue. Its progressive involvement of parts is as follows:—

It is seen—(1.) In the points of villi; (2.) Involving entire villi; (3.) In the mucous and submucous capillaries of inflamed parts; (4.) As annular infiltration round solitary glands; (5.) As degeneration of the vessels surrounding the sacculi of Peyer's patches.

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* Not recognized by the College of Physicians.

Virchow says he has seen the whole tract of arterial capillaries from the mouth to the anus in a uniform condition of lardaceous disease. I have repeatedly met with this condition in dead soldiers, at the invaliding hospital of the army now at Netley. Anæmia of the mucous membrane, with a peculiar glistening or shining aspect of its surface, are the most characteristic features. Otherwise there are no outward signs of the lesion to attract attention. Pallor, anæmia, and atrophy ought to excite suspicion; but the application of the iodine re-agent is absolutely necessary. The atrophy has advanced so far that Virchow has known the villi to drop off, and the intestine to be bare of villi where villi are usually present. The walls of the fine arterial twigs of blood-vessels become pellucid, transparent, glistening, rigid, and thick, and a reduction in the size of their calibre is the result. Blood ceases to pass through them, nutrition is impaired, and atrophy results, extending over large tracts of the bowel,—most decided towards the duodenum. The substance of the villi has frequently been found changed into the lardaceous or albuminoid material. Sometimes the mucuous membrane is also destroyed, and ulcers are developed which penetrate deeply into the tissue. The vesicles of Peyer's patches have been seen enlarged, as well as the solitary glands (FRERICHS). Lambl has traced the degeneration and destruction of the intestinal epithelium through the substance of the villi, the follicles of Lieberkuhn, and the muscular coat itself.

Hayem records lardaceous disease of the intestines in cases of scrofulous children supervening upon chronic suppuration of bone; and in whom similar lardaceous disease existed in other organs, with evidence of having commenced in these organs prior to the intestinal lardaceous lesion; thus showing that the intestines became involved at an advanced stage of the general disease. (*Compte Rendus de la Soc. de Biol. Gar. Med.*, 1866; and *Syden. Society Year-Book* for 1865–66.)

The lesion was most intense in the lower part of the small gut; and was found also in the large intestine, and also in the stomach.

Two stages may be recognized in the course of the disease, namely:—

First Stage.—Characterized by lardaceous lesions of the blood-vessels of the mucous membrane of the intestines, and enlargement of the submucous solitary glands.

Second Stage.—Breaking up of the lardaceous material and of the gland tissue, with destruction of the surrounding mucous membrane.

The morbid conditions and phenomena during life associated with these stages are as follow:—

During the first stage the evacuations are fluid, serous, and green; and the mucous membrane is seen after death to be covered by a thick layer of mucus (catarrh), easily removed by washing.

The small arteries and the capillaries are thickened, especially in the vicinity of Peyer's patches and solitary glands. These glands are tumid, and form projections varying in size from a millet-seed to a hempseed. They seem surrounded by a cup; and the projecting glands have a white semi-transparent aspect, are of firm consistence, and do not collapse if punctured. They give the usual re-action with the iodine solution as the thickened vessels and glands; and the minute vascular ramifications become visible on the pale surface of the gut, indicating an appearance of intense hyperæmia, if the vessels could be filled with blood.

The lesion commences in the cell-fibres of the small arteries and inner surface of capillaries. The small vessels in the interior of the gland follicles are affected; and the follicles themselves may contain minute concretions of the lardaceous material. Thus the appearance seems analogous to the condition of the spleen, as regards its glomeruli in lardaceous disease of that organ.

In the first stage of lardaceous disease of the intestine, the *submucous vessels* are scarcely affected, while the catarrhal changes exist; and as the disease advances there is enlargement, with similar degeneration of the mesenteric glands.

The second stage involves a transformation of the diseased follicles, and extension of the degeneration through the whole thickness of the mucous membrane. The follicles become yellow, and are afterwards replaced by the ends of thickened vessels lying in a pulpy yellow substance. This condition is sometimes followed by ulceration. The patches of Peyer become reticulated from loss of substance of the sacculi; and small ulcers may result in the patches, or in the solitary glands of the intestine.

The reticulated sieve-like patches of Peyer are represented by whitish prominent lines, circumscribing depressed spaces—giving an appearance of a fine lace-work or honeycomb, which represents exactly the distribution of the blood-vessels anastomosing and ramifying round the follicles in Peyer's patches.

In proportion as more fine vessels are implicated, so do the meshes become smaller and smaller; and the lesion extends along larger trunks of the vessels. Eventually the mucous membrane becomes thin. The iodine re-action follows the reticulation of the meshes of morbid blood-vessels over the patches. The ulcers of solitary follicles have the appearance of small depressions or losses of substance, with neatly-rounded borders, as if a round piece of membrane had been punched out; with a base smooth and granular formed of submucous tissue.

Yellow debris precedes loss of substance, which is due to molecular disintegration of the lardaceous lesion rather than to ulceration implying pus formation.

The destructive process is a granulo-fatty disintegration, through which the follicle or gland entirely disappears—as it does in enteric fever lesions sometimes—without ulceration (see p. 548 vol. i.)

The vessels on section appear as glassy cylinders, with an extremely small opening and thick walls. The lesion eventually extends to the submucous areolar tissue, and even to the blood-vessels of fat vesicles, and to smooth muscular fibres.

Thus the lesion is traced—(1.) To the blood-vessels; (2.) In the parts supplied, and especially in the gland follicles and surrounding parts.

Symptoms.—Diarrhœa in the first stage, followed by hæmorrhage in the second stage. The stools become gradually liquid, but do not increase in frequency (one or two only daily) which are liquid, greenish, serous, white. The diarrhœa persists, and does not remit; and there is no colic nor tenderness.

Dr. T. Grainger Stewart noticed, for some years previous to 1868, that hæmorrhages from the stomach and intestine occur in cases of lardaceous disease independently of any ulceration of the mucous membrane. The principal symptoms during life are diarrhœa and hæmorrhage (HAYEM); the latter chiefly associated with the ulcerative changes, as erosion of the mucous membrane, and probably proceeding from ruptured vessels surrounding the follicles.

The conclusions to which Dr. Grainger Stewart has arrived at regarding hæmorrhage in lardaceous disease are as follow:—

(1.) That it is not a frequent occcurrence.

(2.) That next to the spleen the intestinal tract is its most common seat.

(3.) That it may occur independent of any ulcerative process.

(4.) That it probably depends upon rupture of the capillaries at the affected part.

(5.) That the lardaceous disease of the liver does not of itself induce hæmorrhage from the bowels.

(6.) That the hæmorrhage from the intestine occurs when the liver is free of the disease.

(7.) That the occurrence of hæmorrhage increases the danger of the patient.

(8.) That sometimes it comes and goes for years without markedly depressing the vital powers (*Brit. and For. Med.-Chir. Rev.*, 1868, p. 201).

COLIC.

LATIN EQ., *Colum;* FRENCH EQ., *Colique;* GERMAN EQ., *Kolik;* ITALIAN EQ., *Colica.*

Definition.—*A painful affection of some portion of the abdomen, caused by violent contraction of the muscular fibre of some portion of the intestinal canal.*

Pathology.—The remote causes of this affection are indigestion, exposure to cold, or other general cause, the effect of lead poison; and all periods of life, from infancy to old age, are liable to the affection. It also attacks either sex. It is seldom that persons die of colic; but such instances have occurred; and dissection has shown some portion of the intestines intus-suscepted—affording a strong presumption that the beginning of cases of intus-susception depends on a spasmodic constriction of some part of the intestinal canal. This view is supported by Mr. Blane's experience in veterinary practice, who states that in fatal cases of colic in horses, different portions of the alimentary canal are found strongly contracted, and much oftener the small than the large intestines, which also sometimes contain gas. The bladder appears to participate in the spasm, the urine being either frequently ejected or suppressed.

Symptoms.—Colic is usually sudden in its attack; and the patient consequently, without any previous indisposition, is often unexpectedly seized with a severe fixed pain in some part of the abdomen, but which is relieved on pressure, so that he either sits doubled up, or rolls on the ground, or lies flat on the belly. In other cases, where much air is secreted, the bowels are greatly distended, and the pain is compared to a twisting or wringing around the navel, accompanied with soreness. The walls of the abdomen also participate in the internal spasm, so that the navel is often drawn in towards the back, or the heads of the *recti* muscles are exceedingly prominent, resembling so many round balls. The bowels are generally but not always constipated, and the stomach may or may not be irritable. In the latter case it often rejects both food and medicine. The pulse is little altered at the commencement of the attack; but if the paroxysm be prolonged, and the patient exhausted by pain, it may be hurried and frequent. The tongue is generally clean, although sometimes white and coated.

Gastralgia, or stomach *colic*, is a severe pain in the stomach, often so completely idiopathic that the slightest cause produces it. One person cannot eat a strawberry, another a gooseberry, another an egg, without being seized with it. In other cases every sort of diet produces it, so that the patient is racked with pain after every meal. Those affected are usually adults; and women are more frequently the subject of it than men.

The attack of *colic* is generally so sudden, that the patient is unexpectedly seized with a pain, which attains its greatest height on the instant, round or above the umbilicus. This attack is generally accompanied by sickness or vomiting, by great flatulence, and by a confined or sometimes by a purged state of the bowels. It may last from a few minutes to a few hours, and often ceases as soon as the stomach is emptied or the bowels have acted; but when the patient is costive, it very generally continues till the bowels are relieved by medicine, when it subsides almost as rapidly as it commenced, leaving, however, a soreness behind it. The pulse in this affection is natural, there is no fever, and the pain is relieved on pressure—circumstances which readily distinguish it from inflammation. The disease may subside after one attack; but genuine gastralgia sometimes lasts for many months.

Diagnosis.—Colic is distinguished from inflammation by the pain being relieved on pressure, and by the quiet state of the pulse.

Prognosis is in every case favourable.

Treatment.—The treatment of colic is by *opiates, chlorodyne stimulants,* and *purgative medicines.* When the bowels are constipated, *five grains of calomel, fifteen grains of jalap,* and *one grain of opium* should be administered immediately, and followed by the following aperient:—

℞. Mist. Camphoræ c. Magnesiæ Sulphat., ℥j.; Tinct. Hyoscyami, ♏xv. to xx.; Tinct. Cardamomi, ʒj. To be repeated every five or six hours until stools are obtained.

In mild cases a scruple of *rhubarb*, or half an ounce of *castor oil*, or other mild purgative, combined with a grain of *opium*, may be substituted for the opium, calomel, and jalap. Some have doubted the propriety of administering opiates at the onset of the disease; but it is certain that a mild purgative, combined with a mild narcotic, will effect more than a drastic purgative without such combination. *Opium* in full doses (gr. i.

to ii.) is, indeed, more generally useful in colic than any other remedy. Enemata often give immediate relief. Externally, the application of large bags filled with hot *chamomile flowers*, or of *heated sand*, or *heated salt*, or of the *stomach-warmer filled with hot water, are useful.* The *warm bath, fomentations*, or a *large linseed* or *mustard poultice* over the abdomen, are also highly useful auxiliaries. The diet should be sago and arrowroot, with a little brandy: and for some time after the patient has recovered it should be light, and perhaps limited to fish and puddings.

CONSTIPATION.

LATIN EQ., *Alvus adstricta;* FRENCH EQ., *Constipation;* GERMAN EQ., *Verstopfung;* ITALIAN EQ., *Constipazione.*

Definition.—*A retention of fæces beyond the usual period, so that when they are passed it is with difficulty, and in a comparatively hard indurated state.*

Pathology.—Constipation is essentially a disease of function, and often exists without the slightest trace of organic lesion. Its physiological cause appears to consist in want of sensibility of the nerves of the mucous membrane of the alimentary canal to the stimulus of their ordinary fæcal contents, so that the peristaltic motion downwards is retarded; also, to general absence of mucous secretion from the glands of the intestine. It has been a question in what portion of the alimentary canal constipation takes place; and most authors have placed its seat exclusively in the large intestines. In posthumous examinations, however, formed, lumpy, hardened fæcal matter is sometimes found in the small intestines; and hence it is manifest that the seat of constipation may be either the small or the large intestines, and, perhaps, commencing with impaired digestion in the upper bowel, the lower one ceases to act with its normal energy in the expulsion of abnormal fæces.

Causes.—The remote causes of constipation are extremely numerous. Every form of impaired digestion, for instance, may be a cause of constipation. The existence of hæmorrhoids, or piles, is another frequent cause; as well as a too sedentary life, especially if too strictly applied to study. Also women labouring under amenorrhœa, or other functional disease of the uterus, have often constipated bowels; and almost every acute disease is occasionally ushered in by constipation. It is likewise a common concomitant of most chronic affections, as dropsy, diabetes, hydrocephalus, pyrosis, rheumatism, or mania. Many articles of diet are causes of constipation; many mechanical accidents also, as stricture of the alimentary canal; many medicinal substances, as *lead, opium,* or other *astringents,* are all causes of constipation.

Persons of all ages are liable; but it is most common, perhaps, after the middle periods of life. Both sexes suffer from it; but women, from their more sedentary lives, the greater capacity of their colon, and their greater delicacy on these subjects, are most disposed to it. When pregnant, it is a frequent complaint with them, as some suppose, from the pressure of the enlarged uterus on the colon.

Symptoms.—It is a law of the animal economy that most persons in health have one evacuation daily, and at the time when the organic sensibility is heightened by repose, as on getting up in the morning; or when it is excited by a meal, as after breakfast. If this usual period be prolonged the fæces become hard, knotty, or scybalous, and ultimately form large round balls. This retention of the fæcal matter often causes great distension of the abdomen from generation of gases, as well as pain, irritation, and a flow of blood from the rectum on passing a stool. In some instances the fæcal matter, whether retained in the *caput coli* or other part of the intestinal canal, causes so much irritation that constipation and diarrhœa co-exist at the same time, the solid matters being retained, set up an irritative diarrhœa, while the more fluid portions give rise to repeated stools—a complication often confirmed by the evidence of repeated examinations after death. Such are local symptoms.

The general symptoms are not less distressing than the local affections. The appetite is in general lost, the head aches, a gloom is cast over the spirits, the mind and body are indisposed to exertion, the temper is soured, and every pleasure of life embittered. Besides this general influence of constipated bowels over the healthy state of every function, there are few disorders which are not aggravated by its continuance, and few that are not benefited by its removal, while many are cured altogether. There is, indeed, no rule of health more important than that the bowels be kept regularly and daily open.

Instances of constipation of two, three, four, five, to perhaps fifteen days are not rare. A gentleman under the care of Mr. Benjamin Phillips passed thirty-seven days without any evacuation. In a case related by Dr. Willan, of a monomaniac who destroyed himself by a voluntary religious fast, the patient had a stool on the second day of this course, but not again till the fortieth day. An instance occurred to Dr. Williams, St. Thomas's Hospital, and related by Dr. Burne, in which the patient, a lady, had only four stools in a year; while a young lady, aged 18, was attended by Dr. Burne, who passed neither flatus nor fæces for six months.

The quantity of fæculent matter discharged in a state of health is about 8 to 10 ounces, but varies with the size and bulk of the person, the quantity and quality of the food he eats, and amount of exercise; but in cases of constipation where the fæces have been retained for a lengthened period, the quantity passed at one motion is often quite extraordinary. In some instances the fæcal matter retained collects in the caput coli, and forms a tumor so considerable that it has been mistaken for *fungus hœmatodes*, or an *aneurism.*

Treatment.—When the constipation is *occasional* and *accidental*, any of the milder cathartics, as the *sulphates of soda* or of *magnesia*, *castor oil*, *rhubarb*, *aloes*, or the *confectio sennœ*, or the *pilulœ colocynthidis comp.*, will in general open the bowels. In obstinate constipation, *tartarate of antimony*, to the extent of ¼th of a grain, combined with a drachm of *sulphate of magnesia* every hour, may often bring fæces away after nausea supervenes. If, however, the constipation is habitual, the *healthy habit* of a daily evacuation is not easy to re-establish.

The remedies that have been mentioned, though often successful, yet occasionally fail from the low tone of the sensibility of the mucous membrane of the intestine. In such cases of *habitual constipation*, the combination of a tonic with a laxative will often produce a more efficient action than a perseverance in the use of purgative medicines alone. Thus we often find two grains of the *ferri sulphatis*, or *an ounce and a half of infusion of gentian*, combined with a drachm of the *sulphate of magnesia*, given according to the urgency of the case, three times a-day, or every six hours, will often empty the bowel when the salt alone would fail.

In old persons, a combination of aromatics with the purgative, as in the *decoctum aloës*, is a more useful and effective remedy than the same or even a greater quantity of aloes alone.

Aloes is a most useful remedy when the colon seems specially at fault as a result of fever and debilitating diseases, sedentary habits and occupations. A most useful form is that of the watery extract, combined with aromatics alone, such as *essential oil of ginger;* or, with *quinia, extract of nux vomica, ipecacuanha,* and some aromatic oil.

Where *slow digestion*, with a *deficiency of mucus secretion* prevails, the following formula for a pill is useful, of which one ought to be taken an hour before dinner, and another an hour before breakfast also, if required :—

℞. Pulv. Ipecacuanha, gr. viii.—gr. xii.; Ext. Aloe, gr. xii.; *Sulphatis ferri*, gr. xviii.—xxiv. *Misce, et divide in pil.* xii.

Or, the *ipecacuanha* may be omitted; or the *aloes* only may be combined with some bitter extract such as *gentian.* Such pills ought to be continued regularly till the occurrence of a "loose motion," after which one only, an hour before the principal meal, will be sufficient.

Another formula for a pill of a similar nature is of use in habitual constipation, namely:—

℞. *Pil. Rhei. Co., Vel. pil. coloc. Co.—Vel. Aloe*, gr. ii.—iii.; *Pulv. capsici*, gr. i. To be taken *with* the *food*, and repeated daily as occasion may require (Pavy).

Purgatives ought never to be permitted. The diet ought to be especially attended to. More food, both solid and liquid, may require to be taken. When constipation is obstinate, and has resisted the stronger purgative remedies, a full dose of *opium* sometimes seems to relax the spasms of the muscular coat of the bowels, on which the constipation may depend. *Belladonna*, also, is often given with advantage in doses of $\frac{1}{6}$th to $\frac{2}{3}$rds of a grain every morning at rising, combined with *extract of gentian* (Trousseau).

All medicines for the relief of constipation ought to be given just before or with the food; and are to be regarded as acting *with the food* in bringing about the result. Pills or draughts at bedtime are not so useful.

In the constipation of children, *sulphate of potass* is recommended alike by Drs. Hillier and West. The following are useful formulæ:—

℞. *Potas. Sulphatis*, gr. xl.; *Syrup Rhei*, ℥ss.; *aq. Carni ad.* ℥iii.; *misce.* Dose—A table-spoonful for a child six years of age (Hillier).

℞. *Potas. Sulphatis*, gr. xii.; *Infus. Rhei*, ʒvss.; *Tinct. Aurant*, ʒss.; *aq. Carui*, ʒii.; *misce.* Dose—A table-spoonful for a child three years of age (West).

A severe case of *colic* with *constipation*, in the practice of Dr. Murchison, recovered under the use of one-half grain of the *extract of belladonna* every four hours, with *belladonna ointment to the abdomen, a warm bath*, and *castor oil enemata* (*Lancet*, Jan. 19, 1871).

When constipation arises from torpor of the colon, equal parts of *compound gamboge pill*, and of *compound colocynth pill* is recommended by the late Dr. Symonds of Bristol (*Lib. of Medicine*, vol. iv., p. 139).

Colchicum has also been found of service in doses of *ten* drops several times daily of the tincture of the root (Chapman).

The *compound extract of colocynth*, or *compound colocynth pill*, variously combined with small doses of *blue pill*, or with *ipecacuanha*, or with *podophylline*, or with *nux vomica*, is a safe and useful form for the relief of the bowels.

When constipation does not yield to the simple treatment which has been mentioned, recourse must be had to larger doses, or to more active purgatives. Thus *calomel*, gr. v.; *c. jalapæ*, gr. xv., is a dose which rarely fails to produce motions, and this, if necessary, may be followed up four hours after, either by the neutral salts in divided doses, or by a black draught in one dose. If a stronger medicine than the above be necessary, *elaterium* is of greater power, and from $\frac{1}{16}$ to $\frac{1}{2}$ grain is sufficient to begin with so as to avoid hypercatharsis.

When the stools indicate a deficiency of bile, *inspissated ox-gall* has been recommended. It may be given in the following formula:—

℞. *Fel. bov. purificat*, ʒii.; *Ol. Carui*, ♏x.; *Magnes. Carb.*, q.s.; *misce. Divide in pil.* xxxvi. Dose—Two to be taken daily (Waring); or:—

℞. *Fel. bov. purificat. Pil. Assafœtid. Co.*, ā ā. gr. xxx.; *Ex. Aloes*, gr. xx.; *Sapon. dur.*, gr. x.; *Pulv. Ipecac.*, gr. viii.; *misce. Divide in pil.* xxx. Dose—One or two may be taken daily before dinner (Copland).

The *resin of podophilline*, to the extent of $\frac{1}{6}$th to $\frac{1}{4}$th of a grain, combined with *compound rhubarb pill*, acts similarly to the combination of *rhubarb pill* with *calomel*. A lengthened period sometimes elapses before it operates.

The *tincture of benzoin*, to the extent of gtt. xx. three times a day, will, it is said, keep the bowels active and regular (Hastings, Streetan).

If medicines by the mouth have been insufficient, it may be desirable to hasten their action by enemata. The enemata may be simply a pint of warm water, 100° Fahrenheit; or the same quantity of warm water, with half an ounce of *common salt*. The *common soap* enema (a strong solution of soap) is likewise a valuable remedy; and when the constipation is great, half a pint to a pint of *castor oil, neat*, may be thrown up.

Sometimes the fæcal matter accumulated in the colon is so large in quantity, and so hard and impacted, that manual assistance is necessary to relieve the patient. Women sometimes suffer for years from constipated bowels; and a contrary state of bowels may take place from the irritation of hardened fæces, when they are much harassed by purging, which may exist more or less for many months. At length violent tenesmus comes on, with a bearing down most intolerable, much worse than a woman suffers in her confinements. On examining the rectum, a mass of hard matter may be found, which cannot be broken to pieces without the aid of an instrument; and consisting of a variety of undigested substances, which, when broken down, may be washed away by injections, to the perfect relief of the patient.

Dietetic Treatment.—The patient suffering from constipation should avoid *port wine* or *brandy,* and should eat freely of sub-acid fruits. The advice of Mr. Locke should also be strictly followed, that he should go daily at the same hour to stool; for such is the periodical regularity of all the functions of the body, that they are more regularly performed at accustomed hours than at any other time. *Oatmeal,* in the form of cakes, or well-boiled porridge, should be eaten for breakfast.

SECTION XI.—RELATIVE WEIGHT, MEASUREMENT, BULK, AND SPECIFIC GRAVITY OF THE SOLID VISCERA OF THE ABDOMEN.

Weight.—The *liver,* the *kidneys,* the *spleen,* and the *pancreas* are the organs which require special notice here; and in order to obtain positive data for the solution of many questions regarding their pathology, it is necessary to know the relative weight of these viscera.

The Weight of the Liver ranges in the adult from *fifty* to *sixty* ounces (avoird.)—*i. e.*, from 800 drachms to 960 drachms.

According to the experience of Frerichs, the relative weight of the liver in healthy individuals may vary from $\frac{1}{17}$th to $\frac{1}{50}$th of the body; and in adults it fluctuates between $\frac{1}{24}$th and $\frac{1}{40}$th. During the period of greatest growth of the body, the liver does not become enlarged in a manner proportionate to the increase of the entire body; and its diminution in old age is for the most part in advance of that of the body. Its substance in this respect, therefore, presents a marked contrast to the muscular tissue of the heart; for whilst the heart increases progressively up to an advanced age (BIZOT, BOYD), the mass of the liver diminishes (FRERICHS, BOYD); and in old age, as a general rule, there is senile atrophy of the organ.

The weight of the spleen in the adult ranges from *four* to *ten* ounces (avoird.)—*i. e.*, from 64 drachms to 160 drachms. The weight in *malarious hypertrophy* has risen as high as eighteen, twenty, and forty pounds. It has, in some conditions of *atrophy,* weighed not more than *half an ounce.* The weight in relation to the body is as 1 to 350 or 400, up to 40 years of age, and as age advances as 1 to 700.

The weight of the kidneys ranges from four and a half to five and a half ounces each; the left being about one fourth of an ounce heavier than the right; and the weight of both together, in proportion to the body, as 1 to 240.

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE SOLID VISCERA OF THE ABDOMEN AS TO AGE AND HEIGHT (Boyd).

Age and Sex.		Body-weight.		Body-height.	Weight of Liver.	Weight of Spleen.	Weight of Pancreas.	Weight of Kidneys.
Years.		Lbs.	Oz.	Inches.	Ounces.	Ounces.	Ounces.	Ounces.
1 to 2	Male,....	14	6	28·5	11·7	1·34	·44	2·65
	Female,.	13	2	27·7	11·17	1·04	·49	2·4
2 to 4	Male,....	20	0	31·6	16·85	1·58	·76	3·33
	Female,.	18	7·5	31·6	13·49	1·28	·68	3·14
4 to 7	Male,.....	25	8	37·5	19·13	1·85	·8	4·05
	Female,.	24	9	37·0	19·56	1·65	·8	4·26
7 to 14	Male,....	42	6	47·0	34·71	3·03	1·68	6·58
	Female,.	38	6	45·0	25·86	2·54	1·34	5·75
14 to 20	Male,....	68	0	60·5	57·76	5·19	2·19	9·34
	Female,.	63	14	57·7	54·33	4·68	2·64	9·09
20 to 30	Male,....	92	14·5	66·7	60·29	7·19	3·54	11·57
	Female,.	86	13	62·0	52·74	6·53	2·95	10·17
30 to 40	Male,....	98	3·5	66·5	58·11	7·12	3·47	11·35
	Female,.	87	0	62·0	53·61	6·13	3·05	10·34
40 to 50	Male, ...	102	0	66·8	58·06	6·19	3·48	10·89
	Female,.	84	9·5	62·0	49·03	5·04	2·73	8·8
50 to 60	Male,....	102	0·5	66·0	55·37	6·23	3·46	9·1
	Female,.	86	0	62·0	44·0	4·67	2·83	8·55
60 to 70	Male,....	103	13	65·7	48·24	4·82	3·1	8·83
	Female,.	86	14	61·5	42·98	4·07	2·8	8·28
70 to 80	Male,....	106	13	65·7	46·33	4·93	3·22	10·68
	Female,.	80	4	61·0	38·37	3·57	2·62	7·63
80 to 90	Male,....	99	0	66·7	41·01	4·27	2·83	8·25
	Female,.	79	0	60·0	34·64	3·46	2·37	6·86

Measurements.—The *liver* measures from ten to twelve inches from side to side; six to seven inches from front to back; and about three and a half inches thick at posterior and thickest part of right lobe.

The spleen measures about five inches in length; three and a half inches from front to posterior edge, and one and a half inches thick.

The kidneys each measure about four inches in length, two inches in width, and one inch in thickness; the left being generally a little longer and thinner than the right.

Bulk (represented by cubic inches of water displaced):—

Liver, average bulk from 88 cubic inches (Krause) to 100 cubic inches (Beale).

Spleen, average bulk 9¾ to 15 cubic inches (Krause).

Kidneys, average bulk about 12 cubic inches each.

Specific Weight or Gravity.—The *liver* ranges from 1·050 to 1·060 (Krause); and in fatty degeneration 1·030 or less.

Spleen about 1·060.

Kidneys about 1·050.

SECTION XII.—DISEASES OF THE LIVER.

HEPATITIS.

LATIN EQ., *Hepatitis;* FRENCH EQ., *Hépatite;* GERMAN EQ., *Leberentzündung*—Syn., *Hepatitis;* ITALIAN EQ., *Epatitide.*

Definition.—*Inflammation of the liver.*

Pathology.—Inflammation of the liver, or hepatitis, occurs in various forms, and results in several consequences. In all countries in the temperate zone, at least two forms of inflammation of the liver occur. One of these terminates in simple or granular induration, and has a clinical history of its own; the other terminates in a greater or less extent of softening and acute atrophy of the gland, associated with jaundice and febrile phenomena of a malignant typhoid type (FRERICHS). In tropical climates a third form of inflammation of the liver has a tendency to result in suppuration; and it is described under the name of *suppurative hepatitis* (ANNESLEY, CAMBAY, HASPEL, BUDD, MOREHEAD, MACLEAN).

The inflammation may have its seat in the fibrous envelope of the gland (*perihepatitis*); or in the sheath of the vessels in Glisson's capsule; or it may attack chiefly the glandular parenchyma, the portal or hepatic veins, or the bile-ducts.

Inflammation of the glandular or hepatic parenchyma is either circumscribed, leading to circumscribed abscess or to granular contraction, or it is diffusely extended over the entire organ, ending in a true *suppurative hepatitis,* and then, according as the process involves all the anatomical elements of the gland, or is limited to the areolar matrix between the lobules, it gives rise sometimes to softening and abscess, or acute yellow atrophy, or at other times to induration or cirrhotic degeneration.

(a.) *Inflammation of the Capsule of the Liver* (*Perihepatitis*) and of Glisson's capsule is rarely accompanied by serious derangements, unless the inflammation extends to the portal or hepatic veins, or causes obstruction of the larger bile-ducts—events of rare occurrence (FRERICHS). It may exist alone in some cases of syphilitic infection; but on examination after death it is most frequently found associated with *interstitial hepatitis.* Peritonitis, disease of the liver itself, or inflammation of neighbouring structures, such as pleurisy, are the usual causes of *perihepatitis.* The chief symptoms are, tenderness of the hepatic region on pressure, motion, or deep inspiration, without any change in the volume or situation of the organ. Jaundice, as a rule, is absent; so also are febrile phenomena.

(b.) *Inflammation of the Glandular or Hepatic Parenchyma* occurs either as a circumscribed process limited to isolated patches, or it is diffuse, extending over the entire organ in a more or less uniform manner.

It is the former variety which leads, in most cases, to suppuration and the formation of abscesses. The diffuse form, on the other hand, at one time induces rapid destruction of the glandular elements, with softening and atrophy of the organ, and at another time it induces induration and cirrhotic degeneration.

In the primary stage of the *acute form of diffuse inflammation of the liver* there occur patches of hyperæmia, while the peripheral portions of the lobules are infiltrated with grey matter, and a juice or fluid serum, rich in albumen, flows from the cut surface of the gland. The capsule in the vicinity of the inflamed part is rough and opaque. Destruction of the glandular cells results in all the cases; but whether atrophy of the gland occurs or not depends upon the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration. The disease is usually accompanied by a similar condition of the kidneys and spleen, when large quantities of albumen pass off in the urine, and the spleen exists in a state of acute tumefaction.

How far the affection is one originally of the *hepatic cells*, as Virchow describes, or whether the process starts from the *interstitial tissue* (to which the disintegration of the liver *cells is secondary*), as Liebermister thinks his examinations prove, are points not quite decided. In the former case, the appearance of the hepatic cells being mainly affected consists in their large size and swollen condition, from turgescence with albuminous material. Subsequently the cells seem to disintegrate, and with them the general parenchyma of the inflamed part. When the part involved is a limited portion, a cavity may appear in its interior, filled with the disintegrated elements of the tissue.

Thus the most important *post-mortem* changes to be seen in such livers are, the occurrence of softened portions, having an irregular form, of a pale yellow or reddish-brown colour. All traces of the outline of the lobules are obliterated, while they are distinct in the firmer portions. The secreting cells of these parts are everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of colouring matter. Sometimes the softening follows the ramifications of the portal vein, and the hepatic cells in the softened parts are destroyed, being converted into a granular debris, oil-globules, and pigment-molecules. In the former portions the entire cells are loaded with fine granules. The gall-bladder, when treated with chloroform, fails to yield pigment. *Tyrosine* crystals are formed on the cut surface of such livers after some hours' exposure to dry air; and chemical analysis of the parenchyma yields large quantities of *leucine* and *tyrosine*. The symptoms of this form of inflammation are included in the account of the disease now known as *acute* or *yellow atrophy of the liver*, which will be given subsequently.

Treatment.—*Tartar emetic* in one-eighth or one-fourth grain doses every two or three hours is of service in the acute parenchymotous inflammation, if given sufficiently early—*i. e.*, within the first three days, when there is much vascular excitement, and a full bounding, unyielding pulse, with a dry hot skin and scanty urine. General blood-letting may be adopted if there be evidence of obstruction to venous blood-flow through the right side of the heart. Local depletion by leeches over the region of the liver, and also round the anus, so as more directly to unload the portal system, must be at once resorted to, followed by fomentations and large lintseed meal poultices, made as light and soft as possible, over the hepatic region. Mustard may be added to them, or laudanum may be sprinkled over their surface. *Saline purgation* and *alkalies,* with or without *colchicum,* are also to be freely administered.

Hot turpentine *epithems* are most beneficial. *Iodide of potassium,* in combination with *taraxacum,* is of great service in the chronic forms of hepatitis.

Alcoholic stimulants and fermented drinks must be absolutely forbidden, and the diet restricted to mild nutriment, such as milk, beef-tea, and farinaceous food generally.

ABSCESS OF THE LIVER.

LATIN EQ., *Abscessus;* FRENCH EQ., *Abcès;* GERMAN EQ., *Abscess;* ITALIAN EQ., *Ascesso.*

Definition.—*Suppurative inflammation, ending in a circumscribed collection of pus, or in several separate abscesses.*

Pathology.—Circumscribed inflammation and abscess of the liver, or, as it is sometimes called, *suppurative inflammation of the liver,* is always limited to one or to several isolated portions; and, with the exception of congestive turgidness of the contiguous texture, the remaining portions of gland-tissue are rarely implicated. The entire organ is never inflamed. The inflamed portions are usually found in a state of suppuration, which gradually destroys the hepatic cells. But on making sections of a liver in this condition, and in which the suppurating *foci* are in various stages of pus formation, it is usual to find discoloured, yellowish, or variously colour-marked spots, but all very soft, and set in a hyperæmic portion, from which it is fair to infer that such portions present the early or commencing stage of suppurative hepatitis. A microscopic examination of such spots shows the hepatic cells cloudy or granular, and lying in a granular material; which as a finely granular detritus at last takes the place of the cells. Sometimes the abscess-cavity is enclosed by a cyst; or they are surrounded by disintegrated discoloured parenchyma. At other times there is no defined boundary; but the inflammatory process extends till perforation occurs, or till several contiguous *foci,* or inflamed and suppurating portions unite into one large lesion; or till the pus finds an outlet.

The pus is rarely passed into the abdominal cavity; for adhesive inflammation of the capsule covering the abscess almost invariably occurs, so that attachments form to the abdominal walls and adjoining organs. If the abscess bursts into the abdominal cavity, the result is fatal peritonitis. Frequently the abscess perforates the thoracic or abdominal wall superjacent to the liver, and opens directly outwards. The locality for such spontaneous opening is usually the space below the ensiform cartilage. The pus may also discharge itself into the pelvic, inguinal, or sacral regions, close to the spine. Sometimes the abscess tends in an upward direction, to penetrate the diaphragm, when it generally empties itself into the right pleural cavity, but more often forces its way into the substance of an adherent right lung, by a distinct suppurative process, and in favourable cases passes by a free opening into a bronchus, whence it is discharged. The stomach, the duodenum, and the colon are the principal abdominal organs into which abscesses of the liver discharge themselves.

The hepatic abscesses are sometimes superficial, but more frequently deep-seated, and may be developed in any part of the gland; although they are most frequently found in the posterior portion of the right lobe. In size and in number they vary greatly.

Causes.—Abscess of the liver is rare in temperate climates. It has been caused by—(1.) Contusions or wounds; (2.) *Metastatic* or *pyœmic* inflammation, as inflammation of the portal vein, the irritation of veins from disease about the rectum—*e. g.* fistula in ano, operations for hæmorrhoids; (3.) Inflammation and ulcerative processes in the gastro-intestinal canal, as in dysentery (see p. 939); (4.) Inflammation and ulceration of the stomach, gall-bladders, or gall-ducts.

Symptoms.—Fevers, rigors, and severe headache and delirium are not uncommon as early phenomena of suppurative inflammation of the liver; but sometimes there are no symptoms pointing to disease of the liver; and the difficulties which embarrass the diagnosis of suppurative hepatitis cannot be over-rated. In 13 *per cent.* the disease runs a perfectly latent course, and in only 8 *per cent.* are symptoms at all well marked (LOUIS). In most cases a correct diagnosis will only be arrived at by not relying upon individual symptoms; but by taking a general view of the mode of origin and entire clinical history of the case, and after excluding, by comparison, the diseases of the liver and the neighbouring parts which may give rise to symptoms similar to those of hepatitis.

Practically the expression of symptoms denotes either—(1.) A superficial and adhesive inflammation of the organ; or (2.) A deep-seated suppurative inflammation of the substance of the liver.

The most prominent symptoms of hepatitis are, however, some tumefaction, pain, or uneasiness of the liver, or of the adjoining parts, as the thorax, abdomen, or right shoulder; an affection of the bowels, as diarrhœa or dysentery; and, lastly, pyrexia in a continued, remittent, or intermittent form.

When pain is present, it is found to be in most instances aggravated by lying on the right side, apparently from the greater weight pressing on the liver; while, in a smaller number of instances, the pain is felt most acutely on turning on the left side, probably from adhesions having formed to the ribs. In general, however, the easiest position is on the back, or a little over to the left side; and towards the termination of the disease the patient is sometimes observed lying in a position which he had previously declared himself unable to assume. As the disease advances, the pulse becomes frequent and hard, the skin hot, dry, and constricted, while pain, cough, and dyspnœa increase, and indicate advancing disease (MARTIN). Such a train of symptoms indicates superficial inflammation.

In a few instances, where the abscesses are small, acute hepatitis exists without any pyrexia. Some fever, however, is commonly present, and in general it commences with shivering, vomiting, and purging—symptoms which gradually diminish in a day or two, leaving the patient comparatively free from fever, and the pulse nearly natural.

These paroxysms, however, occur at intervals of various duration, sometimes returning as regularly as those of intermittent or of remittent fever, while in other cases the periods are less marked, the chief symptoms being rigors occurring at irregular intervals, frequent pulse, and sweats, the latter chiefly occurring in the night, and so copious as in some instances to pour off the body of the patient.

The state of the tongue is usually furred and loaded; but in the course of a long disease it becomes clean, or is only slightly foul. In some few instances, however, it continues brown and dry.

These instances of general suppurative inflammation are insidious in approach, and the destruction of the substance of the liver proceeds silently and rapidly. The existence of the disease is often not known till severe structural changes in the organ manifest themselves, and then they run a rapid and often fatal course. This is especially the case when the inflammation is induced by the combined effects of heat and malaria. The miasmatic affection of the blood, from deranged mucous surfaces, and from contaminated matters brought by the splenic vein, contributes to hyperæmia of the liver, which is often unnoticed till extensive inflammation develops itself. A burning sensation, with a mottled appearance of the skin of the hands and feet; an irritable temper; a capricious appetite; languor and persistent feverishness; frequent, settled, and increasing pains in the shoulder and back, are all pathognomonic signs of suppurative inflammation going on in the liver. The fever may be from time to time of a continued or intermittent type, and *the patient emaciates slowly*, becoming sad and desponding. The face becomes pale, cachectic, and has a patched or blotchy appearance. Sometimes there is jaundice, followed by anasarca and ascites,—from compression of the ramifications of the portal vein—intestinal catarrh, bilious diarrhœa, and dysentery. But icterus is by no means a constant symptom of abscess. It is absent in the majority of cases (LOWE).

The animal functions, as in phthisis, are often marked by the "cheerful hope" which illumines every hour the patient has to live; but in others the depression amounts to despondency, with restlessness and want of sleep. At last, delirium may obliterate the past, and throw a veil over the future, till the patient dies.

In the midst of the symptoms that have been mentioned, perhaps an abscess points; and the pus may have formed in the absence of rigors usually indicative of its formation. The patient then becomes hectic, his pulse rapid, and he is covered with a copious and clammy sweat. His life now in a great measure depends on the part where the abscess points.

Treatment.—Blood-letting will not cut short the morbid process in acute hepatitis; and Frerichs is of opinion that it should only be ventured on in cases of traumatic hepatitis, and in robust plethoric persons, where there is great tenderness, with enlargement of the liver, from congestion and urgent dyspnœa, from curdiac enlargement of right side. In the young and sthenic European in the East Indies, where there is obstruction to the flow of venous blood through the right heart, it is in general necessary to take fifteen to twenty ounces of blood, or till the skin becomes soft and relaxed, or the pain abates. Much benefit may be derived from the local application of leeches and large blisters.

The local abstraction of blood is efficiently accomplished by leeches round the anus. They act more directly on the portal circulation than over the hepatic region. When congestion is known to exist, leeches may be applied with benefit, combined with purgative extracts and antimonials. Ice applied to the region of the liver is also of great service, and the diet should be as limited as is consistent simply with the maintenance of life.

One practical rule seems to be established with respect to the use of *Mercury* in the treatment of hepatitis, which is, that it is not only inefficient, but injurious, and should not be given except as an occasional purgative when the local inflammation is subdued.

Purgatives are of use when the intestinal functions are sluggish.

In considering the treatment of abscess, and especially the points bearing on surgical interference, it should be remembered that hepatic abscesses tend to point or empty themselves in the following directions. These are stated in the order of their relative frequency:—(1.) Through the lungs (from 10 to 30 *per cent.*); (2.) Through into the peritoneum, the stomach, or some part of the intestinal tract (nearly the same proportion); (3.) Through the external integuments (in the minority of cases).

With regard to the treatment of abscess when it tends to make its way outwardly, it is still a question whether or not an artificial opening ought to be made, the prominences of the false ribs and obliteration of the intercostal spaces being considered sufficient, in the absence of fluctuation, to justify the operation (FRERICHS). In its performance care must be taken to prevent the entrance of pus into the abdominal cavity. With this object in view, the following method has been recommended by Bégin and Recamier:—(1.) Being decided as to the limits of the abscess, the patient is (2.) laid on his back, with the upper part of the body bent forward and the thighs flexed upon the abdomen; (3.) An incision from two to three inches long is to be made over the abscess, dividing the skin, the subcutaneous adipose tissue, the muscles, and the aponeurosis; (4.) The peritoneum is then laid open, as in operating for hernia, by slitting it up on a grooved director to the same extent as the primary incision; (5.) The wound is then to be dressed with charpie, and to remain untouched for three days; (6.) At the end of three days the dressing is to be removed, when the capsule of the liver will be found to have contracted adhesions to the margins of the wound so firm that the abscess may be opened without the danger of pus passing into the abdomen. I much prefer the method of Professor Lister.

Dr. Budd mentions the following expedient as a method of indicating whether the liver adheres to the abdominal parietes or not. When the liver is large, and the abdominal parietes are thin, it may be made out " by feeling the edge of the liver, or some prominent part of its surface, and marking the place of this with a pen on the surface of the belly. If the liver be adherent to the abdominal parietes, the line or spot so marked will correspond to the edge or prominence of the liver in all positions of the body. If it be not adherent, the liver will slide along the wall of the belly when the patient draws a deep breath, or changes his posture—the liver will fall, for example, towards the left side when he turns from his back over to that side, and the line or spot will no longer correspond to the edge or prominence in question" (*op. cit.*, p. 122).

A simple puncture is only warrantable when the pus has already penetrated through the superficial layer of the abdominal aponeurosis, or the intercostal muscles. In all other cases it ought to be avoided as dangerous. Indeed, Mr. Lowe (whose valuable records of cases of hepatic abscess in the *Madras Quarterly Journal* for April, 1863, merit the most careful study) thinks it better to allow an abscess of the liver pointing through the abdominal wall to open of itself, for the following reasons:—(1.) Because of the inelastic structure of the lobular substance of the liver not permitting the cavity to contract when a free opening has evacuated the pus; (2.) Because air invariably enters when an artificial opening is made, and rapid decomposition of the pus takes place, and renewed inflammation of the walls of the sac sets in; (3.) This renewal of inflammation and fever may end in gangrene, and may thus rapidly prove fatal; (4.) When the operation is entirely left to nature, *small worm-eaten-like openings serve to discharge the pus*, so that it has a slow but constant escape. As these apertures never close up, and as the matter is always oozing out, air cannot enter, no decomposition takes place (no septicæmia occurs), and no secondary fever sets in. The patient feels no shock from the loss of the matter, which escapes so gradually, and as it escapes, so Nature closes up the walls of the sac. Such is also the opinion and advice of Dr. Budd.

But others consider that the abscess ought to be evacuated under certain conditions. For example, a most interesting case of hepatic abscess is related by Sir Henry Cooper, of Hull, in the *British Medical Journal* for May 23, 1863. Sir Henry Cooper justly takes exception to the method of Bégin and Recamier, because it may provoke the very danger it is desirable to avoid; and he believes that in all cases in which the suppuration has extended so near the surface of the liver as to give the sense of fluctuation, irritation and adhesion of contiguous surfaces have taken place. The process also interposes a serious delay at a critical period. Therefore he advocates a direct opening into the abscess, *when there is reasonable ground for believing that an external outlet for the matter is the direction taken by the abscess.* Peritoneal connections are sure then to have taken place. Tenderness of the tumor he regards as the most satisfactory indication for the operation, and a hardened base of effused lymph a certain confirmation. Delay exposes the patient to the risk of rupture of the walls of the abscess by coughing, sneezing, or the like, and to the laceration of any adhesions which may have formed; or it allows him to perish unrelieved from the effects of constitutional irritation and consequent exhaustion.

A middle course was devised and carried out by Dr. Graves, in the Meath Hospital, in the case of a robust man. External elevation, hardness, and pain, eventually confined to one spot in the right hypochondrium, after acute inflammation of the liver, left no doubt of the formation of an abscess. The hardness was followed by deep-seated softness, yet no tendency was shown by the abscess to point outwards. The swelling remained stationary, and the integument of natural colour; but the general strength began to fail, and it became an important question whether the abscess should be opened by operation. It was objected—(1.) That the external tumor was diffuse; (2.) That the exact site of the abscess was uncertain; (3.) That failure to evacuate its contents might prove detrimental. A decision was therefore given *against* operation by the surgeons of the Meath Hospital. Under these circumstances Dr. Graves remembered that he had seen several cases where an incision made over a deep-seated abscess had failed to give vent to the matter in the first instance; yet in the course of a few days the pus found its way to the incision, and burst through it. He therefore proposed that an incision about four inches long should be made, exactly over the centre of the tumor—that it should be carried through a considerable depth of muscle, and, if possible, be continued *to within about one or two lines of the peritoneum.* This incision was then to be plugged up from the bottom of the wound with lint, and thus kept open, in the hope that the pus might tend towards the incision, and finally burst through it. Such an operation was performed. The abdominal muscles were found of considerable thickness, and quite healthy; and although the incision was very deep, yet the situation of the hepatic abscess was not felt more distinctly, so that it now became quite evident that no prudent surgeon would have persevered in an attempt to open directly into it. A result was therefore waited for. In two days afterwards the patient sneezed, when purulent matter in very large quantity burst forth through the wound. On closer examination it appeared that the incision had not been made exactly over the abscess, but rather to one side of it; for the matter did not come from the bottom, but from the side of the wound, and pressure on the liver on that side caused matter to flow out in abundance through the wound, which communicated laterally with the abscess.

This case teaches that if the attempt had been made to open the abscess directly, by continuing the incision, it would have failed, and the opening into the peritoneum and liver perhaps proved fatal (*Dublin Hosp. Report,* vol. iv., p. 40).

After the abscess has opened, strict rest must be enjoined; and sometimes the cavity is very long in closing up. Convalescence is always tedious; and sometimes the cicatrization of the abscess is imperfect, continuing to discharge pus at intervals for years (FRERICHS).

Because the liver has been repeatedly punctured (it is said, deeply) with an ordinary trocar without any evil consequences, beyond slight local irritation, it has been argued that our efforts should be directed to detecting by acupuncture the seat of an abscess, and evacuating it as soon as possible; and that this method of exploration or of "prospecting the liver" should be commenced as soon as symptoms present themselves indicating abscess of that organ! It has been argued that because some cases have recovered after such haphazard methods of finding and evactuating an abscess, it is a practice which should be made the rule, as already stated, in place of waiting for some indications that pus has even formed in the liver (MURRAY, CAMERON). Statistical data regarding the actual value, or even the safety, of this method of thrusting trocars into the liver in search of abscesses have not been published;

therefore the recommendations to the plan rest upon the opinions or impressions of two or three individual men, the records of whose practice are unknown. Certain it is that the plan recommended is opposed to the well-established principles upon which surgical interference rests, and is opposed to all that is known regarding the pathology of hepatic abscess, as set forth in the carefully recorded facts of Waring, Martin, Morehead, Frerichs, Budd, Maclean, and Lowe.

It is also stated by Dr. Cameron (the latest exponent of this method of puncture), that in cases of hepatic enlargement, where the trocar has been used more than once in an unsuccessful search for an abscess, the operation was followed by the speedy absorption of an enlargement of the liver, which had resisted all the routine methods of bringing about its dispersion—and that this is a native mode of dispersing hepatic and even splenic enlargements in India (*Lancet*, August 8, 1863, p. 169).

Puncturing parts, or acupuncture with a very fine neddle, is a surgical operation said to be much in use amongst the native Chinese and Japanese *hakims;* but the statement requires confirmation; and accurate information regarding the results of such operations are not on record. It is obvious, therefore, that such modes of dealing with large livers or *probable* hepatic abscesses, cannot be recommended here; and it should be remembered that a hepatic abscess alway begins from multiple foci (ROKITANSKY).

But we must still look to physicians of large Indian experience for information on this important subject.

Professor Lister's method of opening a lumbar abscess appears to me the one which ought to be adopted with regard to hepatic abscesses.

The time which elapses after opening an abscess till the patient's recovery is generally from one to two months. When convalescence is established, the functions of the liver remain torpid, and its substance often indurated; and subsequently, also, the spleen may be enlarged. In such a state Sir Ranald Martin recommends the use of *nitro-muriatic acid* baths. They promote the depurative functions of the liver, kidneys, bowels, and skin.

"The form and manner of preparing and using the acid bath are as follow:—Take of *hydrochloric acid* three parts; *nitric acid*, two parts; mix the two acids carefully and slowly, so as to avoid evolution of heat; and having waited for twenty minutes, add of distilled water five parts, and mix the whole carefully.

"*For a General Bath in which to Immerse the whole Body.*—(1.) Pour into the bath about five pailfuls of cold water; add two quart bottles, containing sixty-four fluid ounces of the dilute *nitro-muriatic acid*, prepared as above, and then sufficient boiling water to raise the temperature to 96° or 98°. The body is to be quickly and thoroughly dried with warm towels; and afterwards the patient must retire to a well-aired and warm bed. The use of the bath is only to be discontinued when tenderness of the gums or general *malaise* occur; and cuticular irritation from the acid is to be avoided by diminishing its strength. Iron and other forms of tonic remedies may also be administered at the same time, as well as opiates" (Martin *On Climate*, p. 564, *et seq.*)

ACUTE ATROPHY OF THE LIVER.

LATIN EQ., *Atrophia acuta;* FRENCH EQ., *Atrophie aiguë;* GERMAN EQ., *Acute Atrophie;* ITALIAN EQ., *Atrofia acuta.*

Definition.—*Simple jaundice, which gradually increases, with sensitiveness over the region of the liver, followed by violent constitutional disturbance, expressed by pyrexia, headache, delirium, hæmorrhages from various parts, and finally coma. The liver shrinks to one-half or one-third its normal size. A peculiar chemical decomposition takes place in the liver, whereby abnormal proximate principles are formed, which, being carried into the blood, may be discovered in various organs of the body, or, passing out by the kidneys, may be found in the urine.*

Pathology and Morbid Anatomy.—The disease was first described by Bright and Graves, and more recently and minutely by Frerichs in Germany, Wilks and Murchison in this country. The liver undergoes a most remarkable atrophy, and its section presents a mottled red and yellow appearance. The microscope shows that complete destruction of its secreting cells occurs, so that in the most severe cases not a single cell has been left entire; dark masses of biliary pigment, hæmatine, and fat alone remain; and the kidneys are found gorged with new products. The redder portions of the liver are more destroyed than the yellow, which may show remains of cells containing bile. It seems the result of a form of hepatitis, of the nature of *parenchymatous inflammation,* already frequently referred to—*cloudy swelling* of the cell elements, and their subsequent destruction by softening disintegration; so that the acini are not capable of recognition. The parenchyma is thus relaxed, shrivelled, and flabby, and the liver sinks against the posterior wall of the abdomen. The size of the organ is found after death to be diminished in all directions, but especially in its thickness. The gland is flattened out, flabby and wrinkled. Where the disease has advanced the farthest, the cut surface has an ochre-yellow or rhubarb-like colour; the blood-vessels are empty, and the traces of the outlines of lobules are obliterated.

At places where the process is less advanced extravasations exist, or their remains, in the forms of crystals of hæmatoidine. The lobules are encircled by congested vessels; and a dirty yellow substance separates them from each other. Afterwards the capillary congestion recedes, the size of the lobules diminishes, their colour becomes yellower, whilst the intervening grey substance gradually increases.

Bright has recorded the weight of such livers as reduced to *two pounds,* or to *twenty-three ounces,* and even to *nineteen ounces.* Frerichs records the weight of one as low as *one pound thirteen ounces;* its relative weight to the entire body being as 1 to 68 or 54—*i. e.* a reduction of more than one-half. Murchison records 28 ounces; or, one-half the standard for the age of the patient.

The **Symptoms** set in, like a bilious attack, with feebleness, rapidly followed by jaundice, febrile symptoms, and vomiting, delirium supervening by the third or fourth day. Convulsive fits soon pass into unconsciousness, associated with stertorous breathing, foaming at the mouth, clenched teeth, closed eyes, with pupils normal and susceptible.

The pulse is at first abnormally slow, but at the outbreak of the nervous symptoms it gradually rises to 110 or 120, and presents remarkable variations as regards frequency and volume. Towards the close of the disease it increases in frequency, and becomes smaller and smaller till it can no longer be felt. The body-temperature also rises very high. The symptoms generally resemble those of uræmic intoxication; collapse increases, perspiration becomes copious, and the patient usually dies comatose about the second day, more rarely about the fourth or fifth day after the first appearance of cerebral symptoms.

The extent of hepatic dulness diminishes more and more as the disease advances, and not unfrequently the dull space disappears completely, without any tympanitic distension of the bowels to cause the dull sound over the liver to be obscured. The spleen at the same time is increased in volume, and splenic dulness is increased. The bowels are almost always confined; and the stools are firm, dry, clay-like, deficient in bile, and at a later period not unfrequently dark-coloured and tarry, from the presence of blood. The urine, more or less saturated with a brown colouring matter, presents the re-action of bile-pigment, and deposits a light or greenish-yellow precipitate, in which the microscope detects the epithelium of the passages and of the kidney coloured yellow, and *needle-shaped crystals* (*tyrosine*) covered with colouring matter, either isolated or adhering in crystalline masses (FRERICHS). Sometimes the urine ceases to pass, and may be retained in the bladder as a clear bilious fluid, having a specific gravity of 1030 (WILKS); or ranging from 1012 to 1024, always of acid re-action, and occasionally slightly albuminous for short periods (FRERICHS). The evaporation of the urine gives crystals of *leucine*, which are in round masses, and of *tyrosine*, which are needle-shaped. Sometimes pure tyrosine falls as a sediment. These crystals are also found in the substance of the liver, but only in the hepatic vein, not in the portal vein or hepatic artery (FRERICHS). In Dr. Murchison's case they appeared after the liver had been immersed in spirit (*Path. Trans.*, vol. xix., p. 248). The question as to retention of urea or its lessening, as due to a want of complete metamorphosis of albuminous products, is not yet determined (PARKES, l. c., p. 286).

The disease runs a more or less violent and rapid course. In severe cases it has terminated at the end of twelve or twenty-four hours (FRERICHS). In other cases, in from two to eight days, urgent symptoms ending the disease by extreme prostration or excessive vomiting (WILKS). It is scarcely ever prolonged to a week, except when it occurs after jaundice, when the whole duration of the disease may extend over four to five weeks. After the commencement of the characteristic symptoms, the disease almost invariably terminates in five days; and as a rule the termination has been fatal to life.

The **Prognosis** is, therefore, in the highest degree unfavourable. Hæmorrhages are apt to occur (in one-half the cases) simultaneously from various parts of the body, usually from the stomach, bowels, or uterus. *Post-mortem* examination shows also extravasation upon mucous and serous membranes.

Pressure over the region of the liver gives rise to marked indications of pain, which are expressed even during coma.

Causes.—The mode of origin of acute atrophy of the liver is still unknown; but the circumstances under which the affection has occurred may be stated as follow:—Mental distress, dissolute habits, venereal excesses, syphilis, drunkenness, nervous depression, pregnancy, and the influence of miasmata. Violent mental emotions, the condition of pregnancy, typhus, pyæmia, scarlet fever, tropical malarious fevers, are the circumstances under which this form of inflammation is most apt to occur; and the destruction of the liver cell-tissue which ensues is indicated during life by the symptoms of atrophy, and by those peculiar changes in the urine which, at the height of the disease, are almost pathognomonic. In the cases described by Frerichs the urine is described of a brown colour, and smelling of sulphurated hydrogen, having a feebly acid re-action, and containing a small quantity of bile-pigment, but no albumen.

Looking to the occurrence of albumen in the urine of pregnant women, it may be that atrophy of the liver and the *nephritis* of pregnancy are both due to some condition of blood which in certain states of the constitution may be induced by pregnancy (WILKS). Atrophy of the liver has been most frequently observed in females; and more than half were attacked during pregnancy; and the period of life between twenty to thirty years of age seems most predisposed to the disease.

Acute atrophy of the liver is apt to be mistaken for typhus fever, complicated with jaundice or with pyæmia. The range of temperature may help to indicate the disease, inasmuch as the temperature is said not to be increased in cases of acute yellow atrophy (see FRERICHS, vol. i., p. 217).

Treatment.—English physicians recommend emetics and purgatives (CORRIGAN, GRIFFIN, HANLON). Frerichs recommends that the stronger purgatives should be used simply to remove congestion, such as *senna, aloes, colocynth;* and the doses should be such as to secure profuse evacuations. When atrophy has distinctly commenced, no benefit results from medicinal treatment. Severe pains indicate the use of *leeches, cupping,* and *cold cloths,* or *fomentations,* as recommended under enteric fever in vol. i. The occurrence of hæmorrhage indicates the use of *mineral acids.*

SIMPLE ENLARGEMENT—*Syn.,* CONGESTION OF THE LIVER.

LATIN EQ., *Amplificatio simplex*—Idem valet, *Congestio jecinoris;* FRENCH EQ., *Congestion;* GERMAN EQ., *Einfache Vergröserung*—Syn., *Congestion der Leber;* ITALIAN EQ., *Aumento di volume semplice*—Syn., *Congestione del fegato.*

Definition.—*Simple enlargement of the liver, from fulness or distension of its blood-vessels and bile ducts.*

Pathology.—This morbid state is expressed in various forms:—(1.) There may be *congestion from increased secretion of bile,* and the bile ducts especially are then gorged with bile; or there may be (2.) *Passive congestion* of the hepatic veins or of the portal veins; and (3.) *Active congestion,* chiefly involving the arterial capillaries.

Morbid anatomy assigns to each of these forms of congestion peculiar and characteristic appearances.

The liver, after death, is found enlarged principally upwards, so as to encroach upon the capacity of the right side of the chest. In shape it is apt to be increased in thickness, more than in length. Its covering appears smooth, tense, and glistening, and its substance firm. A section lets free a great amount of blood. The ducts are generally distended with bile, and this may arise from various causes, such as compression of the ducts from over-distended veins, frequently brought about by intermittent and remittent fevers. It is also a condition associated with the early stages of *cirrhosis,* when the appearance of the liver is that known by the name of *nutmeg liver.* The distension of the veins seems to be of a passive kind, and to depend on recession of blood from the surface of the body. An ultimate result of hepatic biliary congestion seems to lead to the filling of the hepatic cells with dark yellow matter; and when forms of passive vascular congestion are long associated together, ultimate atrophy and degeneration of the hepatic cells ensue.

The central part of a hepatic lobule is, in health, distinguished by a red spot, centrally situated, and measuring about half the diameter of the lobule. When this dimension increases it is characteristic of *intra*lobular congestion—a morbid state in which the hepatic veins are especially engaged.

In *inter*lobular congestion, where the *portal* veins are specially full or distended with blood, the centres of the lobules are comparatively pale, while the lobules are surrounded by red vessels.

The condition known as the *nutmeg liver* is explained by the various forms and results of congestion, as well as by other morbid changes of texture. It is so named from the resemblance, in combination and arrangement of colours, in a section of the liver to that seen in the section of a *nutmeg.* In its most marked forms a deep red congestion forms patches and streaks, which occupy the central parts of the lobules, and are *partially* surrounded by patches of a greyish or dirty white colour, and which are devoid of blood. The light-coloured parts may be caused either by fatty degeneration of the cells, which, enlarging, compress the capillaries and expel or prevent their being filled with blood; or it may be due to an increase from deposit and condensation of the Glissonian capsule in the early stage of that morbid condition known as *cirrhosis.* It also is a morbid state in which the capillaries are emptied by compression and contraction of tissue, or in which they have undergone atrophy.

Causes.—By far the most frequent cause of hepatic congestion is valvular disease of the heart (soonest in affections of the right side; later in those of the mitral; and latest in those of the aorta); and especially obstruction to the circulation through the right side, interfering with the emptying of the right auricle. These congestions, so far as the liver is concerned, are of a passive kind; and their most marked and constant effect is to produce *ascites,* with or without *anasarca.* If such congestions continue long, they lead to bilious contamination of the blood.

Congestions of the liver are chiefly brought about by sudden chills, the cold stages of fevers, over-abundant feeding, intemperance in alcoholic or malted liquors. Products of faulty digestion are thus at once carried to the liver, and influence its circulation; and so also does excessive bodily exercise in the heat of the sun.

Increased secretion and elimination of bile also leads to enlargement. This condition is brought about by some increased excitement of the liver, as by certain kinds of food or drink; but more especially, as is now well known, it is brought about in Europeans by exposure to unusually high ranges of temperature. In summer and autumn it is a morbid state not uncommon in our own country; but to the European on first landing in India it most commonly occurs, and in either case it occasions what is termed a "bilious diarrhœa." Great increase of temperature, combined also, perhaps, with change of climate and an unsuitable mode of life, is now acknowledged to have a direct influence on the functions of the liver, expressed, in the first instance, by an unmistakable increase in its secretion.

As a complication of other diseases, hepatic disorders are of most frequent occurrence, and for various reasons no true estimate has yet been made relative to the frequency of hepatic disease either in this or in other countries. Like most other exciting agents, however, the prolonged exposure to the influence of increased temperature, under conditions such as are experienced by the European in India, ceases to have a stimulant effect, and a depression in the powers of the organ results, corresponding to the previous excitement. In India the duration of the exaltation of hepatic function is not found to be of long duration, but is confined to the earlier years of residence, declining from that time. This increase of the secretion of bile and its elimination, so long as it lasts, is justly regarded as a salutary effort which nature provides to maintain the health. It is the decline of the powers of secretion, the arrest of the function, and the more or less sudden suppression of the secretion which is attended with danger. It always precedes the expression of the inflammatory tendency, and is associated with congestions of an active kind, in which the arterial system becomes prominently excited. This is consistent with what occurs in every other secreting organ. Previous to the expression of the inflammatory process becoming developed in a gland, the secretion at first flows freely under excitement (not necessarily morbid); but so soon as the inflammatory tendency becomes expressed, the secretion is at the same time dried up. This is well seen in the phenomena of a common cold, when the exciting cause at first merely stimulates the nasal passages, and marked, for instance, by an increased flow of mucus from the nose; but so soon as the inflammatory state becomes fully expressed, the secretion is dried up, and when re-established, is unmistakably altered in its properties. So it is with the liver when the increased flow of bile is suspended, from exposure to cold or some other exciting cause, a warning is given that the inflammatory tendency is about to be expressed by congestive and exudative processes in the texture of the organ.

Symptoms.—With excessive secretion of bile, the patient has purging of bilious stools, causing scalding of the anus. There is slight sickness just before the bowels act. A bitter taste is felt in the mouth, and the tongue is foul.

In all forms of enlargement the liver bulges on account of its swollen condition, which may be accompanied by uneasiness on exploration, or of weight on getting into the erect posture. The countenance and complexion may be pale, sallow, or dusky livid; and the tongue will be found coated, the bowels constipated, the appetite defective, and there may be nausea, vomiting, and headache. The pulse is slow, compressible, and irregular, or it may be quick and feeble; and, generally speaking, the symptoms are obscure. Dr. Budd, in his exhaustive work *On Diseases of the Liver*, thus notices how congestion is so common:—"Amid the continual excesses at table of persons in the middle and upper classes of society, an immense variety of noxious matters find their way into the portal blood that should never be present in it, and the mischief which this is calculated to produce is enhanced by indolent or sedentary habits. The consequence often is, that the liver becomes habitually gorged. The same, or even worse effects, result in the lower classes of our larger towns, from their inordinate consumption of gin and porter" (*op. cit.*, p. 66).

Treatment.—Congestions of the liver are mainly relieved by a restricted and judicious diet—abstinence from all rich dishes and fermented liquors. Active exercise in the open air must be taken till fatigue is produced sufficient to induce a desire to rest. Exercise by riding on horseback is especially beneficial.

With regard to medicine, Plummer's pill at bedtime, followed by a *seidlitz powder* or *Pullna water* in the morning, or by the usual black draught, or *compound mixture of senna*, or by such saline purgatives as *sulphate of magnesia* and *bitartrate of potash*, which cause a drain from the portal system of veins, usually give relief. But a purgative dose of *calomel*, especially combined with *compound jalap* powder, is of all combinations the most efficient in diminishing congestion of the liver. After its action the local weight, the pain, the fulness, and other symptoms subside concurrently, with copious bilious stools. In such cases it seems to act as Dr. Murchison suggests, by irritating the upper part of the small intestine, propelling onwards the bile as fast as it flows into the duodenum, thus preventing its re-absorption. The bilious accumulations thus evacuated constitute the "*bilious stools;*" and which are not to be regarded as the result of increased biliary secretion from the liver itself. An active aperient ought to be prescribed every second or third day for ten days or a fortnight after the action of the *calomel* purgation (Graves).

In chronic congestion the external use of *iodine ointment* sufficiently diluted is of service. Dr. Graves also bears testimony to the value of setons over the hepatic region. Two leeches every second day to the verge of the anus, and repeated from ten to fifteen times, also yield excellent results. *Nitro-muriatic acid*, as already mentioned, is also a valuable remedial agent.

Podophyline is highly commended by Dr. Gairdner as a relieving agent in the engorgement and torpor of the liver in those who have resided long in tropical climates. In such cases it may not act for ten, twelve, or even twenty hours, when he considers the purging which results is due to the large amount of bile passed into the bowels. In this respect, therefore, it seems to act by stimulating the liver to increased secretion

—a mode of action which, however, may be injurious in some cases of blood-congestion.

Taraxicum seems able to diminish abdominal plethora. Sir Ranald Martin prescribes it as follows:—

> *Ext. tarax.*, gr. xxxvi.; *Ext. Aloes*, gr. xii.; *Ext. acet. Colchici; Pulv. Ipecac. rad.* ā ā., gr. vi.; *misce. Divide in pil.* xii. Two pills every night.

An electuary, composed of equal parts of *sublimed sulphur* and the powdered gum resin of *guaiacum*, will be found a most useful remedy in cases where the congestion of the liver is associated with hæmorrhoids, and which is extremely common. When dropsy prevails, doses of the diuretic pill, of *hydrargyrum, squills,* and *digitalis* (a grain and a half of each ingredient), taken night and morning, are attended with benefit.

In bilious congestion copious draughts of hot water act as an emetic, diluting the bile, and if some saline purgative be added, the congestion of the liver will be relieved. If pain prevails over the liver, leeches and fomentations are indicated, to be followed by Plummer's pill at night and a black draught in the morning. The diet must be extremely limited and strictly regulated, as already noticed under blood-congestions of the liver.

In passive congestion, characterized by want of action of the liver, *hydrochlorate of ammonia* in doses of five to ten grains three times a day is of service.

CIRRHOSIS.

LATIN EQ., *Cirrhosis;* FRENCH EQ., *Cirrhose;* GERMAN EQ., *Cirrhosis;* ITALIAN EQ., *Cirrosi.*

Definition.—*Interstitial inflammation, with increase of the connective-tissue and its subsequent contraction, leading to other alterations and results.*

Pathology.—The chronic form of diffuse inflammation expresses itself by simple or granular induration of the substance of the liver, sometimes called *cirrhosis, interstitial hepatitis, hob-nailed* or *gin-drinker's liver.* An accurate knowledge of the nature of this induration was first obtained through the investigations of Kiernan, Hallmann, and Carswell. They showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhosis.

Rokitansky distinguishes two different modes of origin of the granular induration—the one proceeding from a morbid development of the capillary blood-vessels, owing to an excessive secretion of bile; the other due to a chronic inflammation of the hepatic parenchyma, tending to impermeability or obliteration of the finest ramifications of the portal vein. As a rule, the disease only comes under observation when it is more or less completely developed, and when consecutive disorders always associated with it draw attention to the state of the liver.

The commencement of cirrhosis is marked by increased consistence of the liver. The increased consistence is due to increased and increasing hypertrophy of the areolar frame-work, that scanty connective-tissue (almost granular in fineness when normal), which as the continuation of Glisson's capsule accompanies the hepatic vessels throughout the hepatic parenchyma. The inflammation is characterized by a true proliferation of this connective-tissue, without any tendency to free exudation, suppuration, or abscess. The nature of the cell-contents also affects the consistence of the organ. The gland is softer when the cells are loaded with fat, and firmer when they are infiltrated with *albuminoid* material, as when *cirrhosis* is associated with lardaceous disease. The amount of blood and of serous infiltration affects the consistence; but the induration of cirrhosis and the tenacity of the liver are mainly due to the increased development of the areolar framework, or connective-tissue, the increased and increasing amount of which displaces more and more the hepatic cells.

In the slighter forms the liver may be enlarged or of normal size, rarely smaller than natural; its surface covered by an opaque and thickened capsule, and exhibiting flattened projections, varying in size from a pin's head to a pea. Similar nodules are observed on section, separated by narrow strips of grey areolar tissue more or less vascular —the first beginnings of the cicatricial contraction which eventually strangulates the parts. Sometimes the colour is dark from bile-pigment; sometimes pale from fat.

In the advanced form the liver is reduced in size, particularly the left lobe, which not unfrequently is shrivelled up into a small membranous-like appendage, with a soft flabby rim of connective-tissue at the margins of the organ. Semi-globular knobs, more or less prominent, sometimes of a uniform, at other times of an unequal size and form, are thickly scattered over the surface.

In lardaceous disease combined with cirrhosis the liver exceeds its normal size.

The serous capsule is always thickened, and of a greyish-white colour, especially in the depressions between the granulations. Numerous bands of connective-tissue pass from it to neighbouring organs, such as the diaphragm, colon, stomach. On section, the organ has a cartilaginous hardness and coriaceous tenacity, showing at one place narrow, at another broad lines of connective-tissue surrounding the granulations, and sending streak-like processes into their interior. The granulations are generally dark or pale yellow. The elements of tissue are changed as follows:—

(*a.*) *Condition of the Hepatic Cells.*—In such livers a large portion of the hepatic cells are destroyed, and their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly formed connective-tissue. Another portion of the hepatic cells constitutes the substance of the granulations, and may remain for a long period intact; but ultimately they become filled with fat and various sorts of pigment. In nearly one-half of the cases fatty degenerations occur. The connective-tissue, compressing the commencements of the bile-ducts, causes a retention of the secretion and a jaundiced condition of the liver; hence the deposit of pigment, which accumulates in the form of fine orange or sulphur-yellow granules, more rarely diffused through the cavity of the cells. Another tint may occur from decomposition of the red matter of the blood, especially where branches of the hepatic veins are obliterated.

(*b.*) *The Connective-tissue.*—Its increased amount presents numerous varieties as regards its mode of distribution; and to it are due the differences in the size of the granulations.

The earliest increase is usually upon the finer subdivisions of the vessels in the interior of the liver, gradually involving the lobules. The bands of tissue sometimes surround single acini; at another time, three, five, or even seven *acini* may be enclosed. These bands increase at the expense of the *acini,* so that a little mass of brown pigment may be all that remains of the acini; and in this way large masses of connective-tissue take the place of gland-substance. The new connective-tissue varies as to elementary characters: at the circumference of the lobules it is fibrillated; in the substance of the lobules it is disposed to be amorphous; and in the thickened capsule it may be fibro-cartilaginous.

(*c.*) *The Vascular System* of the liver in cirrhosis undergoes great and important changes. The smaller divisions of the portal vein are in most cases narrowed by the shrivelling of the connective-tissue, or by partial impermeability of the finest ramifications of the portal vein from inflammation, obliteration, or compression. The vessels lose their round form, and become angular and bulging. Thrombi ought to be looked for in the branches when destruction of capillaries is obvious; and such destruction of the capillaries of the portal vein is in proportion to the disappearance of the glandular substance of the liver. So long as the hepatic cells exist, the peculiar mesh-like capillary network also exists; but where the cells disappear, their place is supplied by a connective-tissue, and entirely new capillary channels make their appearance, forming elongated meshes, communicating as well with the veins as with the hepatic artery (FRERICHS).

The trunk of the hepatic artery also becomes enlarged, and its capillary ramifications more extensive than in the healthy state, black pigment accumulating in its branches. A branched and tortuous network of vessels, of comparatively large calibre, appears in the connective-tissue, their peculiar and tortuous mode of distribution demonstrating their new formation.

The hepatic veins, as a rule, are unchanged; but their obliteration may take place by inflammation, from the capsule of the liver being propagated to the walls of the vessels, when the capillaries of the hepatic vein are gradually destroyed, and their communication with the portal capillaries is interrupted.

(*d.*) *The Bile-ducts.*—Their origin at the periphery of the lobule is destroyed by the pressure of the new connective-tissue; and there is apt to be catarrhal tumefaction of the mucous membrane of the larger branches.

These various changes may give rise to a long series of functional derangements, which in practice constitute the *symptoms of cirrhosis.* These are mainly—(1.) Derangements of the chylopoiëtic organs, from impediment to the passage of blood through the portal vein into the hepatic veins, and its stagnation in the radicles of the portal vein; (2.) Impairment, passing to complete suspension of the functions of the liver.

Causes.—The chief cause of cirrhotic induration is the abuse of spirituous liquors. In other words, it is due to the specific action of *alcohol* as an irritant or stimulant poison. The forms of alcoholic drink which seem most of all efficient as an irritant seem to be *gin* in this country and *schnapps* in Germany. Gin is known to be also greatly sophisticated, especially with spices, such as *capsicum*, which may also have an influence as an irritant. Niemeyer states the disease as more frequent in men than in women, and as rarely seen in childhood. Wunderlich relates the cases of two sisters, aged eleven and twelve years respectively, and in each of them the liver was in a condition of *cirrhosis.* On careful inquiry, it was discovered that both of them were great *schnapps* drinkers.

Alcohol, however, is not the only irritant capable of inducing the proliferation of cirrhotic or adhesive inflammation. Numerous products of faulty digestion, or of material such as spices taken into the stomach, may act as irritants of which we know as yet little. Other causes are *syphilis* and *malarious fevers*, especially frequent attacks of intermittent fever. The cirrhosis from syphilis is generally associated with the lardaceous disease about to be described.

Symptoms.—Derangements of the stomach, a loaded tongue, nausea, and occasionally vomiting and faint jaundice, are the earliest symptoms in the clinical history of this disease, which usually comes on very insidiously; and although they may abate, the degenerative process in the liver advances, and gradually undermines the constitution. Digestion continues feeble, and distension and tenderness of the epigastrium, along with heartburn, flatulence, and constipation, are developed. The patients lose flesh and strength, and their colour becomes pale, or dirty yellow, while the skin is dry and rough. The abdomen becomes distended and fluctuates, but the liver is found reduced, and the spleen increased in size; and increasing ascites or tympanitis induces more or less dyspnœa. Ascites is the most common and constant symptom of cirrhosis. Hæmorrhages from the stomach or intestines are apt to occur as the disease advances.

Enlargement of the spleen, to the extent of two or three times its natural size, is common in the later stages of cirrhosis (Oppolzer, Bamberger, Frerichs). Niemeyer considers this enlargement as due to the same causes as the enlargement of the liver, and not a secondary lesion to the liver affection. The stomachal and intestinal hæmorrhages often relieve this splenic enlargement. Enlargement of the superficial veins of the belly may supervene, is characteristic of cirrhosis, and demonstrates that the current of blood in the portal vein is greatly impeded. Slight febrile excitement only manifests itself towards the close, and in most cases diarrhœa terminates life by exhaustion. In other cases the fatal end is by pneumonia, acute pulmonary œdema, or peritonitis. Occasionally death occurs under symptoms of acholia; the patients become jaundiced, purpuric spots or ecchymosis are scattered over the skin; while delirium, convulsions, and deep coma close the scene.

Diagnosis.—"Slight sallowness of complexion," writes Dr. Budd, "a dull pain, or some degree of tenderness in the right hypochondrium, with occasional feverishness, in a person above the age of thirty, who has been long in the habit of drinking spirits to excess, are almost conclusive evidence of the existence of cirrhosis, even before there is any direct proof that the circulation through the liver is impeded" (*op. cit.*, p. 158). (See also *British Med. Jour.*, Nov. 11, 1871, for a case in a boy aged eighteen.) The urine is also saturated with much abnormal colouring matter and urates; and when the spleen is also found enlarged, the diagnosis of cirrhosis may be certain.

Prognosis is always unfavourable; and the main question regarding **Treatment** is the possibility of relieving the disorders of function which mainly threaten life, and so to delay the fatal termination. Absolute abstinence from spirits is indispensable, and the diet should consist of mild, simple articles of nourishment, especially easily digested animal food. Coffee, spices, and articles irritant to the liver must be avoided. Swelling and tenderness indicate *leeches* and *fomentations.* Mild saline laxatives may be given; and when the tenderness ceases, the bowels must be kept open by *rhubarb* and *salines.* When nausea or vomiting occurs, *hydrocyanic acid, belladonna, morphia,* or *extract of nux vomica* are particularly suitable. The gastric and intestinal catarrh require to be subdued by *alkaline carbonates.* They lessen the viscidity of the mucus secretion. When pain prevails, cupping or leeches are indicated over the liver. Saline purgatives, such as *sulphate of magnesia* or *bitartrate of potash,* should also be administered, while iced drinks and low diet must be the rule of life.

What has been said under the subject of simple enlargement or congestion of the liver may be referred to here as indicating a line of treatment under the circumstances there provided for. The saline laxatives are best taken as mineral waters, especially at Karlsbad or Marienbad. Springs containing small quantities of iron are also of service, such as Eger, Franzensbrunnen, Kissengen, and Homberg (NIEMEYER).

FATTY LIVER.

LATIN EQ., *Jecur adiposum;* FRENCH EQ., *Foie gras;* GERMAN EQ., *Fettleber*—SYN., *Fettige Entartung der Leber;* ITALIAN EQ., *Fegato grassoso.*

Definition.—(1.) *Infiltration or deposit of superfluous fat in the liver-cells, commencing at the circumference of the lobules, from the blood of the portal vein capillaries;* (2.) *Degeneration of the liver-cells, in which the nutritive process within the hepatic cells is disturbed, so that an abnormal increase of the fat naturally contained in the hepatic cells takes place, which remains there, so that they become engorged with oil.*

Pathology.—In both conditions the nuclei are thus obliterated or obscured; and the *morbid degeneration* thus takes place in a similar mode to the normal physiological infiltration; but the former tends to remain as a permanent lesion—the other is temporary, the deposit of fat being removed in the processes of nutrition, and conveyed into the general circulation; and the liver-cells remain clear, with their nuclei visible.

When the fat remains to such an extent as permanently to obscure the nucleus, the liver is then doughy, like œdema, and pits on pressure with the finger, retaining also the impressions of the ribs after death. On section, it leaves the blade of the knife covered with oil; and a little blood flows from the cut surface.

Extremely fatty livers may contain forty-three to forty-five *per cent.* of fat (FRERICHS, VAUQUELIN) in the form of *olein* and *margarin,* in variable proportions, with traces of cholesterin. The fatty infiltration, except in extreme cases, does not seem to impair the functions of the organ, nor interfere with the circulation of blood in it.

Symptoms.—The existence of a fatty liver is only to be inferred by a study of the surroundings and antecedents of the case. There are no positive subjective symptoms, and it is only in extreme cases that enlargement can be detected. The enlargement is painless and the surface is smooth, and its resistance so diminished that its edge is with great difficulty distinguished. Such conditions in fat persons, or in cases of *pulmonary phthisis,* are sufficient to cause fatty liver to be suspected only. Sometimes it seems to be the cause of diarrhœa in phthisical patients.

Causes.—Fatty liver is generally associated with morbidly fatty degeneration, and contains an abnormal quantity of fat. It accumulates within the cells first as minute particles surrounding the nucleus and clinging to it. These particles increase in size, coalesce, and form large drops of fat, distending the hepatic cells into large globules. The accumulation of fat and the degenerative process begin at the circumference of the lobules, and gradually advances inwards towards the region of the hepatic vein; so that the whole lobule becomes converted into a mass of fat. A slight amount of infiltration of fat does not alter the appearance nor the size of the liver. It can only be recognized microscopically. In extreme cases, which have passed on to degeneration, the liver is enlarged and flattened out, with thick and rounded edges, and the colour varies from yellowish-red to a distinct yellow, according to the relative amount of blood and fat. Its consistence is diminished, and it feels soft, with a large development of fat in other parts of the body—the supply of nourishment being in excess of its consumption or combustion. In cases of great emaciation, the fatty liver may obtain its fat from reabsorption of subcutaneous fat. A free indulgence of rich and fatty foods and alcoholic or malted drinks, with very little exercise, are the conditions which lead to the infiltration of fat. The cod-liver oil, so largely supplied in doses, which in many cases literally flow through the intestines unconsumed, no doubt contributes largely to produce the fatty livers so frequently seen in pulmonary phthisis.

Treatment.—Dr. Murchison recommends that large quantities of common salt be eaten with the food; while, if circumstances permit, the alkaline or saline mineral waters of Carlsbad, Marienbad, Kissengen, Ems, Vichy, Eger, Franzensbrunnen, or Homburg, are to be advised.

The diet must be carefully prescribed and regulated daily—as already indicated at p. 1004.

PIGMENTARY DEPOSITION OR INFILTRATION OF THE LIVER.*

Definition.—*A granular pigment found in the blood, partly free and partly enclosed in cells, due to the influence of malaria, and which, entering all the organs of the body by their capillaries, colours them more or less. Next to the spleen, the greatest amount of pigment is found in the liver and brain.*

Pathology.—*Pigmentary Degeneration of the Liver* is only to be recognized at a *post-mortem* examination, as the result of *melanœmia.* It is seen in cases of severe or pernicious remittent, intermittent, or malarious fevers. It is due to the accumulation of pigment-matter in the vascular apparatus of the gland, especially in the capillary network of the portal and hepatic veins; and the minute branches of the hepatic artery also contain quantities of black colouring matter. In cases where the liver is so affected, similar melanic matter is generally found in the spleen, kidneys, and brain; while the blood itself may be seen to contain dark granular masses, or nucleated pigment-cells, with black granules in their interior. The spleen seems to be the seat of formation of the melanotic matter (FRERICHS), whence it passes into the portal vein.

The effects upon the system of this degeneration are mainly due to the destruction of blood-corpuscles with which it is associated, and tending to a condition like that of *chlorosis.* The bile is usually secreted in large quantity. There is extensive capillary stagnation, which gives rise to obstruction of the circulation of the blood in the roots of the portal veins—and exhausting hæmorrhages of an intermittent kind are apt to occur from the gastro-intestinal mucous membrane. Profuse diarrhœa, vomiting, and serous effusions are also common occurrences. In India the lesion tends to *suppurative hepatitis.*

Treatment of Degenerations.—The main indications consists in the removal of the causes, or the cure of the disease which has induced the degeneration. Easily digested and nutritious food must be given; and, especially where chronic congestions prevail, a strict diet plays a powerful part in effecting a cure. Alcoholic drinks, sugar, and fat are to be avoided, as well as amylaceous substances. *Aloes, rhubarb, sulphate of soda* are useful to remove the torpidity of the bowels. *Muriate of ammonia* is recommended by Dr. Budd, in doses of from *five* to *ten* grains three times a day. He believes it probably relieves the liver, and does good by promoting the action of the skin and the kidneys.

LARDACEOUS LIVER—*Syn.*, AMYLOID DISEASE OF THE LIVER.

WAXY LIVER.

LATIN EQ., *Jecur lardaceum*—Idem valent, *Morbus jecinoris amylodes, Jecur cereum;* FRENCH EQ., *Fois lardacé*—Syn., *Maladie amyloïde du foie;*

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* Not recognized by the College of Physicians.

German Eq., *Speck leber*—Syn., *Speckige oder amyloide Entartung der Leber;* Italian Eq., *Fegato lardaceo*—Sin., *Malattia amiloidea del fegato.*

Definition.—*The existence of a peculiar homogeneous, translucent, albuminoid material in the hepatic arteries, cells, and texture of the liver, resembling an hepatic infiltration, but differing in this respect, that the albuminoid material of the disease is not found in the blood.*

Pathology.—Though frequently associated with fatty degeneration, and sometimes described as *scrofulous* liver, it has no necessary connection with either conditions, but is a substantive disease, affecting the blood-vessels and the glandular hepatic cells, not merely a degeneration. The liver is one of the most frequent seats of lardaceous disease.

The amyloid liver is usually pale or fawn-coloured—sometimes congested, so that it has a distinctly mottled appearance. The limits of the acini are unusually well defined, and the section or cut surface appears smooth, dry, bloodless, and firm. Its peritoneal covering is smooth, tense, and often opaque, and the liver substance as hard as a board. The tissue is extremely slow to decompose; its specific gravity is notably increased, in some instances above 1,080; but it may be reduced by fatty degeneration co-existing.

Microscopically the entire structure seems changed, but it does not appear in which tissue the change begins. Meckel says it begins probably in the cells; but Virchow has traced its origin very clearly to the blood-vessels. The peculiar structural formation of the liver has enabled the advance of the infiltration to be observed. His observations show that,—First the minute branches of the hepatic artery (intralobular vessels) are affected, and from these it gradually extends to the hepatic cells immediately adjacent; and when the disease has become established in an acinus, three zones, varying in colour and appearance, are observable,—(1.) The outermost is composed chiefly of portal vessels, and is generally the seat of more or less fatty degeneration;. (2.) The innermost zone is made up of the intralobular vessels (of which the vein is prominent in the centre when divided in section); these are surrounded by pigmentary deposit normal to the liver; (3.) The intermediate zone, between the external and the central parts, is the site of the albuminoid material, translucent and firm. After the blood-vessels have become affected, the secreting cells about them become involved. They acquire a homogeneous hyaline appearance, like particles of rough ice. They lose their nuclei. A cell-wall cannot be seen enclosing them; but they appear as an ill-defined, pellucid, glistening mass. As the cells become filled with the new material they gradually increase in size and lose their characteristic, irregular, polygonal shape, and cannot be separated from each other, so that several cells seem to have coalesced or grown together. The texture of the blood-vessels in which the disease commences becomes greatly thickened by the infiltration and translucid.

When the disease exists in an extreme degree, the greater is the translucency of the tissue, and the pellucid appearance gradually extends over the entire area of a lobule; while the few blood-vessels it seems to possess emit a little watery blood only from their diminished calibre. Rarely it occurs in insolated patches; generally it is distributed throughout, but more marked in some places. Towards the circumference of the liver, and shining through the clear peritoneal covering, the lobules or acini may be seen mapped out in a remarkably defined manner; in fact, in no disease of the liver is the appearance of lobules (which as a rule are not marked) so distinct as in this particular morbid condition (Wilks's *Guy's Hospital Report*, p. 123, 1856). This is due to the way in which the change begins and progresses. The fatty degeneration at the margins makes the acini still more clearly obvious; and the approximation of the fatty degeneration and albuminoid infiltration gives an opaque white appearance, which encircles the acini and maps them out in this perfectly defined manner. A section well washed will also show the dead-white opaque substance running in the course of the portal vessels, surrounding the more transparent substance of the acini, in the centre of which the hepatic vein is apparent.

By these characters the lesion must be distinguished from the simply fatty degeneration of the liver; for it is now certain that up till the year 1854 these two forms of disease have often been confounded. But in the appearances shown by the livers affected by these several diseases the differences are well marked. Louis evidently confounds the waxy and fatty liver in phthisis under the latter description; and the confusion has been transmitted from this great authority.

The large and hypertrophic livers, in their extreme degree, are for the most part due to the lardaceous disease, either simple or combined with the fatty degeneration; whereas the fatty degeneration is often present in the liver to an extreme degree without much or any hypertrophy. In the purely fatty liver, also, the specific gravity is not raised, but reduced. It is recorded as low as 1,005. When the fatty co-exists with the lardaceous disease, the specific gravity of the organ may be normal. Fatty liver consists of precisely the same elements as the normal liver in a similar structural arrangement, but an essential element is morbidly increased in its cells. The aggregate of solids and water may be slightly increased or diminished, but the *per centage* on the whole is reduced. Oil may be deposited to an enormous extent in the gland-cells; and these growing or swelling out, the volume of the organ, as well as its weight, may be increased to a very great extent.

A form of cirrhosis is due to the lardaceous liver, as well as to the fatty. In the former it assumes at once the *atrophic* form; and all the essential elements simultaneously disappear. The atrophy commences in the glandular epithelium or hepatic cells, and thus gives rise to an apparently relative hypertrophy of the fibrous tissue.

On the other hand, cirrhosis is a complex process, a lesion mainly due to actual growth, hypertrophy, and contraction of connective-tissue.

Characters of the Minute Tissue-elements in Lardaceous Liver—(1.) *Gland-cells.*—The finely granular contents gradually disappear, giving place to a homogeneous clear substance, which ultimately fills the cell. In a few hepatic cells the mark or trace of the nucleus may still be seen of a shining lustre; and, when completely altered, the cell resembles a brilliant pellucid homogeneous mass. The cells are excessively coherent; not easy to separate; and, indeed, so changed that a large solid aggregate mass is alone recognizable, in which neither cell, nor areolar matrix, nor blood-vessel is to be distinguished.

(2.) *Blood-vessels.*—The walls of the more delicate vessels become thickened, rigid, homogeneous, and lustrous. Their channel is narrowed, and ultimately entirely obliterated; while the still patent portions remain patulous on section. They appear as pellucid transparently colourless cylinders, in which no trace of the delicate structure of blood-vessels can be detected.

It is difficult to determine to which system the vessels so affected belong; but, so far, it is an ascertained fact that the minute ramifications of the hepatic artery are chiefly implicated in the first instance. Frerichs has "repeatedly observed diseased capillaries, the locality of which appeared to correspond to the situation of the portal and hepatic veins."

Solution of iodine, in the proportions already indicated, gives a deep red colour. The careful and slow addition of sulphuric acid changes the deep red to a dirty violet, and rarely to a blue tint. The tissue which remains exempt from the degeneration is distinguished by the greater amount of blood, and by greater softness and moisture.

The chemical character of the lardaceous liver has been already considered in the first volume; and it may simply be noticed here that Frerichs has especially examined into any relations which may subsist between the *glycogenic material* normally elaborated by the liver, and the *albuminoid substance* which is the result of the morbid process now under consideration. Both substances are *coloured* by iodine; but a chemical re-action takes place in the case of the *albuminoid substance*, and both are regarded as carbo-hydrogens. The results are mainly negative—sugar being furnished by the *glycogenic material*, when digested with saliva, or when brought in contact with albumen, but not by the *albuminoid substance*.

This form of disease has long been known as a liver affection; and Rokitansky first gave a clear account of its essential characters, and recognized the relation of the lesion to certain *cachexia*. It never occurs in persons otherwise healthy. Budd describes the affection as a "scrofulous enlargement," and Oppolzer as the "colloid liver."

The latest conclusions regarding the nature of lardaceous disease are those by Dr. W. Dickenson, already referred to. He considers the substance to be *fibrine* deprived of the alkali necessary to hold *fibrine* in solution. Thus deprived of its alkali, the fibrine "sets" in the tissue, and so thickens the walls of the smaller arteries with an apparent exaggeration of their transverse fibres. The material is transparent and homogeneous, and soon penetrates the coats of the minute vessels, and gradually works its way into the surrounding tissue.

In solid viscera the material remains about the blood-vessels, and fills the interstices of the structure. The liver particularly increases in size, becoming hard, grey, semi-transparent, as if intimately and uniformly infiltrated with white wax. The kidneys, supra-renal capsules, lymphatic glands, are all apt to assume the same firmness and waxy translucency. In the spleen the deposit exaggerates the Malpighian sacculi, until they look like grains of boiled sago. The lesion cannot be regarded, however, as a mere infiltration, seeing that the albuminoid matter, in the state in which it exists in the diseased organ, does not so exist in the blood.

Diagnosis.—Frerichs relates a case of waxy liver in a girl aged nine years, in whom ascites was present; and the fluid drawn off contained a large quantity of sugar. With the exception of cases of diabetes, sugar has never yet been found in ascitic fluid; and therefore its presence, it is suggested, may be diagnostic of waxy liver. Dr. Wilks found a trace of sugar in the fluid from the peritoneum of a similar case. There are also general and local grounds for suspecting the existence of lardaceous disease. The several symptoms may be stated as follow:—

1. When there is general ill-health, expressed by *marasmus*, *anæmia*, or *dropsy*, which constitute the primary symptoms in cases otherwise ambiguous, and which in some cases are associated with diarrhœa, vomiting, and cardiac systolic murmur.

2. In cases where (after examining the blood) such symptoms as are mentioned cannot be traced to lesions of such organs as we have hitherto been accustomed to refer these phenomena.

3. In cases where the constitution is enfeebled, and health is impaired by *ulcerations of bones*, *syphilis*, *malarious fever*, *tuberculosis*, *malaria*.

The local indications of lardaceous disease of the liver may be thus given from Frerichs's cases:—

(1.) Uniform enlargement of the organ; (2.) Increased consistence, indicated by firmness; (3.) Association of these characters with tumid spleen and albuminous urine; (4.) Association of these characters with any of the conditions enumerated in the general symptoms under (3.)

The prominent general symptoms of this fatal degeneration being "anæmia, prostration, exhaustion," the condition of the liver, spleen, and kidneys should be investigated in all such cases, and their condition recorded in all cases of *syphilis*, *caries*, *necrosis*, and *intermittent fever*. It is desirable to limit the suppuration of bones as much as possible by early surgical interference. Death usually occurs by exhaustion, unless a rapid end is brought about by *purulent peritonitis*, *dysentery*, *pneumonia*, *gangrene*, or *œdema of the lungs*.

Symptoms.—The gradual enlargement of lardaceous disease is not attended with pain. The dropsy is generally that of cachexia and hydræmia, and not from any obstruction to the portal circulation (BAMBERGER).

Treatment.—*Iodine* and *iron* are the remedies indicated by the nature of the disease and the circumstances under which it occurs; but nothing is known as to the effects of remedies.

JAUNDICE—*Syn.*, ICTERUS.

LATIN EQ., *Morbus regius;* FRENCH EQ., *Jaunisse;* GERMAN EQ., *Gelbsucht*—Syn., *Icterus;* ITALIAN EQ., *Itterizia.*

Definition.—*Certain morbid conditions in which many of the different* *tissues and fluids of the body are dyed yellow, but more especially the conjunctiva and the connective-tissue, from the colouring matter of the bile.*

Pathology.—Jaundice, though often a result of organic disease of the liver or duodenum, yet often occurs when those organs are perfectly healthy or simply congested. On posthumous examination, besides the yellowness of the cutis, the serum of the blood is generally found loaded with bile, and perfectly yellow. If the disease has continued for some time, the fat is also yellow, as well as the bones and cartilages, the serous fluids, and even the milk expressed from the breast of the female.

The theories that have been advanced to account for jaundice are, that the bile exists formed in the blood, and is merely removed by the liver, and that consequently jaundice is a consequence of the non-separation of the bile.

A more common opinion is, that bile is a secretion, and not a mere separation, and consequently that in jaundice the bile is first secreted and then absorbed both by the veins and lymphatics; while Portal has proved that it may be absorbed also by the lacteals.

The theory of jaundice especially advocated and expounded by Dr. Budd is, that the disease may arise in two ways,—(1.) By mechanical obstruction to the passage of the bile into the intestines, and the consequent re-absorption of the detained fluid into the blood; (2.) The suppression of the biliary secretion arising from some morbid condition of the liver itself, whereby biliary ingredients accumulate in the circulation. Some of these ingredients or constituents of bile are generated in the liver itself (*e.g.*, the bile acids); others exist pre-formed in the blood (*e.g.*, the green bile-pigment, or *biliverdine*, and the *cholesterine*).

The mechanism of jaundice has therefore been regarded from two points of view, namely,—(1.) Jaundice from suppression, retention, or non-elimination (BUDD, HARLEY); (2.) Jaundice from re-absorption of bile (FRERICHS).

The former of these forms of jaundice is characterized by the rapid accumulation of green bile-pigment in the blood, until the serum, and the tissues, and the urine are saturated with the pigment. Over this secretion the mental state seems to exercise a very remarkable influence—so much so that mental emotion, favouring congestion of the organ, also favours the stoppage of the secretion. Thus jaundice by suppression or non-elimination arises from (1.) Innervation (HARLEY). Active and passive congestion similarly induces jaundice; hence jaundice by suppression or non-elimination arises also from (2.) Disordered hepatic circulation (HARLEY). (3.) Another form of jaundice from suppression or non-elimination arises from loss or destruction of the secreting cells of the liver, as in acute and chronic atrophy, cancer, tubercle, fatty degeneration, and lardaceous disease. Hence jaundice by suppression or non-elimination arises also from the loss or absence of the glandular hepatic substance (HARLEY).

The second class of cases of jaundice arises from the re-absorption of the secreted but retained bile. They are also characterized by the accumulation of pigment in the blood; whence it stains the tissues, the urine, and the serum. The bile in these cases is absorbed from the distended ducts and gall-bladder; and the biliary products manufactured in the liver, equally with those formed in the blood, find their way back into the circulation, to be eliminated by the excretions. Hence the bile-acids (absent in the former class of cases of jaundice) are present in cases of jaundice from re-absorption, as well as the bile-pigment; and these acids are said to possess the property of dissolving the red blood corpuscles. Obstruction may be due to several causes, but chiefly to the two following, namely:—(1.) Congenital deficiency (very rare); (2.) Obstruction by disease, generally of parts in the vicinity of the head of the pancreas, or of the *ductus communis choledochus.*

Symptoms.—Jaundice, from the different intensities of the colour of the skin, has been divided into the *yellow,* the *green,* and the *black* jaundice. Jaundice may be sudden in its attack, or it may be preceded for a few days by great depression of spirits, lassitude, and somnolescence. It may also be preceded or accompanied by some slight pain in the region of the liver, but more commonly pain is not present.

The first symptom of jaundice is a yellowness of the white of the eyes, then of the roots of the nails. The yellowness next appears over the face and neck, and ultimately over the trunk and upper and lower extremities. As soon as the eyes are affected, the urine becomes of a deep red colour, and stains linen yellow, and if nitric acid be added it is changed to a deep green. The bile, however, is not always in the same state of combination in the urine, nor of the same quality; for in some instances, where the colour of the patient is most marked, and the urine of its deepest hue, the addition of nitric acid effects no change. At the same time that the urine is thus discoloured, the stools, often abundant in quantity, are copious and white. The pulse is slow, and the patient complains of a bitter taste in the mouth, has much thirst, an absolute inaptitude for all exertion, and suffers from a lowness of spirits amounting to hypochondriasis. In general the bowels are irritable and easily acted upon; but in a few cases they are constipated. If the patient recovers, the first symptom of recovery is the appearance of bile in the stools, after which the yellowness fades away in the inverse order of the attack.

The duration of this affection is such that in some cases it terminates in about ten days, but more generally it lasts from three to six weeks, and, if badly treated, oftentimes as many months.

Diagnosis.—This disease is to be distinguished from *chlorosis* and that sallow state which results from profuse uterine hæmorrhage. In these complaints the white of the eye is clear, the urine limpid, and the stools healthy, so that the great characteristics of jaundice are wanting.

The difficulty is to determine how far the jaundice is due either to obstruction or to non-elimination (suppressed secretion); and to Dr. Harley we are mainly indebted for methods of clinically distinguishing these two classes of cases by chemical examination of the excretions. His principle of diagnosis from this point of view assumes that in jaundice from suppression of bile, only those biliary products which exist pre-formed in the blood accumulate in the circulation; but that in jaundice from obstruction, the biliary products which are manufactured in the liver, equally with those pre-formed in the blood, find their way back to the blood, to be eliminated by the excretions.

The absence of bile from the stools is indicated by their pipe-clay colour, offensive odour, and presence of fat.

The urine of jaundice ranges from a saffron-yellow to a dark olive-green, or even black colour; and its amount of pigment is best judged of by the intensity of the colour of the uric acid crystals. But it is the presence of *biliary acids* in the urine which Dr. Harley considers characteristic of jaundice from re-absorption, as distinguished from jaundice arising from non-elimination or suppression.

The readiest mode by which the biliary acids may be detected is the following:—

> "To a couple of drachms of the suspected urine add a small fragment of loaf sugar, and afterwards pour slowly into the test-tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes—a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts. On the other hand, the absence of this phenomenon, and the occurrence of merely a *brown* instead of a *purple* tint, although in the earlier stages of jaundice equally indicative of suppression, is no indication of the cause of the suppression, which must be gleaned from other circumstances" (Harley *On Jaundice*, p. 61).

Dr. Felix Hoppe's method is as follows:—

> "(1.) Decompose the icteric urine to be examined with an excess of milk of lime; (2.) Boil for about half an hour; (3.) Filter; (4.) Evaporate the filtered fluid nearly to dryness; (5.) Decompose with a great excess of concentrated hydrochloric acid, and then keep the whole (before being again filtered) at the boiling-point for half an hour; (6.) To avoid spurting of the fluid it is necessary to renew the volatilized hydrochloric acid from time to time; (7.) Leave the liquid to get completely cold, and then add six to eight times its volume of water; (8.) Filter the dark-brown turbid solution thus obtained, wash out with water the residue on the filter, until the same runs through quite colourless; (9.) Dissolve the brown resinous mass on the filter in 90 *per cent.* alcohol; (10.) Decolorize by boiling with animal charcoal, filter, and evaporate to dryness in the water-bath; the residue is a yellow, resinous mass, which, if bile-acids be present, must consist for the most part of pure *choloïdic acid.* In such a case it melts by warming, and emits the peculiar musk or soap odour. (11.) Lastly, dissolve in a very little caustic soda and some drops of warm water, add a very small piece of sugar, and allow three drops of concentrated SO_3 slowly to fall into it. At first the fluid becomes milky and troubled, and resinous flakes separate, which stick pertinaciously to the glass; but afterwards, by the addition of more SO_3, these again dissolve and produce a beautiful purple-red or dark-violet fluid" ("Abstract of Kühne's Paper on the Pathology of *Icterus*," by Dr. George Scott, of Southampton, in Beale's *Archives*, vol. i., p. 343).

But these acids after a time gradually diminish and disappear from the urine in cases of obstruction and re-absorption—namely, when the secreting powers of the liver become impaired, when the hepatic gland-cells waste away; and then the abnormal products *leucine* and *tyrosine* appear in the urine. They are readily detected by the slow evaporation of an ounce of urine to the consistence of syrup, which is to be put aside to crystallize. *Tyrosine* is recognized by the fine stellate groups of needle-like crystals or spiculated cells, which appear like a rolled-up hedgehog with its bristles sticking out in all directions (HARLEY). It may be obtained in its pure state by (1.) Adding a solution of *acetate of lead* to four ounces of urine, till a precipitate ceases to form; (2.) Filtering; (3.) Freeing the liquid from an excess of lead by a current of sulphureted hydrogen; (4.) Again filtering; and (5.) Evaporating the clear solution. The tyrosine is now colourless, and crystallizes with the microscopic characters better marked. *Leucine* is known by its flat, circular, oily-like discs, without crystalline structure. It is soluble in water and boiling alcohol, but insoluble in ether.

If sugar appears in the urine in cases of jaundice, with a diminution of bile-acids hitherto in quantity, it is generally the forerunner of a fatal termination (HARLEY).

Treatment.—As a general principle, the larger number of cases of jaundice from functional disorder (perhaps four out of five) will get well in time spontaneously, but may be aided by remedies judiciously selected according to the diagnosis already indicated. The first indication is to aim at removing the exciting cause; and in jaundice due to congestion of the liver, purgatives seem to act beneficially in the form of *blue pill* or *Plummer's pill*, with *aloes*, and *nux vomica* with *rhubarb pill mass.* In cases of acute jaundice—from suppression of the biliary secretion consequent on a powerful nervous shock or mental perturbation—Dr. Anstie has seen two or three doses of the *hydrochlorate of ammonia*, to the extent of gr. xx. every four hours, produce a restoration of the biliary secretion. He regards it as a most powerful restorative of the biliary functions. Acids and alkalies are alike contra-indicated in cases of jaundice resulting from active congestion of the liver. *Benzoin acid* has been recommended; and Dr. Harley has found it useful in cases of suppression of bile (by innervation, for example); but in cases of jaundice from obstruction it is injurious. *Podophyllin* is also of use in jaundice from suppressed secretion of bile. It ought to be combined with *hyoscyamus.* It is especially useful in cases of feeble liver, combined with vegetable tonics, such as *gentian* and *quinine;* but it ought not to be given in cases of jaundice from obstruction. The medicine of all others which has seemed most generally useful in Dr. Budd's experience is *sulphate of magnesia,* in half drachm to drachm doses, combined with fifteen grains of *carbonate of magnesia,* and half a drachm of *aromatic spirits of ammonia,* given three times a day an hour before food. The *sulphate of magnesia* maintains a free action of the bowels; and the *carbonate of magnesia* neutralizes any excess of acid in the stomach or bowels (*op. cit.,* p. 289).

SECTION XIII.—DISEASES OF THE HEPATIC DUCTS AND GALL BLADDER.

INFLAMMATION OF THE HEPATIC DUCTS AND GALL BLADDER.

LATIN EQ., *Inflamatio;* FRENCH EQ., *Inflammation;* GERMAN EQ., *Entzündung;* ITALIAN EQ., *Inflammazione.*

Definition.—*Inflammation, generally catarrhal, of the mucous membrane of the biliary passages.*

Pathology.—The forms of inflammation are either *catarrhal* or *diphtheritic.* The former is common, the latter is rare.

The *catarrhal* form is recognized by the excessive flow of secretion from the biliary passages; the *diphtheritic* form by a fibrinous exudation which may occur during such severe diseases as *typhus, septicæmia,* or *cholera;* but it is not to be recognized during life.

The catarrhal inflammation is generally an extension of gastric and duodenal catarrh extending through the opening of the *ductus communis choledochus.*

Symptoms.—There is usually excessive hyperæmia of the liver causing simple enlargement, with symptoms of obstruction to the flow of bile and of jaundice. The tongue is coated, and there is a bad taste in the mouth, eructation from the stomach, and dyspepsia. The fæces are free of colour, and of a whitish dirty clayey aspect.

Treatment is similar to that of catarrh of the intestines, and especially in the use of nitro-muriatic acid internally and as baths.

GALL-STONES.

LATIN EQ., *Calculi fellei;* FRENCH EQ., *Calculs biliaires;* GERMAN EQ., *Gallensteine;* ITALIAN EQ., *Calcoli biliari.*

Definition.—*Concretions of certain biliary constituents, accumulating generally in the gall bladder as gall-stones, the passage of which through the duct into the duodenum gives rise to symptoms known as "gall-stone colic."*

Pathology.—Bile in its healthy state is of a deep yellow colour, extremely bitter, followed by a sweetish after-taste, a little heavier than water (sp. gr. 1,018), and miscible in that fluid in every proportion. In the gall-bladder bile is usually of the consistency of thin molasses, having a specific gravity of 1,026 to 1,032—cystic bile. It contains a little free soda, and is feebly alkaline or neutral.

The daily amount secreted by a man is about 56 oz. (BIDDER and SCHMIDT). The proportion of solid matter it contains is from 9 to 17 *per cent.;* the cystic bile about 10 *per cent.;* hepatic bile, 3 to 5 *per cent.,* composed of substances peculiar to bile; among which may be distinguished two resinous acids,—namely, the *Glycocholic* or *cholic acid* in small quantity; and the *Tauro cholic* or *choleic acid* (*bilin*), in which 25 *per cent.* of *sulphur* of the bile exists. They are united in the bile with soda as a base. The following Table shows the results of six analyses:—

ANALYSES OF BILE FROM HUMAN GALL-BLADDER.

IN 1000 PARTS.	FRERICHS.		VON GORUP—BESANEZ.*			
	1. Man aged 18, killed by a fall.	2. Man aged 22, killed by injury.	1. Man, aged 49, Criminal, Beheaded.	2. Woman, aged 29, Criminal Beheaded.	3. Man, aged 68, killed by a fall,	4. Boy, aged 12, killed by injury.
Water,	860·0	859·2	822·7	898·1	908·7	828·1
Solid Residue,	140·0	140·8	177·3	101·9	91·3	171·9
Biliary Acids in combination with Alkalies,	72·2	91·4	107·9	56·5	73·7	
Fat,	3·2	9·2	47·3	30·9		148·0
Cholesterin,	1·6	2·6				
Mucus and Colouring Matter,	26·6	29·8	22·1	14·5	17·6	23·9
Salts,	6·5	7·7	10·8	6·3	...	...

* *Phys. Chem.*, 1862, p. 469, quoted by Dr. Carpenter.

The bile is liable to undergo many morbid changes. It is found green or yellow, and those colours may be pale or intense; or it may be as fluid as water, or as viscid as tar. Its taste is also greatly affected, being sometimes bland, and at others so acrid as to excoriate the lip. These different states do not denote different states of the liver; for the same condition of bile is found in the most oppositely diseased states of that organ.

The most remarkable, however, of the states of diseased bile is that in which it concretes into a gall-stone. Such concretions were known to the ancients, but the chemical composition of these calculi has been more recently determined by Fourcroy, Poultier, Powel, Chevreul, and others.

Biliary calculi are often found filling the gall-bladder, the structure of the liver and gall-bladder being perfectly healthy, even when the gall-bladder contains numerous calculi with sharp angles and edges.

Although their principal seat is the gall-bladder, they have been found "*in transitu*" in the *cystic duct* and in the *ductus communis.* Occasionally they have been found in the *hepatic ducts,* where they are apt to excite *suppurative hepatitis.* Cruveilhier has given one instance in his very splendid work on pathological anatomy. Lastly, they are sometimes found in the intestinal canal, after having passed from the gall-bladder into that cavity.

Although the liver is frequently found healthy when the gall-bladder contains calculi, yet more commonly its structure is more or less diseased.

In some instances the *ductus cysticus* is obliterated, so that *hydrops vesicæ felleæ* may result with more or less excessive biliary congestion. In others the gall-bladder is thickened or ulcerated, particularly at the fundus, where considerable congestion of blood and puffing of the mucous membrane may exist, short of ulceration. When ulceration once commences it may lead to perforation of the gall-bladder, opening directly into the peritoneum or into the cavity of the intestinal canal, and limited by adhesive inflammation. In some cases the walls of the bladder are thickened greatly, and, becoming contracted, the cavity diminishes and its contents become dried up into a chalky mass, in which the previously existing biliary calculi are imbedded. If the body be examined shortly after a large gall-stone has passed into the intestine, the *ductus communis*, so small in health that it is difficult to find it, may then be so enlarged as to admit the finger. In some very rare instances the extremity of this latter duct has been found obliterated from inflammation, in consequence of the irritation to which it has been directly or indirectly subjected.

Modern chemists have determined that gall-stones are composed principally of two substances, *cholesterine* and *colouring matter*, in various proportions, together with some animal matter, the usual bile salts, and perhaps a trace of iron. Bile pigment, with choloidinic acid and its calcareous base, also occasionally accumulates in solid masses. *Cholesterine*, which sometimes exists in the large proportion of 88 to 94 *per cent.* of the whole calculus, is soluble in *boiling alcohol, æther*, and in *nitric acid.* It is tasteless, inodorous, and burns by the flame of a lamp till it is altogether consumed. It is also lighter than water, and insoluble in that fluid. The colouring matter, also, which is generally combined with the cholesterine, and often forms of itself a large portion of the gall-stone, is inodorous, insipid, and heavier than water. It is likewise insoluble in that fluid, in alcohol, or in acids, but is soluble in alkalies, whence it is precipitated, on the addition of water, of a brownish-green colour.

The calculi found in the human gall-bladder have been divided by Dr. Powel into crystallized, deposited, amorphous, and porcupine-like calculi.

The *crystallized* concretions, when fractured, look like spermaceti, and the crystals, like those of that substance, are easily broken into a sort of greasy powder. They are in general semi-transparent, but seldom retain their purity throughout, being, near their circumference, coated or mixed with more or less of a brown colouring matter. At the central point of these colourless crystals, to which the radii converge, there is mostly a small particle of coloured matter, resembling dried bile, and which has served apparently as the nucleus of crystallization. Sometimes this crystallized shoot, having reached perhaps the size of a pea, becomes itself a centre around which many depositions are afterwards made of variously confused and irregular strata, and the surface of the strata may in their turn become the nucleus of a fresh crystallization.

The *deposited* gall-stone is a deposition of biliary matter in laminæ, like the arrangement of an onion or of an urinary calculus.

The *porcupine-like* calculi are small round calculi, having a number of projecting points, and hence termed porcupine calculi. They are generally small, and their structure has not been determined.

The amorphous concretions are such as bear no mark of crystallization, or of any very regular structure, but sometimes as they dry they break into layers, showing their mode of formation.

Biliary calculi vary considerably in their specific gravity; and this does not appear to depend on any peculiarity of structure; for, two of the purest specimens being selected, one may swim while the other will sink in water—a difference perhaps owing to the greater or less quantity of animal matter they may contain.

These calculi vary greatly in number; from 1 to 1,000 have been found in the gall-bladder. When single they are usually of a round or oval figure. In size they vary from a pin's head to that of a nutmeg or a walnut, or they may be even as large as a hen's egg. When extremely numerous, they are usually small, of a dark brown colour, and occasionally slightly agglutinated with viscid bile. When, however, the number is small (from two or three to eight), the size often is considerable, and in this case the gall-stone is often made up of several, loosely adapted or fitted to each other by facets, showing they must once have existed in a soft state.

With respect to the formation of gall-stones, the cholesterine, being a constituent of healthy human bile, only in a very small proportion, is evidently produced in excess, and is secreted in a fluid state. From this, if a nucleus of any kind, as a piece of thick mucus, be present, crystals may immediately shoot or form upon it; and thus a person apparently in good health may have a large gall-stone formed in his gall-bladder. Dr. Powel thinks he has met with cholesterine in a fluid state in the gall-bladder of a patient he examined. The peculiarity of this bile was its remarkably deep and almost black colour, whence he was led to treat it with alcohol, and in this manner he obtained solid cholesterine. The deposited gall-stone must be formed by an excess of colouring matter, or else by some morbid state of the bile, in which that principle is readily separable when any nucleus is present.

Causes.—The remote causes of gall-stones are supposed to be too full an animal diet, combined with a sedentary life, or the indulgence of anger or of those other passions which suppress the flow of bile, and perhaps alter its qualities; also those states of indigestion which re-act on the liver. This affection, however, is not necessarily connected with ill health, for calculi have been found in the gall-bladder of persons who have died accidentally, and apparently in the best health.

Gall-stones appear to be peculiar to adults; and generally occur after twenty, but perhaps most commonly between forty and sixty. They affect women more frequently than men, and persons of sedentary rather than those of active habits of life.

They are particularly frequent in patients with carcinoma of the stomach and liver (NIEMEYER).

Symptoms.—The formation of gall-stones is unattended with pain, and stones once formed often lie latent for a considerable time in the gall-bladder, without causing any trouble to the patient. At length, however, some cause may force a stone into the *cystic duct*, when a series of very formidable symptoms arise, and which continue till the calculus has passed into the duodenum. But occasionally calculi of small size may pass through the *ductus cysticus* and *communis choledoctus* without exciting pain or any other symptom. The experiences at such watering-places as Karlsbad, where the stools are, as a rule, carefully examined for gall-stones, afford numerous examples of this (NIEMEYER).

The more severe and painful symptoms which attend the passage of gall-stones from the gall-bladder into the duodenum have received the name of *gall-stone colic.*

The attack is generally sudden, the patient being seized with shivering, accompanied by violent and acute piercing, griping, insupportable pain at the pit of the stomach, or rather at the point corresponding to the opening of the duct into the duodenum, and from this point it spreads over the whole abdomen to the right side of the thorax and right shoulder, and darts through the back. This pain occurs in paroxysms varying from a few minutes to a few hours, when it intermits and after a short interval returns, and this continues till the gall-stone has passed into the intestine. The pain is so great that patients most tolerant of pain are made to moan and double themselves up, and to roll about on the bed; but generally they select the floor. The patient during this trying period suffers from nausea or vomiting so severe that everything is rejected, and the matters thrown up often contain bile and small biliary calculi, which may perhaps more properly be called biliary sediment.

Excessive pain and vomiting are the leading features of the passage of a gall-stone; and it is impossible for those who have witnessed a case of this kind not to be struck with the resemblance many of its symptoms bear to those of parturition—a comparison women frequently make when describing their sufferings. There is this difference, however, that when the pain intermits there is a deep-seated soreness and fulness of the right hypochondrium and epigastric regions. Like parturition, then, one attack of pain succeeds another, till at length this more urgent symptom ceases, and the calculus may be inferred to have passed into the intestine. After that has taken place, the soreness and uneasiness gradually cease, and the patient is restored to health. In some cases, and at some early period of the attack, jaundice makes its appearance, and may continue for a considerable time after the calculus has passed.

The pulse during the paroxysm is for the most part natural, unless the patient is exhausted by long continuance of pain, when it becomes small, the skin cold, and the face pale and distorted. The patient may sometimes faint, and prolonged faintness is a rare cause of death from gall-stone colic. The heat of the body also is not increased. The dejections, according as the obstruction is more or less complete, are clay-coloured or natural; and, on a close examination from time to time, are ultimately found to contain the offending calculus.

The duration of the attack is various—sometimes only a few hours, sometimes a few days, while sometimes several weeks elapse before the gall-stone is expelled.

It has been imagined that the degree of angularity of these concretions must considerably influence the symptoms; this, however, is not the case, for their edges are never sharp enough to cut nor their points to pierce. Size is of more importance than shape; and in proportion to its magnitude so will be the opposition to its passage. The transit of one concretion, by distending the duct, necessarily facilitates the passage of a second.

The symptoms which have been described are the most usual, but sometimes they are exceedingly anomalous. In one case, a lady was seized with violent pain in the left shoulder, simulating rheumatism. She then fell into a state of somnolescence so complete that even on the night-stool she slept and was obliged to be held. This state lasted for a fortnight, when she was seized with violent pains in the right hypogastrium, and, after some days, passed a gall-stone as large as an olive.

As a general rule the symptoms cease on the gall-stone passing into the duodenum; but sometimes the calculus is so large as to give rise to severe disorder of the intestinal canal. "A lady," writes Dr. Robert Williams, "was attacked with symptoms of ileus, which gave rise to a suspicion of hernia, and an operation was about to be performed, when the patient most unexpectedly passed a stool in bed. On examining the matters passed, a biliary calculus was discovered, $1\frac{6}{10}$ of an inch in length, and $1\frac{1}{10}$ of an inch in diameter; it weighed 228 grains. The lady recovered." Gall-stone colic is particularly liable to occur during digestion.

Diagnosis.—The passage of the gall-stone is to be distinguished from hepatitis by the pains being in general of great intensity, and paroxysmal, and also by the pulse continuing natural.

Prognosis.—The prognosis is always favourable, unless the calculus be of such magnitude as to render its passage almost impossible, or unless it be connected with organic disease of the liver.

Treatment.—When the symptoms of gall-stone passing the duct are present, the curative indications are to facilitate its passage into the intestine, to relieve the intense pain which accompanies it, and to prevent that inflammation which the presence of an extraneous body of any magnitude is calculated to produce in the duct.

The first thing to be done is to calm the sufferings of the patient; and half a grain of solid *opium*, or a quarter of a grain of *morphine*, or twelve drops of *laudanum*, or the *mistura camphoræ*, ℥ xj. conf. opii ʒ ss. to ℈ ij. c. sp. æth. nitr. ℥ j., should be given every hour, or every two hours, till some relief is obtained, or till there is slight narcotism; and then similar doses may be repeated every four or six hours, till the pain has ceased. If the vomiting be severe, and the above medicines be rejected, the opiate may be given *hypodermically*, commencing with not more than $\frac{1}{4}$th of a grain of the *muriate of morphia* in solution; or, opium may be administered by enemata of *laudanum*.

Careful inhalation of chloroform, till anæsthesia is produced, also gives great relief, and is a valuable remedy when paroxysms are most violent: it not only calms pain, but may lead to the cessation of the spasmodic contraction of the biliary passages, and thus favour the expulsion of the calculus (MURCHISON). Chloroform also, when given internally, appears to exercise some influence as a solvent of biliary concretions. Dr. J. Barclay relates an instance in which ♏ ii.–♏ iii. of *chloroform*, given three or four times a day, afforded great and immediate relief (*Brit. Med. Journal*, 1870). Dr. Murchison has seen marked benefit from the *extract of belladonna*, given in half grain doses every two or three hours. The *hydrate of chloral*, as it produces extreme muscular relaxation, is also worthy of a trial.

Small pieces of ice in the mouth are most efficacious in relieving vomiting. Emetics or laxatives are not to be given during the attack.

Besides these medicines, a warm bath should be immediately prepared, and the temperature should be as high as 100° to 110°, or indeed as hot as the patient can well bear it, and the immersion should continue till he is in some degree exhausted. The intention of the bath is to relax by means of heat the muscular fibre of the ducts, and thus relieve the pain and facilitate the passage of the gall-stone; and the effect is always so agreeable to the feelings of the patient that, on the recurrence of the pain, he constantly asks for a repetition of it; and his wishes should be complied with. If a warm bath cannot be procured, fomentations, or a large linseed poultice, should be applied over the abdomen. Dry heat is always at hand, and hot flannels, hot sand, or hot camomile flowers afford some relief.

Bleeding during the passage of a gall-stone is injurious; for, by debilitating the muscular fibre, it is rendered more irritable, and consequently its contraction is irregular, morbid, and prolonged. If local pain be great, and apprehension be entertained that the duct may have inflamed in consequence of the irritation it has suffered, a few leeches to the side, or a few ounces of blood taken by cupping, are admissible; but this practice is rarely necessary.

The calculus having passed, and the patient being relieved, the secretions of the liver should, if possible, be rendered more healthy; and a short course of neutral salts, or of the Cheltenham or Leamington waters ought to be taken in this country. Niemeyer writes that, under the use of the Karlsbad waters, immense quantities of gall-stones are evacuated with proportionately little difficulty. The same is true of other *alkaline* mineral waters, such as Vichy, Marienbad, and Kissengen.

The radical treatment of biliary calculi ought to be attempted during the intervals between the attacks of gall-stone colic. Lutten, on this principle, describes the treatment of biliary calculi under two heads:—

(1.) *Solvents believed to act on the calculi.* These are,—

(*a.*) *Alkaline solvents*, which are to be preferred above all others. Under their influence the calculi seem to be really dissolved or broken up, so as to disappear without leaving any trace; but more usually they are expelled entire in abundant bilious evacuations. This alkaline treatment comprises various medicines, such as the *fixed alkalies, soap-lye, salts of soda, carbonate of ammonia;* but especially he recommends the waters of Vichy, Vals, Karlsbad, and Ems. These waters are used as drinks and as baths; and they must be employed perseveringly, at different periods, during several years in succession.

(*b.*) *Durande's remedy*, proposed by himself in 1790, which consists in the administration of half a drachm to a drachm every day, in the morning, of a mixture composed of *fifteen grammes* (*i. e.*, about ʒ iii.) of *sulphuric æther*, and *ten grammes* (*i. e.*, about ʒ ii.) of *oil of turpentine*, has enjoyed a reputation as a solvent of biliary calculi ever since that time. It has been extensively used and greatly recommended by many Continental physicians, especially by Richer, Soemmering, and Martin-Solon; and in this country Copland speaks favourably of it; but whatever virtue the remedy may possess as a solvent is probably due more to the *ether* than the *turpentine*, the former being a ready solvent of cholesterine (WARING). The dose is to be gradually increased till about a pound of this mixture has been taken. Its mode of action is not known; but where it has succeeded, the calculus has been expelled without being dissolved. It seems in some way efficient in promoting the expulsion of calculi, rather than acting as a solvent. Recently, says Niemeyer, various substitutes have been proposed in place of Durande's remedy, and also variations from the original dose. One is also a popular remedy—a mixture of *oil of turpentine*, ℈ ii., with ʒ i. of spirits of *sulphuric ether*—which has been prescribed in *drop doses* by RADEMACHER.

(*c.*) *Diet* has a great influence upon the quality of the bile. Fresh laxative vegetable food, grapes, fruits, and whey ought to constitute the staple articles of a regulated diet. All fat should be as much as possible excluded from food, and the plainest diet, in the form of roasted meats or boiled meats, with vegetables and farinaceous food, should be rigidly proportioned to the wants of the system. The drinks should be water or lemonade, and an avoidance of alcoholic or malted stimulants.

(2.) *Treatment which seeks to assuage pain.*—With this end in view Lutten prescribes *opium* in larger doses than those already mentioned —namely, 2½ to 3 grains of solid opium at once; but he considers the hypodermic injection of *solution of morphia* as preferable (*Nouveau Dictionnaire de Médicine et de Chirurgie Practiques;* also, *Society Med. de Paris*, 1866; and *Sydenham Society's Biennial Retrospect* for 1865–66).

Dr. Waring considers that the intense agony is more effectually relieved by these large doses of opium than by any other remedy, particularly if it be combined with the use of the hot bath. He also prescribes two grains of *solid opium*, or ♏ xl. of the *tincture of opium*, either by mouth or in the form of enema, to be repeated in half an hour if the pain is not relieved. It may also be advantageously combined with a full dose of *ether* or *chloroform*.

Dr. Thudichum regards opium in such cases rather as an auxiliary than as a remedy to be relied on alone; and he speaks strongly of the danger of over-dosing such patients with opiates (Ranking's *Abstract*, 1863, xxxvii., quoted by Waring).

Large draughts of hot water, containing *carbonate of soda* in solution (ʒ i.–ʒ ii. to twenty ounces of water) gives more immediate relief than any other means, according to the experience of Dr. Prout. The first dose or two may be rejected; but the addition of a few drops of laudanum and steady perseverance in the remedy will eventually ensure its success (MURCHISON).

SECTION XIV.—DISEASES OF THE PERITONEUM.

PERITONITIS.

LATIN EQ., *Peritonitis;* FRENCH EQ., *Péritonite;* GERMAN EQ., *Bauchfellentzündung;* ITALIAN EQ., *Peritonitide.*

Definition.—*An inflammation of the serous membrane lining the cavity of the abdomen, and covering the viscera contained in that cavity.*

Pathology.—The peritoneum, like the pericardium and pleura (*q. v.* p. 548–852), is liable to inflammatory processes, either acute or chronic, in their progress.

Acute inflammation of the peritoneum (as of all serous membranes) begins in the connective fibrous tissue, which becomes red and injected, with proliferation of its elements; and at length the same phenomena pervade the serous membrane itself, when free exudation occurs, either as a fibrinous material or as a serous fluid, with or without pus-corpuscles.

Its colour when inflamed is a bright arterial scarlet hue—being first dotted with a number of small red points, which become confluent, and form streaks and patches, which in their turn coalesce; or a small central nucleus of inflammation may form and spread till the whole extent of the peritoneum is one entire bright red colour. In addition to the redness, some interstitial growth or exudation accompanies diffuse inflammation of the peritoneum, so that the membrane loses its transparency and is thickened. When redness does not exist, opacity is often the only evidence of the previous existence of the inflammatory state. The consistence also of the subperitoneal tissue is greatly impaired, and rendered easily lacerable, so that the peritoneum is capable of being detached in considerable portions. This inflammation may terminate by resolution, or it may advance to the effusion of serum. The quantity may be trifling, not exceeding a few ounces, but occasionally it is large, fills the cavity of the abdomen, and constitutes inflammatory dropsy.

The exudation may be of the fibrinous type, when coagulation of the effused fluids tends to occur, and the opposed surfaces to be glued together. In some cases, however, the fluid predominates, and the fibrinous coagulated masses are loose, so as to float unattached in the serum; or, of such consistence as to unite opposite parts together, and of such extent as sometimes to form an adventitious membrane, covering the entire surface of the abdominal walls as well as the whole of the intestines. The period at which organization of the lymph thus effused may begin, Mr. Hunter determined to be about twenty-four hours. If the disease proceeds, pus forms, sometimes not to a greater amount than a few ounces; but in other cases it amounts to many pints, or even fills the whole of the abdominal cavity. Ulceration of the peritoneum is not frequent, and generally takes place from without inwards, as from a perforating ulcer of the small or large intestines, or from the rupturing of an abscess or other tumor. The different acute inflammations described have been mentioned as though succeeding each other; but in many instances all these different forms co-exist in different parts of the peritoneum at the same time, and perhaps have been irregularly set up.

Experience has also shown that although the structure of the peritoneum appears to be uniformly the same, yet certain parts of it are more liable to inflammation than others, as the convex surface of the liver or spleen, the right iliac fossa, the surface of the small intestine, and in females the broad ligaments, the Fallopian tubes, and the parts immediately adjoinining them, as also the space covering the rectum and bladder. The parts the most rarely affected are those covering the stomach, bladder, omentum, and the mesentery. It will be seen that the liability of different parts of the peritoneum to inflammation is in proportion to the liability of the organs they cover to become diseased, and that these partial inflammations are for the most part the result of contiguous irritation. The tendency to peritonitis also is much greater in persons affected with *scrofula*, *morbus Brightii*, and other exhausting diseases, as well as in women at the menstrual periods, than in healthy persons.

The forms of peritonitis to be distinguished are,—(a.) *Metro-peritonitis*, or *puerperal peritonitis;* (b.) *Chronic peritonitis;* (c.) *Suppurative peritonitis;* (d.) *Tubercular peritonitis;* (e.) *Adhesive peritonitis;* to which may be added (f.) *Encysted inflammatory products* and local adhesions of the parts opposed to each other. These forms indicate at once a local source of irritation as the immediate cause of the peritonitis.

Symptoms.—Peritonitis may be acute or chronic, partial or general. It is occasionally ushered in by some previous shivering and fever, but in many cases there are no preliminary symptoms. The symptoms of peritonitis from perforation of the intestine or stomach are characterized by the *suddenness* and *intensity* of pain, often referred to a particular region of the abdomen; but the whole abdomen soon becomes painful to pressure. The pain is constant, exquisite, and leads to such lowering of the heart's action that death rapidly follows by *asthenia*, preceded by the most marked symptoms of collapse.

If acute peritonitis should not terminate by resolution, but by effusion of serum or of lymph, the patient complains of a severe pain in the abdomen, which is increased on pressure. He lies on his back, fearing to move. His pulse is from 90 to 120, and peculiar, as an inflammatory pulse. In proportion as it is frequent, so is it smaller. The tongue is coated, and the bowels constipated or regular. If serum be effused, that event can often be determined by fluctuation, or by percussion in some parts; or if lymph, by a rubbing sound heard under the stethoscope when the abdominal movements of respiration are not suppressed. The course of these forms of acute peritonitis varies from a few hours to ten or fourteen days.

When acute peritonitis terminates in effusion of pus, the symptoms are infinitely more formidable. The pain in the abdomen is often the severest that human nature can suffer. The patient lies on his back, but his legs are drawn up and bent so as to relax as much as possible the abdominal muscles. By fixing his pelvis he endeavours to keep the abdomen still; he is restless, and unable to bear the slightest pressure, not even the weight of a sheet, and is incessantly tossing his arms about.

in every direction. The state of his tongue and bowels is similar, perhaps, to what has been described; but his pulse is excessively small and rapid, varying from 130 to 150, while the patient is often distressingly affected by retching and vomiting. These symptoms perhaps continue without intermission for twenty-four, forty-eight, seventy-two, or more hours; when, with or without some previous shivering, pus is effused, and the pain, from being agonizing, is now bearable. The subsidence of the pain, however, is not followed by any amendment; on the contrary, a most alarming collapse succeeds,—a cold clammy sweat breaks out over the body, while hiccup, and a pulse hourly increasing in frequency, proclaim the entire hopelessness of the patient's surviving beyond a few hours.

When acute peritonitis is local—confined, for instance, to the surface of the liver or other organ—the pain is often limited to that part, while the other symptoms vary according to the severity of the affection and the organ whose covering is affected.

Chronic peritonitis often takes place to a great extent, and yet without any great amount of suffering. The symptoms are rather those of abdominal soreness and uneasiness than of pain, together with a full but sometimes rapid pulse. The intestines, indeed, may be glued together, and sometimes pus has been found effused, without the patient suffering more than in ascites. When chronic peritonitis is partial, as of the liver or spleen, the patient often experiences a dragging pain, which is increased by a change of position, and arises from the parts being suspended by adhesion.

Causes.—Inflammation of the peritoneum often manifests itself during the course of some specific disease, such as *paludal fevers, scarlet fever, Bright's disease, scrofula,* and the like. Mechanical violence, as the kick of a horse, or a penetrating wound, the operation for hernia, lithotomy, ovariotomy, gastrotomy, or of paracentesis, are occasional causes. Rupture of the intestine from ulceration, or the bursting of an abscess, or of an aneurismal tumor into the abdominal cavity, are examples of another class of causes. Errors of diet and drink, and especially frequent intoxication, are also occasional causes. The disease termed *gin colic* is a chronic inflammation of the peritoneum. Sudden and great changes of temperature are also causes, especially in women at the period of menstruation. Intus-susception of the intestine, or strangulation of the intestine from hernia, or other accidents, are also occasional causes. As a secondary disease it is frequently produced by hepatitis, splenitis, enteritis, and by *cancerous, typhoid,* and *tubercular* deposits in the subperitoneal tissue.

Children sometimes die of peritonitis after fevers; but the disease is most common between the ages of twenty and forty. Women appear to die more frequently from it than men; this greater liability to peritonitis in the female arising, perhaps, from the great sympathy between the uterus and the peritoneum—a sympathy which is strongly marked, not only at the period of menstruation, but also at the time of parturition. At the latter period, indeed, puerperal peritonitis often becomes contagious among parturient females.

Diagnosis.—The pain being greatly increased on pressure, and the pulse rapid, together with the general uneasiness and evident danger of the patient, readily distinguish peritonitis from colic. Its salient points of difference from enteritis will be noticed under that head.

Prognosis.—Partial peritonitis often terminates without in any sensible degree impairing the general health. Thus we often find extensive adhesions of the liver without any marked symptoms, as well as limited opacities of the membrane. In every case, however, in which the structure of the peritoneum is thickened or otherwise impaired, the patient may recover, but generally he relapses and dies of dropsy; for the peritoneum, like all other serous tissues, appears to possess little power of restoration after disease. Every attack of acute inflammation is of grave import, and when pus is effused it is very generally fatal; neither will the patient likely recover if the peritonitis is caused by subperitoneal tubercles, typhoid or cancerous lesions.

Treatment.—The treatment of acute peritonitis must be active—the activity of the treatment being proportioned to the amount of pain, the rapidity of the pulse, and intensity of the inflammatory fever, which is frequently marked by the peculiar depressing influence of the inflammation on the heart's action. In the milder forms of the disease, when the pain is bearable, and the pulse steady and under 100, twenty leeches over the abdomen, followed by warm fomentations, together with the administration of opium in frequently repeated grain doses, ought to be the basis of treatment. In the severer forms of disease the first indication is to relieve pain. The stomach may be unable to retain food; and vomiting may be present. Under such circumstances, Dr. Anstie has found great benefit from the injection *per rectum* of a pint of strong meat soup slowly thrown up in three successive portions. About three hours' relief of pain was obtained, when a new injection of soup being given, the same relief to pain followed, and the pulse fell from 124 to 104. Small quantities of broth and wine could then be borne by the stomach, which were administered every two hours (*op. cit.*, p. 117). All action of the bowels should be prevented for several days. *Opium* should be given in doses of one to two grains, repeated as often as its effects subside—generally every two, three, or four hours. *Morphia* may be given in doses of a quarter to half a grain, and similarly repeated. *Morphia* combined with *chloroform*, as in the formula for *chloromorphine* or *chlorodyne*, will be found useful in allaying pain.

Leeches applied to the abdomen is the only method of blood-letting likely to be useful, and a poultice afterwards may be required to encourage the bleeding in sthenic cases. It is not proven that *calomel*, given for the purpose of inducing mercurialism, has any curative tendency in peritonitis. It has been prescribed traditionally; but the experience of Dr. Taylor regarding *mercury* in *pericarditis*, as well as of other physicians regarding the influence of mercury generally in the cure of these inflammations, tends to discard it now from our methods of cure in such affections. Fomentations are to be diligently employed, on the same principle as described under *enteric fever*, vol. i., p. 882.

Niemeyer recommends that the entire abdomen be covered with cold compresses and renewed every ten minutes; at the same time he acknowledges, that warm cataplasms are better borne than cold compresses by many patients.

In chronic peritonitis *iodide of potassium* combined with *alkalies* I have found a most useful combination; together with inunction of the abdomen with the *iodine ointment*.

Acute peritonitis has also been treated by *quinine* alone, in large and repeated doses, by M. Beau, at the Hôpital de la Charité. The remedy was given in the proportion of twenty to thirty grains of the drug in the twenty-four hours. When the abdomen can bear pressure, a flannel roller should be firmly applied round the body.

Chronic vomiting is one of the most distressing concomitants of this affection, and is to be met by effervescing draughts, combined with ♏ j. or ♏ ij. of *dilute hydrocyanic acid*, or with ♏ iij. to ♏ v. of *tinct. opii.* One or two drops of *creosote* in some aromatic water may also be tried. These are perhaps our most efficient remedies; "but the physician," says Sir Robert Christison, "ought not to be surprised if he finds all these remedies ineffectual." *Diarrhœa* also is common, so that it is almost always necessary to combine any laxative remedy with some opiate; and sometimes the bowels are so singularly irritable as to oblige us to abandon all opening medicine, and to prescribe astringents, as *kino*, *catechu*, or *hæmatoxylum*, or else the *mistura cretæ composita c. opio*, and even *pure opium*, to the amount of two, three, or four grains a day. Sir Robert Christison speaks highly of the *acetate of lead* in these cases; but under any treatment this symptom is dangerous and distressing.

The diet of the patient in the acute forms of peritonitis should be of the mildest and least stimulant kind.

ASCITES.

LATIN EQ., *Ascites;* FRENCH EQ., *Ascite;* GERMAN EQ., *Ascites*—Syn., *Bauchwassersucht;* ITALIAN EQ., *Ascite.*

Definition.—*A collection of serum slowly effused by transudation into the cavity of the peritoneum.*

Pathology and Morbid Anatomy.—Cases of ascites are often examined after death, in which no affection of the peritoneum, or of any organ or tissue, can be discovered. More commonly, however, the peritoneum shows evidence of having been either acutely or chronically inflamed, some viscus diseased, such as the liver or the heart, or some tumor pressing on the large vessels, and causing the effusion which constitutes the ascites, in consequence of the hyper-distension of small blood-vessels. It may also be the result of tuberculous or carcinomatous affections.

The most frequent concomitant affection with ascites is disease of the heart and large blood-vessels, to which it is believed that at least one-fourth of all the cases of ascites is owing. In these cases the cavities of the heart are often enlarged, and their walls either hypertrophied or atrophied, or the valves are ossified, or their action otherwise impeded, and the aorta may be pouchy—its elasticity and contractility being impaired by calcareous or other degeneration.

Morbid states of the liver and spleen are the next most frequently associated affections. These organs may be found in every possible state and stage of disease.

Sometimes *anasarca* accompanies *ascites.* It is an infiltration of serous fluid amongst the elements of the general connecting or areolar tissue of the body, passing, therefore, up through and amongst the more loosely connected parts of the body generally, such as between the skin and the muscles. In these cases the areolar tissue is found in various states: in some cases the interspaces or areolæ are greatly enlarged, while the tissue itself, generally thickened, tears most readily in some cases, while in others it is not only greatly thickened, but also greatly indurated. The fluid which it contains is generally limpid and watery, composed merely of the serous part of the blood; while in other instances the fluid is viscid, contains lymph, and the organizable elements characteristic of inflammatory origin.

The quantity of fluid contained in the abdomen in cases of ascites varies from a few ounces to many gallons: three to four gallons are by no means unusual, and as much as eighteen gallons are said to have been drawn off at one time by the operation of *paracentesis.* The quality of this fluid varies. In colour it is generally green or yellow; in consistence viscid, often containing so much coagulated matter as to be incapable of flowing through the canula.

Symptoms.—The symptoms of ascites are extremely well marked, but vary in some degree according to the cause, so that it is better to give first a general outline of its more prominent features, and afterwards to point out those particular symptoms which indicate the cause from which it springs.

In ascites, if the quantity of fluid effused be considerable, the abdomen is distended and shining, with a number of large superficial veins creeping over its surface. From the weight of the abdomen, the gait of the patient is upright, like that of a pregnant woman; and if anasarca be present, he generally walks with his legs widely apart. In bed he is unable to lie down, on account of the fluid in the abdomen gravitating towards the chest and compressing the lungs, so that he is obliged to be raised towards the head and shoulders.

If anasarca be limited to the lower extremities, the upper portion of the body is in general greatly emaciated, the sharp and pinched features and the withered arms forming a striking contrast to the protuberant abdomen and swollen legs. On the contrary, if *anasarca* be general, as the true signification of the word implies, the trunk, the arms, the hands the eyelids, and face generally, are tumid and swollen to a most unsightly degree. The urine is often defective in quantity, but is sometimes natural and sometimes in excess. The skin is dry, and the patient thirsty; his appetite greatly impaired, and his spirits generally greatly depressed.

The progress of ascites is seldom accompanied by any severe constitutional symptoms. Bronchitis may take place, or the urine may become nearly suppressed, and similar effusions may occur in the cavities of the pleuræ.

The favourable circumstances are, the secretion of urine being re-established and becoming natural, the subsidence of the anasarca and of the ascites, and then a gradual return to health.

The presence of water in the abdomen may be determined by percussion of that cavity; and the best mode is to place one hand on the abdomen, and to give a sharp but gentle tap on the opposite side with the fingers of the other, when, if water be present, a fluctuation will be felt. If, however, the quantity of fluid be small, the fluctuation is best felt by percussing the side of the abdomen from before backwards. The distension and peculiar form of the abdomen is characteristic. So long as the effusion is moderate, the shape of the belly will change with every position of the body. If he stands, the lower part of the belly is prominent—if he lies down it is broad.

The existence of fluid in the areolar tissue of the trunk or extremities constituting *anasarca* is determined by the finger leaving a mark or "pit;" and the fluid being thus displaced, the part does not recover its original form and fulness for some seconds, and is said "*to pit on pressure.*"

The ascites may form suddenly, and the abdomen of the patient may be distended in a few hours, or the fluid may take weeks or months to accumulate. The duration of the disease is indefinite. If the effusion be general, the patient's life may terminate in a few days; but more commonly the affection is chronic, and the patient survives many weeks or months. Such are the more general phenomena of *ascites* and of *anasarca;* but it is now necessary to pass to those particular forms which constitute its varieties.

Causes.—*Ascites* sometimes results from the large effusion of serum which is poured out occasionally into the cavity after undue exposure to cold and wet. To this form the name of *active ascites* has been given; and although such cases have not the marked symptoms of inflammation, such as pain, yet the febrile re-action which generally accompany such cases, and the fact that they yield to those remedies which subdue the inflammatory tendency, indicate such a connection. There are cases of a similar kind in which the *ascites* obviously results from *chronic peritonitis;* and now, although the patient sometimes suffers much pain, more commonly this symptom is wanting, or only occurs in occasional paroxysms. In this latter form the patient generally appears to die from the conjoint effects of anasarca and of ascites. The urine is scanty, and may be free from albumen in both these forms of disease.

The forms of *ascites* resulting from the following causes are examples of *passive* ascites, because they are all explicable by the fact that the cause tends ultimately to retard the flow of blood through the system of the *vena portæ.*

A *diseased heart,* or diseased state of the aorta, is often the primary cause of ascites. The heart's sounds, its impulsion, together with the character of the pulse, will indicate the particular lesion under which the patient labours, as explained under cardiac diseases. Dropsy from this cause may first show itself either by effusion into the abdomen or into the areolar tissue of the lower extremities, causing *anasarca.* When effusion has taken place, it is often remarked that the action of the heart becomes more regular, its impulse more natural, the pulse slower and steadier, while perhaps the murmur also may disappear. This apparent amendment, however, is fallacious; the dropsical symptoms increase, effusion takes place, first into one cavity and then into another, so that the patient seldom long survives this fatal symptom. The urine in this form of dropsy is generally deep in colour, small in quantity, and of a healthy density.

Diseases of the liver offer the best illustration of how morbid states obstructing the *portal* circulation are the main causes of *passive* or *mechanical* ascites. When ascites arises from a *diseased liver*, that viscus is generally enlarged; but mere enlargement is not a common condition giving rise to ascites. The liver in most instances is smaller than usual; it is contracted and condensed, so that its shrunk and diminished bulk compresses the portal circulation. The condensation is also of a peculiar kind. It generally results from a compression of the proper hepatic substance, by a contracting tendency in the new connective-tissue which accompanies the portal vessels—namely, the *capsule of Glisson*, the proliferation of which gives rise to the condition known as *cirrhosis*, or *hobnail* liver. The ascites in this case has no new feature, except that the patient may or may not be jaundiced. In the former case all the fluids effused are of a yellowish or greenish-yellow colour. The urine also is loaded with bile, which is generally turned green by the addition of nitric acid; while in a smaller number of cases the bile appears to be in a peculiar state of combination with the urine, so that the acid has now no effect on it; the urine likewise is always small in quantity, much loaded with the usual salts, and of a high density. The bowels are difficult to act upon, and the patient is liable to severe abdominal pains, simulating chronic peritonitis. The pulse continues throughout the disease for the most part natural, but the patient usually falls into a typhoid state, from which there is no recovery.

In ascites associated with disease of the *spleen*, the viscus is uniformly enlarged, and can readily be felt occupying the left hypochondriac region, and thus the cause, though not its exact nature, may be determined; for we have no diagnostic symptoms denoting whether the spleen be simply hypertropied or in a cancerous or tuberculated state. The early symptoms are similar to those which occur in dropsy from disease of the liver, and for the most part are secondary to hepatic obstruction; and the termination of the disease, if the patient dies, is generally by hæmorrhage from the stomach and bowels, often so profuse as to amount to many pints in a few hours, greatly exhausting the patient, and hastening the fatal issue.

In dropsy from disease of the *kidney* the urine may or may not contain *albumen;* but in the great majority of cases it does so. When albumen is absent (as the chronic forms of diseased kidney are all devoid of pain), we are unable to determine either the seat or the nature of the disease with which ascites is associated; and the ascites is consequently in general attributed to an affection of the peritoneum, of the liver, the spleen, the kidneys, the heart, or other viscus.

The causes of ascites have been already sufficiently indicated, and may be summed up as comprehending undue exposure to sudden changes of temperature, loss of blood, obstruction of the portal circulation from morbid states of the liver, or spleen, or heart, and especially from dilatation of the chambers of that organ. The lesions of the kidney associated with ascites generally exercise their pernicious influence through the diseases of the heart which supervene during their course. Such cases prevail most between the ages of twenty and forty-five, while cases from disease of the heart and liver are most common from the ages of forty to sixty.

Diagnosis.—Ascites is readily distinguished in the male from every other intumescence of the abdomen by the fluctuation on percussion. In the female it can only be confounded with pregnancy, or with *ovarian dropsy*, which consists in the accumulation of fluid in one or more cysts, generally multilocular, within the substance of the ovary, or in a serous cyst connected with the uterine appendages.

The grounds of diagnosis may be thus shortly stated:—

(1.) The uniform and symmetrical appearance, and general increased breadth across the flanks, possesed by the abdomen in *ascites* from the first, contrasted with the general *one-sided* growth of an ovarian tumor.

(2.) Percussion gives useful diagnostic results when performed in the different positions in which a patient may be placed. In ovarian dropsy dull sounds are fixed and invariable in one place, whatever position is assumed by the patient. In ascites the dull sound follows the gravitating fluid in all positions.

The development of hydatids in the abdominal cavity may also simulate ascites, and so may an over-distended bladder.

Prognosis.—The *prognosis* in anasarca, in young persons not labouring under any organic disease, is always favourable. If, however, it be consecutive to organic disease, a fatal termination is ultimately to be feared.

Ascites arising from indeterminate causes is often recovered from; but no case is free from danger, the peritoneum often being so much impaired that the function of absorption ceases to be effected through it, and so the disease resists the action of all remedies.

Ascites depending on moderate inflammation of the peritoneum is often recovered from, and especially if the inflammation depends on the action of exposure to cold or to paludal poison.

Ascites with albuminous urine, arising from mere functional disorder of the kidney, is generally recovered from; but if the structure of the kidney be impaired, the disease is always grave, and generally fatal. In a few cases, however, the ascites may subside, and the patient may continue well for two, three, or four years, when he generally relapses and dies.

Ascites from disordered function of the heart is often recovered from; but if it depends on diseased structure, either of the heart or large vessels, some temporary amendment may take place, while the patient quicky relapses and finally sinks.

Ascites depending on diseased structure of the liver or of the spleen is rarely recovered from unless the primary disease be cured.

Treatment.—When ascites occurs without any obvious organic cause, and without albumen in the urine, the best remedy is the *bitartrate of* *potash*, administered in divided doses, as one drachm three times a day, or every six hours, or in one large dose, as half an ounce, combined, if the patient's bowels be confined, with ten to fifteen grains of *jalap.* When the smaller doses are used, it is often exceedingly useful to add ten grains of the *citrate* or *tartrate of iron* to each dose. If these remedies should fail, one-sixth to half a grain of the *extract of elaterium* every night, or every other night, may be given. When ascites is accompanying with anasarca, *squills* appear to afford most relief; and by giving five to eight grains of the *pulvis scillæ* three times a day, the dropsy is generally relieved, and the patient sometimes cured. If the stomach be irritable, *half a grain of opium* should be added to each dose, so that the remedy may be retained. When the ascites arises from disease of the heart, the kidney being sound, and the urine free from albumen, the treatment must have reference to the nature of that disease. If the valves of the heart are diseased, the patient, though he cannot recover, may be greatly relieved by the administration of tonics, stimulants, and saline or drastic purgatives. An *ounce and a half of camphor mixture, with a drachm* of the *spirit of nitrous æther, fifteen minims* of the *tincture of hyoscyamus,* and *a drachm* of the *sulphate of magnesia* will form a draught which, taken *three times* a day, will often greatly reduce the dropsy. When the stomach will bear it, the *tincture of squills* (♏x. to ♏xx.), with a *drachm* of the *acetate of potash,* has occasionally suceeded. Small doses of *elaterium,* as one-eighth to one-fourth of a grain three times a day, is a medicine that is also sometimes useful. Should the liver be merely inflamed or hypertrophied, without other alterations of structure, the dropsy may disappear with the cure of the hepatic disease. The treatment is by bleeding, if the hepatic lesion be the congestion of inflammation, and the neutral salts, such as the *sulphates of magnesia* or *of soda;* or should they fail, by moderate doses of calomel. When, however, its structure is otherwise altered, the patient is seldom cured; but the disease may still be alleviated and life prolonged. In this form of ascites the patient suffers greatly from abdominal pains, which are relieved most effectually by fomentations. In these cases, also, the bowels are often greatly constipated, and require the most powerful drastic purgatives, as the *black draught, castor* or *croton oil,* or even *elaterium.* In this form of dropsy, however, the peritoneum becomes more impaired in its power to absorb the fluid than in most of the others; the fluid is therefore seldom reduced, and the patient generally requires the last imperfect resource of art—namely, tapping, or *paracentesis.*

Ascites depending on enlarged spleen is also difficult of cure. If the spleen be simply hypertrophied, the *bromide of potash* and the *iodide of potassium,* in doses of five to eight grains three times a day, have been recommended. The patient, however, often dies from hæmorrhage from the stomach after all the more prominent symptoms have been relieved.

My friend and colleague, Professor Maclean, informs me that rubbing the *biniodide of mercury,* in the form of an ointment, on the skin over the region of the enlarged spleen, has a very marked beneficial effect in reducing the enlargement. The ointment is to be rubbed into the skin while the patient sits before a strong fire, or in the rays of an Indian sun; and, as a further evidence of efficacy, the invalid soldiers invariably ask to be furnished with a supply of the remedy when they go from the hospital. The *biniodide of mercury*, similarly used, has been noticed as of great service in reducing the swelling of a goitre.

The dropsy which often occurs in young chlorotic women, in whom the urine *contains albumen* (the kidney being healthy in structure though disordered in function), is generally curable,—the most efficient remedy being the *bitartrate of potash* in *drachm* doses three times a day. It acts as a diuretic and as a purgative in these cases.

During the course of several of the diseases of the digestive system that have been described in this chapter, immense distress and suffering often occur from the accumulation and retention of gaseous fluid in some portions of the intestinal canal (*tympanitis*). When this symptom reaches such an extreme development as to endanger life; and after the introduction of a rectum tube fails to procure relief, experience justifies the propriety of making a puncture into the cavity of the bowel (generally the colon), through the abdominal parietes, to permit the gas to escape, and so relieve the tympanitis. Life may thus be saved, and cannot be materially shortened by the operation, if it is performed by a trocar of the very smallest exploring size. The air gradually and rapidly escapes by the very smallest orifice, which is rather a separation of tissues than a wound, and soon closes up so completely as to leave no trace behind. The operation gives the greatest possible relief.

For records of such cases see *American Journal of Med. Science*, N. S. xxiv., p. 543; *Obstet. Trans.*, for 1869, pp. 47, 48, by Dr. J. Braxton Hicks, in 1868: *Brit. Med. Journal*, for Oct. 21, 1871, p. 464, by Mr. J. Hancocke Wathen. The operation is not the same as that usually performed on cattle. In them it is generally the *rumen* or first stomach that is punctured (M'BRIDE in *Brit. Med. Journal*, Nov. 4, 1871).

CHAPTER XXI.

DISEASES OF THE URINARY SYSTEM.

SECTION I.—THE KIDNEY AND ITS SECRETION IN RELATION TO DISEASES OF THE URINARY SYSTEM.

A CAREFUL study of the structure and functions of the kidney is of the greatest importance to the student of medicine in relation to diseases of the urinary organs. The determination of the quality and quantity of the *excreta* eliminated in the form of urine is indeed one of the best methods for enabling him to determine not only morbid conditions of the kidney, but to appreciate many changes which go on in the body during disease—changes which have frequently been referred to throughout these volumes. In every case of disease (in wasting, febrile, and constitutional diseases especially) much may be learned regarding their natural history by a careful examination of the urinary excretions, and of the microscopic characters of the sediment, especially when studied in connection with the ranges of temperature of the body, the number of respirations, and the state of the pulse, during daily periods of twenty-four hours. A formula has been already suggested at pages 240, 242, and 243, vol. i., as a method to be followed in daily records; and it is now clearly understood that the *absolute amount excreted in a given time, and referring that amount to body-weight,* are the only conditions which will yield anything approaching to accurate scientific results (PARKES). "In a medical point of view it is a mere waste of time to estimate the constituents in a certain quantity of urine, passed at any particular hour of the day or night, without ascertaining the relation which that quantity, with its constituents, bears to the whole quantity passed during the *twenty-four* hours; and this is the more necessary as the amount of fluid secreted varies considerably in healthy persons (SUTTON). The volumetric methods of determining the urinary constituents have greatly facilitated such investigations; and these methods are clearly expounded in the works of Hoppe, Neubaur, and Vogel, Thudichum, Beale, Hassall, and Sutton; and, above all, the profession is indebted to Dr. Parkes for his philosophical exposition of the value of our present knowledge regarding the state of the urine in health and disease, as set forth in his admirable work on that subject, already frequently referred to. From these works the following sections are compiled.

SECTION II.—ON DETERMINING THE COMPOSITION OF THE URINE IN DISEASE.

It is examined for two purposes,—(1.) To discover the condition of the urinary organs; (2.) To determine the course of the abnormal metamorphoses of tissue in the body which lead to alterations in the composition of the several excreta.

The normal urinary constituents have probably a fixed physiological range concurrent with age and weight of the body, so that a person ought not to continue passing regularly an amount of any constituent very greatly above or below the limits of the range proper to him, otherwise some morbid condition must be sought for to explain the nature of the occurrence. The averages of the excreta for at least ten days should be taken in health, in order to arrive at an accurate standard. But as this is not possible in cases of disease, certain empirical formula are laid down by Dr. Parkes for calculating the urinary excretion in a sick person whose normal excretion is unknown. The following are the details of the facts to be determined and recorded:—

I. Record the age and weight of the patient in pounds avoirdupois.*

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* Beneke recommended, in 1854, the very useful "*Patent Decimal Waagen*" *of Schoenemann* (patent decimal balances), for the determination of the body-weight of patients, and also for physiological and clinical investigations. These balances

II. Collect all the urine passed in twenty-four hours; measure it in cubic centimetres, and record its absolute amount.

III. Observe and record as to the following general properties, viz.:—(1.) Specific gravity; (2.) Uræmatine, as determined by Vogel's colour table;* (3.) Clearness or turbidity on emission or after rest; (4.) Determine the absolute weight, by multiplying the quantity passed, expressed in c. c., by the figures expressing the specific gravity,—the result is the weight in grammes.

IV. Set aside the following quantities for the volumetric determination of—

1. Urea,	40 c. c.
2. Uric Acid,	300 to 500 c. c.
3. Phosphoric Acid,	40 c. c. †
4. Chloride of Sodium,	40 c. c.
5. Sulphuric Acid,	100 c. c.
6. Degree of Free Acidity—	
7. Sugar,	20 c. c.
8. Albumen—	
Solids,	20 grammes.

V. Collect and examine the sediment.

VI. Determine the amount of excretion normal to the individual by the following empirical formula (PARKES):—Multiply the following figures by the weight of the person in pounds avoirdupois; the result is the excretion in grains, in twenty-four hours, of the several ingredients of the urine:—

The following corrections are required:—(1.) If the person be between forty and fifty, calculate according to columns 1 or 2, and then deduct 10 *per cent.;* for ages between fifty and sixty deduct 20 *per cent.;* for ages between sixty and seventy deduct 30 *per cent.;* for ages upwards of seventy deduct 50 *per cent.* (2.) If the person has been starving for two or more days (as in some fevers), deduct one-third from the calculation made according to the table; if the diet be meagre, deduct one-eighth or

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are manufactured by Messrs. Kuhtz & Co., at Brandenburg, on the Havel. They are made either entirely of iron or partly of wood. In the latter case the absolute weight of the wooden seat may changes lightly with the atmosperic moisture, although perfectly seasoned dry wood is used in the construction. The balance turns with 1 gramme in 130 pounds. A small ivory scale, opposite the end of the lever, enables the appreciation of even still smaller differences of weight. The price of such a balance, constructed in wood (with weights, large and small, in a box), is 45 thalers. (See *Berlin Klin. Wochenschrift*, viii., 18, 1871; also Schmidt's *Jahrbeucher*, 1871, 8.)

* Vogel's colour table is made by taking certain colours as starting points, and representing the following groups for comparison:—

I. *First group—Yellowish urines*, represented by gamboge mixed with a greater or less amount of water, giving three grades, the first of which is almost colourless, as follow:—(1.) *Pale yellow* (gamboge with much water); (2.) *Light yellow* (gamboge with less water); (3.) *Yellow* (gamboge with very little water).

II. *Second group—Reddish urines*, represented by the above yellow grades mixed with a greater or less amount of red—*i. e.*, gamboge with crimson lake in three grades, as follow:—(4.) *Reddish-yellow* (gamboge with a little crimson lake); (5.) *Yellowish-red* (gamboge with more crimson lake); (6.) *Red* (crimson lake with little gamboge).

III. *Third group—Brown or dark urines*, represented by gamboge, crimson lake, and more or less Prussian blue, in three grades, as follow:—(7.) *Brownish-red* (red with an admixture of a little brown); (8.) *Reddish-brown* (more of the brown than in the last); (9.) *Brownish-black* (almost black, with a touch of the reddish-brown). (See Thudichum *On the Pathology of the Urine*, p. 134.)

† The precipitate from the urea estimation is sufficient.

one-sixth ; if pretty plentiful, yet still below that of health, deduct one-tenth. (3.) If there be total inactivity, deduct one-tenth ; if there be merely quietude, deduct one-twentieth.

	In Men between 20 and 40.	In Women between 20 and 40.	In Children between		In Young Men and Women between 16 and 20
			3 and 8.	8 and 16.	
Urea, ...	3·53	2·96	6·83	5·20	4·39
Chlorine, ...	0·875	0·817	1·44	1·097	0·926
Sulphuric Acid, . .	0·214	0·25	0·414	0·315	0·266
Phosphoric Acid, .	0·336	0·336	0·65	0·495	0·418

SECTION III.—VOLUMETRIC ESTIMATION OF THE MORE IMPORTANT CONSTITUENTS OF THE URINE, AND THEIR PATHOLOGICAL RELATIONS.

The Volumetric Method of Analysis is one which enables ordinarily skilful operators, or medical men who cannot devote much time to practical chemistry, to determine with sufficient accuracy the amount of the most important constituents of the urine, such as *urea, uric acid, chloride of sodium, phosphates, sulphates*, and the *free acidity* of this excretion. But there are also many volatile substances excreted which are not easily estimated. For example, the kidneys are the agents by which many extraneous substances received into the circulating system are removed from the body, as *turpentine, copaiba, myrrh, iodine, rhubarb*, the *odoriferous particles of asparagus*, or of other substances.

Certain conditions are necessary for success, namely,—(1.) Solutions of the re-agents or tests, the composition and strength, or chemical power of which are accurately known; (2.) *Burettes*, graduated tubes or vessels from which portions of the test-solutions may be accurately delivered; (3.) The power of determining by the eye when the decomposition produced by the test-solution with the urine has ceased, so that the quantity of test solution used (*i. e.*, with which certain components of the urine have combined) may be accurately determined.*

It is necessary to exercise the greatest care in the graduation of the measuring instruments, and in the strength and purity of the standard solutions; because a very slight error in the process is greatly magnified—in proportion, in fact, as it is multiplied to represent the amount in large quantities when the actual observation has been made upon a small amount.

1. **Estimation of Chlorides.**—They are calculated as *chloride of sodium ;*

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* The apparatus and the test-solutions required, graduated to the proper strengths, are supplied by Mr. Griffin, 119 Bunhill Row ; and by Messrs. Bullock and Reynolds, Hanover Street, London ; also Harper and Sutton, Operative Chemists, Norwich.

the test-solutions required being—(1.) The *standard solution of the nitrate of mercury*; and (2.) The "*baryta-solution*," as it is commonly called. The method was devised by Liebig, and its principle is as follows:—"If the solution of *nitrate of mercury*, free from any excess of acid, is added to a solution of urea, a *white gelatinous precipitate* is produced, containing urea and *oxide of mercury* in the proportion of 1 eq. of the former to 4 eqs. of the latter. But when *chloride of sodium* is present in the solution, *the precipitate does not occur until all the chloride of sodium is converted into chloride of mercury* (*sublimate*) *and nitrate of soda*, the solution *remaining clear*. If the exact point be overstepped, the excess of mercury immediately produces the precipitate above described, so that the *urea* present acts as an indicator of the end of the process. It is therefore easy to ascertain the proportion of chlorides in any given sample of urine by this method, if the strength of the mercurial solution is known; since 1 eq. of *oxide of mercury* converts 1 eq. of *chloride of sodium* into 1 eq. each of *corrosive sublimate* and *nitrate of soda*" (SUTTON).

The steps of the process are as follow:—

(1.) Take 40 c. c. of urine; (2.) Mix with 20 c. c. of the baryta-solution; (3.) Pour the thick mixture upon a small dry filter; and when sufficient clear liquid has passed through, (4.) Take 15 c. c. of it (= 10 c. c. of urine), and just neutralize it, or render it acid by a drop or two of nitric acid; (5.) Bring this urine fluid under the burette which contains the test-solution of the nitrate of mercury, which is to be allowed to drop gradually, drop by drop, into the beaker containing the urine, which is to be constantly stirred with a glass rod; (6.) As soon as the precipitate does not disappear by stirring, the operation is finished, but a permanent precipitate is produced (= urea and oxide of mercury); (7.) The volume of the test-solution used is to be read off the burette, and the amount of *chloride of sodium* calculated therefrom; (8.) The chlorine may be estimated by the following formula:—As 58·8 eqs. of chloride of sodium contain 35·5 eqs. of chlorine, the chlorine in the urine is obtained by the equation,—

$$58{\cdot}8 : 35{\cdot}5 :: \text{amount of chloride of sodium in the urine} : x \text{ (the chlorine it contains).}$$

The average amount of chlorine excreted in twenty-four hours is about 8·21 grammes, or 126·76 grains; 13·6 grammes, or 210 grains of chloride of sodium, were the chloride always united with that substance—an amount which Dr. Parkes thinks is too great. Vogel and Parkes consider the mean to be 7 grammes, or 108 grains = 11·5 grammes, or 177 grains of chloride of sodium in twenty-four hours; the range above and below the mean being from 30 to 60 *per cent.*

In relation to body-weight, the averages of chlorine are as represented in the second line of the table at p. 1041, paragraph VI.

Pathological Relations.—The chlorine contained in the urine is wholly derived from the food; and a part, chiefly united with *sodium*, passes out of the system without having entered into the composition of the tissues; another portion, uniting with the tissues, is only set free on the disintegration of these tissues; and therefore whatever exceeds the amount taken in by the food is derived from and represents change of tissue. In *ague* its elimination is increased during the cold and hot stages. In *acute pneumonia* their elimination is greatly lessened, often entirely absent, but re-appearing in the urine eight or ten days after resolution has set in and excess of urea has passed away. It evidently accumulates in the inflamed portion of the lungs. Their amount is also reduced in *typhus and typhoid fever, febricula, scarlatina, erysipelas, puerperal fever, pleurisy, acute capillary bronchitis, acute pulmonary phthisis, acute rheumatism, cholera, acute and chronic Bright's disease.*

2. **The Estimation of Urea** in urine is based on the combination which forms between urea and oxide of mercury in neutral or alkaline solutions. The method was devised by Liebig. The precipitate which is formed is insoluble in water, or in weak alkaline solutions. The standard test-solutions are the same as in the preceding estimate; and the indicator which shows when all the urea has entered into combination with the mercury, and when the latter predominates, is a solution of carbonate of soda.

(1.) Take one volume of the baryta-solution (20 c. c.), and mix with two volumes of urine (40 c. c.);* (2.) After filtration, take 15 c. c. of the fluid (= 10 c. c. of urine) for each analysis, in small beakers; (3.) Bring the beaker under the burette containing the mercurial solution, which is to be added in small quantities so long as a distinct precipitate is seen to form, the mixture being stirred constantly; (4.) A plate of glass or porcelain is now to be sprinkled with a few drops of solution of carbonate of soda, and a drop of the mixture brought from time to time in contact with the drops of soda-solution by means of a glass rod. So long as the mixture of the two drops thus brought in contact remains white, free urea is still present in the mixture, and more of the test-solution must be added to the urine, till the contact of the drops with the soda-solution produces a yellow colour, which is distinctly apparent; (5.) Record the quantity of mercurial test-solution used, and so calculate for the amount of urea contained in the 10 c. c. of urine, and hence in the total discharge for twenty-four hours; (6.) Repeat the analysis at least twice.

Another method has been recommended by Dr. Edmund W. Davy (*Phil. Mag.*, vol. vii., fourth series, p. 385). The process is based on the fact that *urea* is readily decomposed by *hypochlorite of soda*, when the *nitrogen* being evolved as a gas, the amount of urea is estimated from the amount of nitrogen gas produced by the decomposition. A strong glass tube is required, about fourteen inches long, closed at one end, and its open extremity ground smooth. The bore of the tube must not be larger than the thumb can conveniently cover—*i. e.*, half an inch in diameter. It ought to be graduated into cubic inches, commencing from the closed end, and each cubic inch again sub-divided into 10ths and 100ths.†

The following are the details of the process:—

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* The precipitate may be reserved for determining the phosphoric acid.

† Cassela, 23 Hatton Garden; Negrette & Co., Holborn Hill; also Messrs. Griffin 119 Bunhill Row, London, keep such tubes.

(1.) Fill the tube more than one-third full of fluid mercury; (2.) Pour in carefully half a fluid drachm to one drachm of urine; (3.) Holding the tube in one hand near its open extremity, and having the thumb in readiness to cover the aperture, quickly fill it completely full, with a solution of hypochlorite of soda (taking care not to overflow the tube), and then instantly cover the opening tight with the thumb; (4.) Rapidly invert the tube once or twice to mix its contents; and (5.) Finally open the tube under mercury contained in a strong cup or small mortar; (6.) The tube is left in the upright position till the evolution of gas ceases, which it generally does in from three to four hours; (7.) Record the amount of gas found, and estimate the urea by the following data:—1·549 cubic inch of gas represents one grain of urea.

The hypochlorite of soda used should always be five or six times the volume of the urine operated upon; and the *liquor sodæ chlorinatæ* should be perfectly pure, prepared according to the process of the Dublin Pharmacopœia, as the *liquor* of commerce always gives erroneous and exaggerated results.

A third method for determining the urea consists in the use of the following table (already printed in vol. i., and repeated at pages 1046 and 1047 for convenience of reference), which is founded on many observations of urine, both of health and disease, of specific gravities from 1,003 to 1,028. The Rev. Samuel Haughton has devised this table, and its results are approximations to the daily excretion of urea, in all cases in which sugar is absent, and albumen either absent or only present in small quantities (*Medical Times*, Oct. 22, 1864). It is a table of double entry, to be used by finding the daily excretion of urine in fluid ounces, and its specific gravity, by means of a carefully graduated urinometer. When these data are found, at the intersection of the corresponding columns, the excretion of urea is given in grains.[*]

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[*] My friend Dr. De Chaumont has compared the trustworthiness of these processes in twenty-six cases (mostly healthy, and none either albuminous or diabetic), with the following results:—"By Liebig's process the actual average gave 25·78 parts in 1,000. The average of nineteen of these taken from Haughton's Table (the remaining seven being of specific gravity above 1,028, the limit of Haughton's Table) gave 28·17 parts in 1,000. The average of the whole twenty-six, deduced from Haughton's second formula (viz., *excess* of *sp. gr. over* 1,000 × 10 = grs. in a pint) gave 32·81 parts in 1,000.

The average obtained by the formula A:—*Excess of sp. gr. over* 1,000 × 10 = parts in 1,000, gave 28·71 parts in 1,000.

The average obtained by the formula B:—*Excess of sp. gr. over* 1,000, *after elimination of the sp. gr. due to chlorides*, ÷ ·5 (= × 2) = parts in 1,000 gave 26·200 per 1,000.

The average obtained by the formula C:—*Excess of sp. gr. over* 1,000 *after elimination of sp. gr. due to chlorides*, ÷ ·52 = parts in 1,000, gave 25·65 parts in 1,000.

Tabulating these, we have:—

True Average, ...	= 25·78	*error.*
Haughton's Table, ...	= 28·17	+ 9·2 *per cent.*
Haughton's Second Formula, . . .	= 32·81	+23·4 ,,
Dr. De Chaumont's Formula A, . . .	= 28·71	+11·3 ,,
Dr. De Chaumont's Formula B, . . .	= 26·203	+ 1·6 ,,
Dr. De Chaumont's Formula C, . . .	= 25·650	— ·5 ,,

The mean amount of urea excreted by adult males between twenty and forty years of age during the twenty-four hours is 33·18 grammes, = 512·4 grains. If the mode of life be equable, the amount remains pretty constant; but the maximum and minimum amounts passed on any one day by an individual are usually about *one-fifth* above or below the mean amount. In relation to body-weight, the average excretion of *urea* is as stated in the first line of the table at p. 1041, paragraph VI.

Pathological Relations.—All the nitrogenous tissues contribute, by their disintegration, to the formation of urea; and the urea excreted may be taken as a measure of the extent of tissue-change or waste. In all *pyrexial states* its formation is increased; but its elimination may be increased or diminished (see chapter on "Fever," vol. i.) When freely eliminated by the urine, its amount stands in close relationship with the exaltation of temperature and intensity of the fever. Retention of urea is a very unfavourable circumstance. No retention occurs in *acute yellow atrophy of the liver*, in *epilepsy*, or in *jaundice*.

There are some cases, which stand by themselves, in which the excretion and elimination of urea is augmented, which were originally noticed by Prout, and which Willis subsequently described as cases of "*azoturia.*" They have recently been noticed in an excellent paper by Dr. Sieveking in the pages of the *British Medical Journal* for May, 1865. They are characterized by a great excess of urea in the urine, without increase in the quantity of the urine, and apparently without febrile re-action, but with languor, weakness, and nervousness, associated sometimes with dyspepsia, intemperance, mental anxiety, or sexual excess. Such cases are apt to pass into *diabetes mellitus* (PROUT).

3. **Estimation of Sulphuric Acid** requires the *chloride of barium* test-solution and Beale's filter. The details of the process are as follow:—

(1.) Measure off 100 c. c. of urine into a Florence flask; (2.) Add a little hydrochloric acid (twenty or thirty drops); (3.) Apply heat through a sand bath till the acidulated urine boils; (4.) Allow the chloride of barium test to flow very gradually into the urine; (5.) Remove the heat, and allow the precipitate to subside after each addition; (6.) Continue adding the test till the precipitation is complete; (7.) Use Beale's filter for ascertaining the end of the precipitation. When no precipitate is formed either by chloride of barium, or by sulphate of potassa or soda, the analysis is complete.

The mean of sulphuric acid excreted in twenty-four hours is 2·012 grammes, = 31·11 grains, with a range of 45 *per cent.* above or below. In relation to body-weight, the average excretion of sulphuric acid is as stated in the third line of the table at p. 1041, paragraph VI.

Pathological Relations.—Sulphuric acid originates in the urine from various articles of food and drink, in which it exists ready formed: from the sulphur of the food, by oxidation in its passage through the system; from oxidation of the sulphur of the tissues; or from the oxidation of sulphur contained in substances such as *taurine* and *cystine*. Its elimination is increased in *febricula, typhoid fever, typhus, variola, pyæmia, milk fever, delirium tremens, acute pneumonia, rheumatic fever, chorea.*

Fluid Ounces.	SPECIFIC GRAVITY.																									
	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028
20	35	36	43	57	71	85	100	103	106	119	130	136	142	151	160	196	233	241	249	257	265	274	276	278	279	280
21	37	38	45	59	74	89	105	108	111	124	136	142	149	158	168	205	245	253	261	269	278	288	290	292	292	294
22	38	40	47	62	78	95	110	113	116	130	143	149	156	166	176	215	257	265	274	282	292	301	303	305	306	308
23	40	41	49	65	81	97	115	118	121	136	149	156	163	173	184	225	268	277	286	295	305	315	317	319	320	322
24	42	43	51	68	85	101	120	123	127	142	156	163	170	181	192	235	280	289	299	308	319	329	331	333	334	336
25	43	45	53	71	88	106	125	129	132	147	162	170	177	188	200	245	291	301	311	321	332	342	345	347	348	350
26	45	47	55	73	92	110	130	134	137	153	169	176	184	196	208	254	303	313	324	334	346	356	359	360	362	364
27	47	49	57	76	95	114	135	139	143	159	175	183	191	213	216	264	314	325	336	347	359	369	372	374	376	378
28	48	50	59	79	99	118	140	144	148	165	182	190	198	221	224	274	326	337	349	360	372	383	386	388	390	392
29	50	52	61	82	103	122	145	149	153	171	188	197	205	228	232	284	337	349	361	373	386	397	400	402	404	406
30	52	54	64	85	106	127	150	155	159	177	195	204	213	226	240	294	349	361	374	386	399	411	414	416	418	420
31	54	55	66	87	109	131	155	160	164	182	201	210	220	233	248	303	361	373	386	398	412	425	428	429	432	434
32	55	57	68	90	113	135	160	165	169	188	208	217	227	241	256	313	373	385	398	411	425	438	442	443	446	448
33	57	59	70	93	116	140	165	170	175	194	214	224	234	249	264	323	384	397	411	424	438	452	455	457	460	462
34	58	61	72	96	120	144	170	175	180	200	221	231	241	256	272	333	396	409	423	437	451	466	469	471	474	476
35	60	63	74	99	124	148	175	180	185	206	227	238	248	264	280	343	407	421	436	450	464	479	483	485	488	490
36	61	64	76	102	127	153	180	185	191	212	234	244	255	271	288	352	419	433	448	462	477	493	497	499	502	504
37	63	66	78	105	130	157	185	190	196	218	240	251	262	279	296	362	430	445	461	475	490	507	510	513	516	518
38	65	68	80	108	134	161	190	195	201	224	247	258	269	286	304	372	442	457	473	488	503	520	524	527	530	532
39	67	70	82	111	138	166	195	200	206	230	253	265	276	294	312	382	453	469	486	501	516	534	538	541	544	546
40	69	72	85	114	142	170	200	206	212	236	260	272	284	302	320	392	465	482	498	514	530	548	552	555	558	560
41	71	73	87	116	145	174	205	211	217	241	266	278	291	309	328	401	477	494	510	527	543	562	566	568	571	574
42	74	75	89	119	148	178	210	216	222	247	273	285	298	317	336	411	489	506	523	540	557	575	580	582	585	588
43	75	77	91	122	152	182	215	221	228	253	279	292	305	324	344	421	500	518	535	553	570	589	593	596	599	602
44	76	79	93	125	156	186	220	226	233	259	286	299	312	332	352	431	512	530	548	566	584	603	607	610	613	616
45	78	81	95	128	160	191	225	231	238	265	292	306	319	339	360	441	523	542	561	579	597	616	621	624	627	630
46	80	82	97	130	163	195	230	236	243	271	299	312	326	347	368	450	535	554	573	592	611	630	635	638	641	644
47	82	84	99	133	166	199	235	241	249	277	305	319	333	355	376	460	546	566	586	605	624	644	648	652	655	658
48	84	86	101	136	170	203	240	246	254	283	312	326	340	362	384	470	558	578	598	618	637	657	662	666	669	672

	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028
49	85	88	103	139	174	207	245	251	259	289	318	333	347	370	392	480	569	590	611	631	651	671	676	680	683	686
50	87	90	106	142	178	212	250	257	265	295	325	340	355	377	400	490	581	602	623	644	665	685	690	694	697	700
51	88	92	108	144	181	216	255	262	270	301	331	346	362	385	408	499	593	614	635	656	678	699	704	708	710	714
52	90	94	110	147	185	220	260	267	276	307	338	353	369	393	416	509	605	626	648	669	692	712	718	721	724	728
53	92	96	112	150	188	225	265	272	281	313	344	360	376	400	424	519	616	638	660	682	705	726	731	735	738	742
54	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	529	628	650	673	695	718	740	745	749	752	756
55	95	99	117	156	195	233	275	283	292	325	358	374	390	415	440	539	639	662	685	708	732	753	759	763	766	770
56	96	100	119	159	199	238	280	288	297	331	364	380	397	423	448	548	651	674	698	720	745	767	772	776	780	784
57	98	102	121	162	202	242	285	293	303	337	371	387	404	430	456	558	662	686	710	733	758	781	776	790	794	798
58	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
59	102	106	125	168	209	251	295	303	314	349	384	401	418	445	472	578	685	710	735	759	785	808	814	818	822	836
60	104	108	128	171	213	255	300	309	319	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
61	106	109	130	173	216	259	305	314	324	360	397	414	433	460	488	597	708	734	760	784	811	836	842	845	850	854
62	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
63	109	112	134	179	223	267	315	324	335	372	410	428	447	475	504	617	730	758	785	810	838	863	869	873	878	882
64	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
65	112	116	138	185	230	276	325	335	345	384	423	442	461	490	520	637	754	782	810	836	864	890	897	901	906	910
66	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
67	115	120	142	190	237	284	335	345	356	396	437	455	475	505	536	656	778	806	835	862	891	918	925	929	934	938
68	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
69	118	124	146	196	244	292	345	355	367	408	449	469	489	520	552	676	802	830	860	888	917	945	953	957	962	966
70	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
71	121	127	151	201	251	301	355	366	377	419	462	482	504	536	568	695	826	855	884	913	943	973	980	984	990	994
72	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
73	124	130	155	207	258	310	365	376	388	431	475	496	518	551	584	715	849	879	909	939	969	1000	1007	1012	1018	1022
74	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	725	861	891	921	951	982	1014	1021	1026	1032	1036
75	128	134	159	213	266	318	375	386	398	443	488	510	532	566	600	735	872	903	934	964	995	1027	1035	1040	1046	1050
76	130	136	161	216	269	323	380	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
77	132	138	163	219	273	327	385	396	409	455	501	523	546	581	616	755	895	927	959	989	1021	1055	1062	1068	1074	1078
78	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
79	136	142	167	225	280	336	395	406	420	467	514	537	560	596	632	775	918	941	984	1015	1047	1082	1090	1096	1102	1106
80	139	144	170	228	284	340	400	412	425	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120

4. **Estimation of Phosphoric Acid** is based on the fact, that when nitrate or acetate of uranium is added to a solution of tribasic phosphoric acid, containing acetate of ammonia and free acetic acid, the whole of the phosphoric acid is thrown down as double phosphate of uranium and ammonia, having a light-brown colour and a composition represented by the formula $2\ (Ur_2\ O_3),\ N\ H_4\ O,\ PO_5 + Aq$. This volumetric method for the estimation of phosphoric acid was devised by Mr. Francis Sutton, of Norwich, independently of Neubauer and Pincus, who, independently of each other, also arrived at the same process; but Mr. Sutton states that Neubauer was the earliest discoverer of the method. The standard test-solution required is the nitrate of uranium, of which 1 c. c. represents 0·1 grain of phosphoric acid. A solution of ferrocyanide of potassium (yellow prussiate of potash), in the proportion of 1 to 20, is to be used as an indicator to stop the process, so soon as the re-action is complete.

The details of the process are as follow:—

(1.) Take the precipitate produced by the baryta-solution in 40 c. c. of urine, as mentioned at page 1041, paragraph VI., having been set aside after the fluid is filtered from it. (2.) Wash the precipitate once with cold water. (3.) Treat it, while still on the filter, with warm acetic acid, to dissolve all the phosphate of baryta, which passes through the filter, leaving the sulphate behind. (4.) Wash the filter with a small quantity of boiling water, so as to remove the last traces of phosphate. (5.) Add sufficient ammonia to the solution to neutralize the acetic acid, unless the quantity of the latter be large, when somewhat less than enough to neutralize may be added. Under any circumstances the liquid must be fully acidified with acetic acid before being tested as to its strength (*titrated*), and must contain a tolerable quantity of acetate of ammonia. (6.) Take a measured quantity of the urinary phosphate-solution (say 20 c. c.) in a beaver, and gently warm it, and bring it under the burette containing the uranium-solution. (7.) Portions of the uranium-solution are to be delivered in and constantly stirred, until, when a drop is taken out with a thin glass rod, and placed in the middle of a large drop of the solution of yellow prussiate of potash on a white plate, a faint but distinct chocolate-brown colour is produced at the point of contact. A slight excess of uranium-solution is required to produce the brown colour, which indicates that all the phosphoric acid contained in the amount of urine in the beaker has been thrown down. (8.) Record the amount of uranium-solution used, and estimate accordingly—1 c. c. of the solution precipitating 0·1 grain of phosphoric acid.*

The mean amount of phosphoric acid excreted in twenty-four hours is 3·164 grammes, = 48·80 grains, with a range in the same person of from 35 to 50 *per cent.* The amount in relation to body-weight is given in the fourth line of the table at page 1041, paragraph VI.

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* Collect all the precipitates in a large bottle, and when sufficient has been obtained, recover the uranium by igniting the dry precipitate in a porcelain crucible, with the carbonaceous residue produced by burning tartrate of soda and potash in a covered crucible. The uranium is thus reduced to protoxide, while the phosphoric acid unites with potash and soda, and can be entirely extracted with boiling water. The protoxide of uranium left may be dissolved in nitric acid, and evaporated to dryness in a water-bath (Sutton, *Volumetric Analysis*, p. 208).

Pathological Relations.—Its elimination in excess represents tissue-change or waste, beyond what the food or drink accounts for. In rickets and softening of the bones the phosphates are increased. In other diseases our information respecting phosphoric acid is very uncertain. In alkaline urine the *earthy* phosphates are deposited as a white sediment. Mineral acids, anodynes, alkaline bicarbonates, opium, and preparations of iron, zinc, and strychnia, are often of service, variously combined, according to the nature of the case (see HASSAL, p. 237).

5. **The Estimation of Uric Acid.**—An improved method of estimating the uric acid by iodine has been recently devised by my friend Dr. De Chaumont, and published by him in the *Medical Report of the Army Medical Department*, for 1862 (published in 1864). The determination of uric acid by iodine is founded on the fact that solution of iodine is decolorized by uric acid, in a definite proportion—which appears to be four equivalents to one of uric acid. Dr. De Chaumont has kindly given me the following short notice of the method:—

The Solutions required are as follow:—

(1.) A standard solution of iodine. The alcoholic tincture being liable to change, it is best to dissolve the iodine in water with the aid of iodide of potassium; thus, 6·35 grammes ($=\frac{1}{20}$th of an equivalent) of iodine, with 12 grammes of iodide of potassium, are dissolved in a litre of distilled water. Of this solution, 1 c. c. contains ·00635 of iodine, and will decompose ·0021 of uric acid. If the materials be accurately weighed and measured, it will be unnecessary to graduate the solution; but, should this be desired, then the following solutions must be prepared:—(2.) Solution of uric acid, ·168 grammes are to be dissolved in a litre of water. This is best made by dissolving the uric acid in a small quantity of *liquor potassæ*, and neutralizing the excess with dilute acetic acid. Of this solution, 12·5 c. c. will completely decolorize 1 c. c. of the solution of iodine. (3.) A solution of starch, carefully filtered and free from suspended grains.

The Process.—(1.) Filter the urine from mucus. (2.) Acidify the urine, should it be alkaline, with acetic acid. (3.) Put 10 c. c. into a beaker glass, and dilute it to 50 c. c. with distilled water. (4.) Add 5-10 c. c. of starch liquor. (5.) Drop in the solution of iodine from a burette graduated to $\frac{1}{10}$th c. c. (6.) Stir each time, and wait till blue colour disappears. (7.) Do not add more than $\frac{1}{10}$th c. c. at a time. (8.) When the blue colour has remained permanent for *an hour*, read off the number of c. c. of iodine used, and multiply by 21, which will give the quantity per litre.

Example.—10 c. c. of urine took 3·5 c. c. of iodine,

$$3{\cdot}5 \times {\cdot}21 = {\cdot}735 \text{ grammes per litre.}$$

For a more accurate analysis, it is necessary to wait for twenty-four hours until all the uric acid is decomposed, taking care to add fresh portions of starch from time to time as it becomes converted into dextrine. A correction is then applied as follows:—(1.) Precipitate the salts with Liebig's baryta-solution (see process for urea). (2.) Filter and acidify with acetic acid. (3.) Test with iodine as before. (4.) Deduct the number of c. c. used after the baryta from the gross amount first used, and from the remainder calculate the uric acid as before.

Example.—10 c. c. of urine took 3·5 c. c. of iodine.

10 c. c. so precipitated, filtered, and re-acidified, took ·45 c. c. of iodine.

Then 3·5 — ·45 = 3·05 c. c. of iodine.

3·05 × ·21 = ·6405 grammes in a litre—the net result.

The mean amount of uric acid excreted in twenty-four hours appears to be ·555 grammes, = 8·569 grains. The observations to determine the average in any individual must extend over at least *five days.* The range between the maximum and minimum amount is as great as from 20 to 30 *per cent.* (PARKES.)

Pathological Relations.—Its elimination indicates metamorphoses of tissue. It increases after full, heavy, indigestible meals; and exists not alone in the spleen in health, but in still larger quantity in most of the affections, including *ague,* in which the spleen is more especially implicated. It is increased in most of the active febrile and inflammatory affections. It is greatly increased during the paroxysm of *intermittent* fevers, also in *typhoid* and *typhus* fevers. In *small-pox* and *scarlatina* it is increased; and in *pneumonia* the increase is very large. The sediments of the *urates* and of *uric acid* are most abundant from the seventh to the thirteenth day. It is increased in most cardiac affections, in hepatic cirrhosis and leucocythæmia; and considerably increased in rheumatic fever. During attacks of gout it is lessened or absent in the urine, and present in the blood; but as soon as fever and paroxysms abate, precipitates of *urates* and of *uric acid* become abundant. Its free elimination is a favourable symptom.

In the majority of chronic affections the uric acid is lessened in elimination. When the excess in the urine is due to indulgence in animal food, its mal-assimilation and defective cutaneous excretion, the amount of animal food must be reduced, the diet regulated carefully, and the functions of the digestive organs improved and strengthened. When *lactic, acetic,* or *butyric* acids occur as products of faulty digestion, all food known to produce such a result must be avoided. The vegetable bitters—e.g., *cinchona, gentian, calumba, serpentaria,* with or without *rhubarb* and *soda,* and followed by such remedies as keep the colour free, such as *compound galbanum pill,* with *extract of colocynth* and *croton oil,* will be found of great service. The action of the skin must be promoted by warm clothing. Exercise in the open air must be indulged in as much as possible; while cold baths in the morning, and friction with horse-hair gloves aid the action of the skin. The best solvent is obtained by drinking freely of soft or distilled water; but alkaline remedies, like the carbonates and bicarbonates, often give relief, as well as the salts of the vegetable acids, the *acetates, citrates,* and *bitartrates,* given in sufficient doses. The occurrence of *uric acid* in excess in the diseases mentioned must be managed according to the nature of the treatment of each disease.

6. Estimation of Diabetic Sugar.—The principle of the process is thus explained by Mr. Sutton. It is based on the fact that although a mixture of pure sulphate of copper, tartrate of potash, and caustic soda, mixed in proper proportions, may be boiled without undergoing change; yet, if only a trace of sugar be added, a very slight warming is enough to precipitate a portion of the copper as a protoxide ($Cu_2 O$). It is found that one atom of pure sugar, = 180, is capable of reducing exactly 10 atoms, = 307, of oxide of copper ($Cu O$) to the state of protoxide. Therefore, if the quantity of copper reduced by a given solution of sugar is known, it is easy to find the quantity of sugar present (*Volumetric Analysis*, p. 210).

A standard solution of pure sulphate of copper with tartrate of potash and caustic soda is required. It is to be prepared as follows:—

(1.) 34·64 grammes, = 346·4 grains, of pure sulphate of copper, previously powdered and pressed between blotting-paper, are weighed and dissolved in 200 c. c. of distilled water; (2.) In another vessel 173 grammes, = 1,730 grains, of pure crystallized tartrate of soda and potash (Rochelle salt) are dissolved in 480 c. c. of solution of pure caustic soda (specific gravity, 1·14); (3.) The two solutions are then to be mixed, and the deep clear blue solution diluted with distilled water till the whole measures 1,000 c. c. *One* c. c. of the solution so prepared represents 0·05 grains of grape or diabetic sugar. It must be preserved for use in a dark place, and in well stoppered bottles kept full. It should bear heating when diluted with about four or five times its quantity of distilled water, without any precipitate taking place; and should always be submitted to this test before being used. If any precipitate does occur, it probably arises from the alkali having absorbed carbonic acid; and in this case the addition of a little fresh caustic soda-solution remedies the evil (SUTTON, l. c., pp. 211 and 233).

The following are the details of the process for analysis:—

(1.) 10 c. c. of clear urine are diluted by means of a measuring flask to 200 c. c. with water, and a large burette filled with the fluid; (2.) 10 c. c. of the copper solution (= $\frac{1}{2}$ grain of sugar) are then measured into a flask, or white porcelain capsule, and 40 c. c. of distilled water added; (3.) The vessel is to be arranged over a spirit-lamp under the burette, and brought to boiling; (4.) The diluted urine is then delivered in cautiously from the burette until the last traces of blue colour are removed from the copper solution, and the precipitate is of a distinct red colour; (5.) It must be remembered that the urine has been diluted twenty times; so that the quantity used, divided by twenty, will represent the amount of the original urine used; and the estimate is to be made accordingly.*

7. **Estimation of Free Acid** is measured by a solution of corbonate of soda containing 530 grains in the 10,000 grain measure, = 53 grammes in the litre; and is represented by determining how many grains of

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* Haughton recommends the following formula:—"Multiply the excess of specific gravity over 1,000 by 20,—the result is grains of sugar in a pint of urine." The average of 234 determinations by Dr. De Chaumont gave 61·06 parts in 1,000; the average by Haughton's formula gave 77·65;—error, + 27· *per cent.* Dr. De Chaumont proposes to divide excess of specific gravity over 1,000 by ·54—result, = parts in 1,000. Haughton's factor, = ÷ ·4348, or × 2·3. Dr. De Chaumont's factor, = ÷ ·54, or × 1·89.

crystallized oxalic acid a certain quantity of the soda-solution will neutralize. The details of the process are as follow:—

(1.) Take 50 or 100 c. c. of perfectly fresh urine; (2.) Add from a burette a standard solution of soda in small portions at a time (say 5 c. c., or drop by drop); (3.) After every addition test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper; when the streaks cease to become red, the analysis is complete; (4.) Estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallized oxalic acid.

8. **Estimation of the Total Solid Matter.**—(1.) Measure 5 c. c. into a shallow platinum or porcelain capsule; (2.) Place it beside a vessel of strong sulphuric acid under the receiver of a powerful air-pump, and keep it *in vacuo* till all moisture is removed.

9. **Estimation of Total Saline Matter.**—(1.) Measure 10 c. c. into a small porcelain crucible; (2.) Evaporate to dryness; (3.) Add about ten drops of nitric acid; (4.) Heat the crucible to dull redness; (5.) Suffer it to cool, and add ten more drops of nitric acid; (6.) Heat it up again gradually to a moderately strong heat, until all the carbon is destroyed and the residue is white; (7.) Let it cool, and weigh.

10. **The Specific Gravity of Urine** is best taken by measuring 100 c. c. into a beaker or flask whose weight is accurately known. The increase of weight in grains will be the specific gravity, water being 1,000. Instead of 100 c. c., 50 or 25 c. c. may be taken, when the weight, multiplied by 2 or 4, will be the specific gravity (SUTTON). The urinometer gives the least accurate of all results.

For further details regarding the processes of volumetric analysis the reader is referred to the excellent little treatise of Mr. Francis Sutton, already noticed.

SECTION IV.—THE MICROSCOPIC EXAMINATION OF THE URINE, AND THE PATHOLOGICAL RELATIONS OF THE DEPOSITS.

In perfectly healthy urine there ought to be no sediment whatever, unless it be the very merest haze of mucus, or the slightest precipitate of urates caused by a low temperature. Even these may be abnormal (PARKES).

Urinary sediments, being precipitated in an amorphous state are termed *sand;* in a crystallized state, *gravel;* and when concreted into masses, *stone* or *calculus.* Besides this excess of the natural constituents of the urine, there are also some other precipitable substances occasionally found in the urine, which are entirely new or morbid formations, as the *oxalate of lime*, and the *xanthic* and *cystic oxydes*, substances, although soluble, perhaps, in certain proportions in healthy urine, yet become deposited, and form urinary sediments, which concrete into calculi.

The sediments have been broadly arranged by Dr. Parkes into the following three classes (l. c.):—

CLASS I.—SUBSTANCES SUSPENDED IN THE URINE WHICH HAVE NEVER BEEN DISSOLVED.

They commence to precipitate as soon as the urine is passed. The most important sediments belong to this class, and consist chiefly of organic bodies derived from the structures composing the urinary organs, or of the *productive* effects of disease upon the kidney, such as *inflammation*, tubercle, cancer. They often afford the only signs of kidney disease, or of the implication of the kidney in some general affection. They are made up of the following substances:—

1. *Mucus and Epithelium from the Urinary Passages.*—In many diseases the quantity of epithelium from the bladder is increased, indicating catarrhal inflammation of the mucous membranes. The epithelial cells are of various sizes and stages of formation (fig. 59); and frequently free nuclei are seen. In catarrh of the bladder the mucus, from its cohesion, is apt to form thin transparent flakes or cylinders, resembling casts from the prostate or kidney.

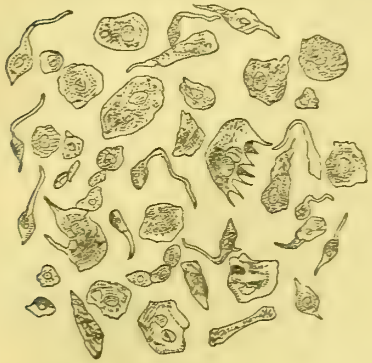

Fig. 59.*

The epithelium-cells from the pelvis of the kidney are often triangular or caudate, with well-defined nuclei. They generally adhere together in groups of three to ten, when they appear to have an imbricated arrangement, and perhaps are more closely connected than natural, by adhesive mucus (fig. 60). They are never found in healthy urine, but are present very commonly in catarrhal and calculous cystitis. Tailed or caudate cells are also sometimes present with pelvic epithelium.

ig. 60.†

The epithelium from the ureter is columnar in its character (fig. 61), and not unlike that found in the male urethra, which is mostly columnar, and is more flattened than that of the bladder, and less regular than that of the pelvis. Mixed with it there is a good deal of scaly epithelium, especially towards the orifice of the urethra.

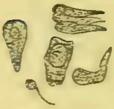

Fig. 61.‡

Large cells of scaly epithelium are also often met with in the urine of females. They are derived from the vagina. They vary much in size and form, and are sometimes very irregular in shape, with uneven ragged edges (fig. 62).

Renal epithelium is only found in disease. It consists of round or slightly compressed cells, or masses of material, with well-defined central portions or nuclei, which are not cleft like the pus nucleus under the action of acetic acid, but become at first more defined and afterwards

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* Epithelium from the bladder. Many of the large cells lie upon the summit of the columnar and caudate cells, and depressions are seen on their under surface. One is seen near the centre of the figure (after Dr. BEALE, *On the Urine*, p. 259).

† Epithelium from the pelvis of the kidney (after Dr. BEALE, l. c., p. 188).

‡ Epithelium from the ureter (after Dr. BEALE, l. c.)

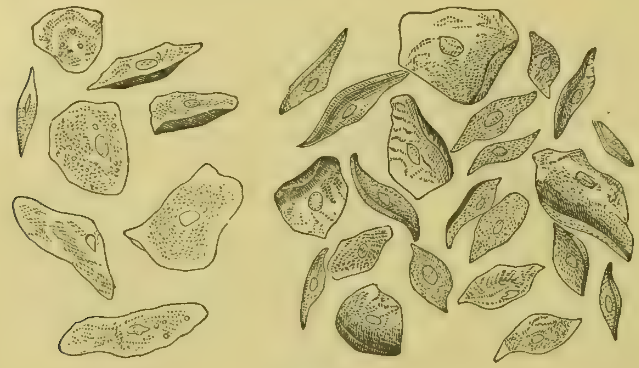

Fig 62.*

paler and smaller. In the urine they are less polygonal and more rounded than they are in the renal canals (fig. 63). Their presence indicates more or less desquamation from the tubes; while their morbid condition and admixture with other products may indicate still greater disease. Thus it is sometimes fatty; the whole space between the nucleus and the cell-wall being filled with fatty globules; but these changes and their value have been fully considered under chronic Bright's disease, p. 1080, *et seq.*

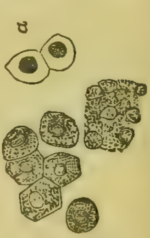

Fig. 63.†

2. *Other Cell-Forms occur in the Urine, the productive results of inflammatory diseases* of the mucous membrane of the urinary passages. They are chiefly granules, singly or in clusters, and pus. The pus is known by its nucleus becoming cleft into two, three, or five divisions, under the action of acetic acid. The amount of pus is generally far greater in *cystitis* than it is in *pyelitis* or *nephritis;* and wherever there is a markedly large quantity of pus it is generally from the bladder, unless there be a prostatic or other abscess communicating with the urethra.

3. *Cancer-Cells* occur in cancerous disease of the bladder, kidney, or urethra.

4. *Tubercle-Masses* may occur in tuberculous disease of the bladder or kidney.

5. *Cylinders* occur in the urine, and have various modes of origin:—(*a.*) From the *bladder*, as long, flat, membraniform, twisted, or folded bodies. (*b.*) From the *prostate*, as coagula, two or three times as broad as renal cylinders. They are soluble, in acetic acid. Amylaceous corpuscles may exist in them. (*c.*) From the *ureter* and *pelvis* of the kidney the coagula are cylindrical, pyriform, or globular. (*d.*) From the kidney-

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* Vaginal epithelium from urine (after Dr. Beale, l. c., p. 260).

† Epithelium from convoluted portion of uriniferous tube.—(*a.*) Treated with acetic acid × 215 (after Dr. Beale, l. c., p. 187).

tubes cylinders or renal casts are formed in various diseases. They vary in breadth from $\frac{1}{500}$th to $\frac{1}{1000}$th of an inch—*i. e.*, from about the breadth of the straight renal tubes to half or a third of that size. Their length varies from $\frac{1}{200}$th to $\frac{1}{50}$th of an inch. The terms used by Dr. George Johnson, to whose researches on diseases of the kidney we owe much, express very well the special character of the several varieties of casts. They are—(1.) Epithelial casts; (2.) Large waxy casts; (3.) Small waxy casts—the hyaline casts of Vogel and Basham; (4) Granular casts; (5.) Oily casts; (6.) Bloody casts; (7.) Purulent casts.

Solution of iodine is the best re-agent for making the waxy hyaline or transparent casts visible. The chemical composition of these casts is unknown. They are probably albuminous.

6. *Kidney-Structures* may occur in the urine if the structure of the kidney has commenced to break up.

7. *Blood-Corpuscles* indicate ruptures of vessels somewhere in the urinary passages. Copious hæmorrhage is usually from the bladder, carcinoma of the kidney, or calculus of the pelvis. The corpuscles are often very much modified in form.

8. *Fibrin* is present in many cases of bloody urine.

9. *Corpora amylacea* may occur from the prostate gland.

10. Fat. 11. A fatty substance, to which the name of urostealith has been given. 12: *Spermatozoa.* 13. *Sarcinæ.* 14. Hair. 15. *Various entozoa* have been occasionally found in the urine. (See under "Entozoa," vol. i.)

CLASS II.—SEDIMENTS FORMING IN THE URINE AFTER SECRETION, BUT WHICH MAY DEPOSIT IN THE RENAL PASSAGES OR AFTER EMISSION, EITHER IN CONSEQUENCE OF CHEMICAL CHANGES OR FROM CHANGE OF TEMPERATURE.

(1.) *Uric Acid, its Forms and Pathological Relations.*

(a.) *Uric Acid* occurs in various combinations with bases, such as *soda, potash, lime,* or *ammonia,* and more or less coloured with urine-pigment. It is also associated sometimes with phosphate or oxalate of lime. The uric acid sediments in combination with bases are generally associated with increased acidity of the urine, either by normal acids, such as uric, sulphuric, phosphoric, hippuric, or with the formation of acids developed after emission. They are known as "urates," and commonly spoken of as yellow lateritious "brick dust," or "fever sediments." They dissolve when the urine is heated to 130° Fahr. They are also soluble in potash and liquor ammonia. Acids decompose them after warmth, and liberate uric acid. Three forms are distinguishable —(1.) Irregularly formed or amorphous particles; (2.) Round globules of various sizes; (3.) Fine acicular prismatic crystals. These forms may deposit at *two* different periods after emission—(1.) As soon as the urine has cooled down to the temperature of the atmosphere. In such cases the water is diminished—the urine being that known as febrile urine;

or it occurs when there is an absolute increase of uric acid. (2.) They deposit some hours after the urine has been passed, and long after the urine has been of equal temperature with the air. Under these circumstances the deposit betokens increased acidity from changes in the pigment or extractives, or uroxanthin. The acids so formed may be lactic, acetic, or butyric; and a drop of acid added to such urine will anticipate the deposit.

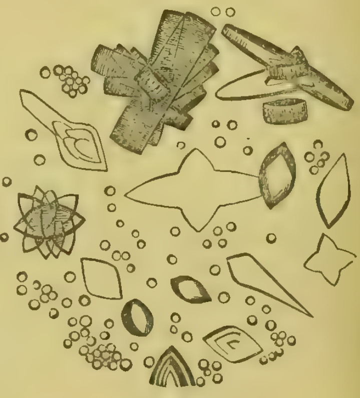

Fig. 64.*

(b.) *Uric Acid Sediments* in their pure state occur in the form of rhombic prisms, or rhombic plates, or of thin hexagonal plates like cystin; but the most usual forms are referable to some variety of the rhomb (fig. 64). Such deposits often present the aspect of a granular sand of a golden lustre, sometimes mingled with blood-discs; and generally the deeper the colour of the urine the darker is the uric acid sediment. Its appearance does not necessarily indicate that an excess of uric acid is forming in the body. The urine is generally yellow and transparent, and the acid is deposited slowly without admixture of urates. Liquor potassæ, and also nitric acid in excess, dissolve uric acid.

Pathological Relations:—*Lithuria*, or *Lithic*, or *Uric Acid Diathesis*, are names which have been given to that general constitutional state in which the urates are secreted in such excess as to be deposited in inordinate quantities in the chamber-vessel on the urine cooling; or, when in still greater abundance, deposited in an amorphous or crystallized state, either in the cavities of the kidneys or bladder. The specific gravity of such urine varies from 1,015 to 1,035, it always gives an acid re-action, and is of a deep copper or red colour.

Uric acid deposits not only occur when the patient is in his best health; but in many persons who have died while labouring under this constitutional state the kidney has been found healthy. On the other hand the kidney has been found occasionally studded all over with crystals of uric acid in the tubes, especially in children. Uric acid diathesis, however, may co-exist with most forms of disease of the kidneys, ureters, or bladder. It is by no means uncommon to find a calculus of this substance, the nucleus of a mulberry, or other calculus, formed in the tubuli or pelvis of the kidney, and sometimes in the bladder.

The colour of uric acid crystals and sediments of the urates varies greatly. The pure lithate of ammonia is white; but, owing to the presence of the colouring matter in the urine, it is usually deposited of a yellow or wood colour. The purpurates, owing either to the nature of the colouring matter of the urine, or to other circumstances not yet determined, are of a pink, light red, or brick colour. These different salts may be deposited in a crystallized or in an amorphous state, or as small calculi.

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* The most usual forms of uric acid sediment, with blood-corpuscles intermixed after DR. OTTO FUNKE).

The lithic acid which exists in healthy urine in such a state and in such proportions as to be held in solution at ordinary temperatures, in certain conditions of the system may be precipitated from that secretion in a *crystallized* and nearly pure state.

The *amorphous* and impalpable lithic acid sediments consist in general of lithic acid in combination with ammonia, and only in a very few instances with soda. The sedimentary deposits of the lithates of ammonia may be white, yellow, pink, or red; while the sedimentary deposits of the lithates of soda are white. The crystallized salts form gravel. The yellow amorphous sediments may concrete in the bladder or kidney, and form calculi.

In general the lithates and purpurates, whether in a crystalline form or as an amorphous sediment, even when in great excess, are held in solution at the temperature of the body; but in some instances their superabundance is so great that they are deposited, even at this temperature, either within the bladder or kidney, so that the last portions of urine are so loaded with them as to resemble a stream of blood. If the excess be yet greater, or if a nucleus be present, a concretion may form either in the kidney or bladder, but infinitely more commonly in the former. The nucleus may be either a piece of hardened mucus, or a portion of fibrine or other substance, or it may be a crystal of lithic acid.

The lithates, however, are very frequently only the nucleus of a calculus of different formation, as the phosphates or oxalates. Indeed we not unfrequently see the lithates, the phosphates, and the oxalates deposited in alternate layers in the same calculus, thus affording absolute demonstration of three or four different conditions of the constitution having prevailed during the formation of the same calculus.

Persons labouring under idiopathic uric acid diathesis are in other respects generally healthy, and the remote cause is for the most part referred to errors in diet, and to sedentary habits. A too full animal diet, as rich old black meats and game, are among the most frequent. When the predisposition, however, to the diathesis is great, every substance, even the most opposite, *that causes indigestion* will produce it, as a heavy dumpling or new bread, the richer sorts of fish or salted meats, acid fruits, or saccharine matters. Wines which are so-called "heavy bodied," *i. e.*, full of sugar and solids not yet decomposed by the vinous fermentation, and malt liquors, are still worse, from the rapidity with which they ferment and turn acid.

Besides being the result of many errors in diet, a deposit of the lithates is incident to many diseases, as gout and rheumatism. The appearance of urates is also often a critical termination and first faint indication of recovery from fever, or severe forms of inflammation. It also results from morbid states of the liver. Besides denoting remote diseases, it sometimes results from an irritable state of the bladder, or from stone in the kidney or bladder.

The effects of diet are so marked in children, that we can hardly feel surprised that any error of diet, as overfeeding them, should be followed by lithic acid deposits. Stone cases are consequently common in children, and occur chiefly among those of the poorer class, in whom those errors are likely to be most considerable. When stone forms in childhood the ages most affected are between four and nine years. Of 506 children operated on at the Norwich Hospital, 223 were under 12 years of age, while 271 were between 14 and 15. Two-thirds of all the cases of stone result from an uric acid diathesis. After these periods the ages of 40 and upwards present the greater number of cases of gravel, either because the frame then begins to break, or that increasing age enables us to enjoy the pleasure of the table as well as leads to a more sedentary life. Majendie has assigned as a cause of these morbid states of urine in the extreme of life that the temperature of the body is from one to two degrees below the healthy standard of the adult.

In many instances this disease appears to be hereditary, and those attacked are usually of sthenic constitution.

Symptoms.—The fact of lithic acid being in excess is palpable enough, from the yellow, red, or pink deposit in the chamber-vessel as the urine cools; and when this is moderate in quantity the patient, perhaps, suffers neither local nor general inconvenience; indeed many persons are never better than when they are passing an excess of the lithates. When, however, it is deposited as an amorphous sediment in the bladder, the last portions of urine are so loaded with it that the patient apprehends he is passing blood. In this case, in the first instance, he is only troubled with itching and pain in the urethra in making water; but if the disease becomes chronic the bladder becomes irritable, the urine loaded with mucus, the healthy sympathy between the bladder and prostate is destroyed, so that the urine is only passed after great forcing, and in trifling quantity, and his sufferings are singularly painful and severe. The secretion of a great excess of the lithates is seldom a purely local disease, but is accompanied by some more general affection, as asthma, palpitation of the heart, rheumatism, or gout; and during its continuance these diseases often, in a great measure, subside.

Although the passage of an amorphous sediment, unless it be in such quantity as absolutely to obstruct the passage, is seldom productive of much local inconvenience, unless it be of long continuance; yet when the lithic acid crystallizes so as to form gravel, or a still larger concretion, the expulsion of this foreign body is always attended with much pain, and gives rise to what has been termed nephritic colic (*colica nephritica*), similar to the biliary or hepatic colic which attends the passage of gall-stones.

The passage of a calculus from the kidney into the bladder may be preceded by dull pains in the back and some sickness; but more commonly the attack is sudden, and the patient, perhaps, in his best health, and engaged in the ordinary transactions of life, is on the instant seized with excruciating agony in the loins, with retractation of the testicle, irritation of the bladder, and often with nausea and vomiting; but in all this suffering the pulse retains its healthy frequency, and the heat of the body is natural. At length the pain intermits, and the patient has a short interval of ease. The paroxysm, however, returns more or less frequently, till the patient is relieved as by a charm, the calculus having passed into the bladder. Again, after an uncertain interval, the gravel or calculus becomes impacted in the neck of the bladder, when the same phenomena present themselves, but have a different locality, as the urethra, till at last, after an effort to pass water, the noise of a stone falling into the chamber-vessel is heard, as the sound of most pleasant music to the ear, and the gravel or calculus is found.

The duration of this fit is various, lasting perhaps from one hour to many, and sometimes continuing for many days. Occasionally, however, the calculus has acquired so great a magnitude that it becomes impacted in the ureters, and death has ensued from this cause. In general, calculi pass from one kidney only at a time, but sometimes they pass simultaneously from both kidneys; and should they be large, or the passage long, an entire suppression of urine has been the consequence, and the patient may die from that cause.

Sometimes the calculus so rapidly increases by sedimentary deposits that it is detained altogether in the kidney, when it not only takes the form of the pelvis of the kidney, but branches out in every direction like a piece of ginger. In a few instances, a calculus thus formed in the kidney may lie latent and cause little inconvenience to the patient. More commonly, however, the calculus acts as a foreign body, and the kidney becomes the seat of abscess or other disorganization, and the patient suffers immensely with pains in the back, irritability of the bladder, aggravated by the frequent discharge of pus, of blood, or mucus. Existence under these circumstances becomes a burthen; and death at length terminates the patient's ceaseless sufferings.

A calculus having passed into the bladder sometimes increases so fast that it acquires a magnitude too great to escape by the urethra, and in this case a stone in the bladder is formed. This condition, as it necessarily requires an operation, will be found treated of in the several text-books on surgery.

Diagnosis.—The red lithates can alone be confounded with blood, and from which, when intense in colour, they often can with difficulty be distinguished. On cooling, however, the subsidence of an impalpable or gritty deposit, the presence generally of much mucus, the absence of fibrine, and also of albumen, when the urine is treated by nitric acid, enable us readily to distinguish them. The white or light-coloured lithates are distinguished from the phosphates by the urine being acid, by the absence of the abundant mucous discharge which always accompanies a large deposition of the phosphates, and from the urine not becoming alkaline or fœtid if kept for a few hours. The precipitated lithates also are readily dissolvable by heat, which the phosphates are not.

Prognosis.—While the deposits are yet but sedimentary, the prognosis is always favourable, however large the quantity discharged. Even when gravel or small calculi are formed, the chances are very many that it will be discharged before it attains any considerable size. When the calculus is so large, however, as to be retained in the kidney or ureter, the disease is necessarily fatal. Also, when retained in the bladder, nothing but a surgical operation can remove it; and consequently the chances resolve themselves into the proportionate numbers which recover or die after the operation of lithotrity or lithotomy at the age and under the circumstances of the patient.

Treatment.—The medical treatment of the lithic acid diathesis is by alkalies or neutral salts, turpentine, and saccharine matters.

The celebrated Morgagni suffered greatly from lithic acid gravel, and his remedy was half a drachm of *carbonate of potash* night and morning, gradually increasing the dose till he took three drachms during the day. "The acid of his urine," he states, "soon became saturated, the pain in his loins diminished, his urine became less loaded, and potash was at length found in that fluid in excess." He also adds, "I have repeated this remedy as often as I have been threatened with an attack, and always with success." The particular salt, however, is not perhaps of great moment. Much benefit may be derived from the *citrate of potash*, or the *common effervescing draught*. When the patient's bowels require a more active agent, the *sulphate of magnesia*, the *sulphate of soda*, or the *iodide* or *bromide of potash* may be substituted.

The pure alkalies, from the much smaller doses in which they can only be administered, are much less beneficial than the neutral salts. *Magnesia* also, in Mr. Brande's experiments, produced much less marked effects on the urine than either the *subcarbonate of potash* or *soda*, while *lime-water* produced no very sensible effect whatever. *Phosphate of ammonia*, in doses of ʒi. daily, largely diluted, is of service; and Dr. Garrod reports favourably of it in chronic conditions of the gouty habit.

Colchicum wine (♏xv.) twice or thrice a day, followed by a saline aperient, such as Pullna water, and from forty to fifty minims now and then in the morning, is a mode of treatment often attended with advantage.

Dr. Garrod also calls attention to the value of the *salts of lithia* in uric acid formation, associated with gout, and to the value of the Baden Baden and Bath mineral waters, both of which contain *lithium*. The *citrate of lithia* may be given in doses of five to ten grains, and the *carbonate* in three to six grains.

Frequent use of the Turkish bath is of great service.

Besides *alkalies*, *turpentine* has some character in the cure of the lithic acid diathesis. The celebrated Dutch drops are supposed to be principally composed of *sp. terebinthinæ* and of *tinct. opii*, coloured by some colouring matter. Dr. Henry gives two cases of the beneficial effects of this remedy. One of them was a lady, who, when threatened with an attack, always had recourse to it; and the uniform effect was the discharge of a sandy substance in such quantities that often four ounces of gravel were discharged in two or three days.

The above-mentioned treatment is often successful; but there are cases in which it fails, and the patient continues to be tormented for months, with little relief. In these instances, the *inf. diosmæ* or the *pulv. uva ursi* may be tried, combined with some mild opiate, which latter substance always gives relief.

If the urinary sediment should concrete into gravel, an attack of nephritic colic may take place. The treatment of this attack is the same as for the passage of a gall-stone—as the *warm bath, mild purgatives,* and *opiates.*

If the calculus, having escaped from the kidney, is retained in the bladder, an operation for its removal is necessary, and the case then becomes purely surgical.

Dietetic Treatment.—The dietetic treatment is of the greatest importance in the cure of the lithic acid diathesis. The experiments of Wollaston and Vauquelin have shown that in proportion as animals are fed on animal diet or on azoted substances, their urine becomes more and more loaded with lithic acid. While Majendie has shown by a counter-proof, that if a dog be fed on non-azoted substances, as sugar, every trace of lithic acid disappears from the urine. A lady at Paris, suffering from gravel, having heard of Majendie's experiments, made trial of sugar on herself, eating more than a pound daily. She persevered in this regimen for six weeks, when her gravel disappeared. She now returned to her old regimen, and at the end of three months her fits of gravel returned.

It is plain that of all diets the quantity of animal food should be reduced. It is necessary also that Port as well as French wines should be abandoned, as well as all those things which, according to the idiosyncrasy of the patient, are likely to produce indigestion or acidity of the stomach. The patient also should be warmly clad, rise early, and take a considerable amount of exercise.

(2.) *Sediments of Hippuric Acid.*

Owing to the solubility of this acid its sediments are rare. When it does occur, it is in the shape of long four-sided, acuminated prisms, or acicular needles fixed on uric acid crystals, with which they are sometimes confounded, as well as with phosphates. They are distinguished from phosphates by being insoluble in acids; and from uric acid they may be separated by boiling with strong alcohol (PARKES).

(3.) *Phosphoric Acid, its Forms and Pathological Relations.*

(a.) *Sediments containing phosphoric acid* are formed of the *ammoniaco-magnesian phosphate,* the *phosphate of lime,* and the *phosphate of magnesia.* The ammoniaco-magnesian phosphate occurs in the form of beautiful transparent prisms, or in foliaceous, penniform, or stellar prisms or crystals.

(b.) *The phosphate of lime* and the *phosphate of magnesia* form a white amorphous powder, or they occur as round small globules, or as prismatic crystals with oblique summits (HASSALL).

Acids dissolve all those sediments; heat has no effect upon them.

(c.) *The ammoniaco-magnesian phosphate* is almost always deposited as a result of the decomposition of urea, and the deposit usually commences on the surface of the fluid, where the urea is most exposed to the air.

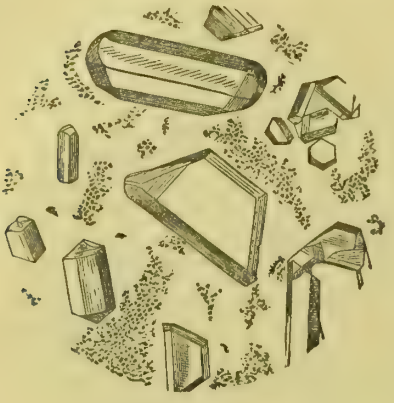

Fig. 65.*

The *phosphate of lime* and the *phosphate of ammonia* are thrown down if the urine becomes alkaline from fixed alkali, as after vegetable food or the carbonates of the fixed alkali, or after the salts which form them have been taken. Decomposition of the urea in these cases is generally rapid, and *ammoniaco-magnesian* phosphate is also produced. Such also is the case with urine of chronic diseases, or during convalescence from acute diseases, when the urine is feebly acid. The phosphates and urates occur together under the following conditions:—(1.) When urates having been deposited, the urea decomposes to a slight extent—enough to form the ammoniaco-phosphate, and yet not enough to dissolve the urate; (2.) When crystallized uric acid has formed and been acted upon by the ammonia formed from the urea, the crystals of uric acid disappear, and are replaced by round globules of *urate of ammonia*, mixed with the precipitate of phosphates. These deposits are always white, and unchanged by heat; soluble in dilute hydrochloric acid, but insoluble in ammonia and in liquor potassæ. Mucus, pus, or blood may mask the chemical re-actions. A small quantity of solution of sesquicarbonate of ammonia, added to a large quantity of healthy urine, will yield prisms of the triple phosphate.

Pathological Relations.—*Ceramuria or Phosphatic Diathesis* are names which have been given to that general state of the body in which phosphates tend to appear in the urine. The phosphates are secreted in a state of health in the proportion of one part in 1,000 of urine. When this proportion is abnormally increased, so that they are largely deposited either in the kidney or bladder, or even in the chamber-vessel, the condition has been termed the *phosphatic diathesis.*

The remote causes of this constitutional state are, exposure to cold and wet, poor diet, blows on the back which injure the spinal marrow, and more especially diseases of the bladder, as *vesical catarrh, stone in the bladder*, the introduction of a bougie, or other irritating cause.

This form of disease sometimes occurs in children, but more commonly in adults between thirty and forty years of age. It affects both sexes, but more commonly the male than the female. Those affected are usually of an asthenic, pale, leucophlegmatic temperament, and in some instances are believed to have inherited it.

Though often resulting from disease of the bladder or kidney, yet occasionally it exists when no such disease is present. It is the prevalence of this diathesis that causes those large calculi sometimes found in the kidney or bladder.

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* Ammoniaco-magnesian phosphate in prisms, mixed with amorphous granules, phosphate of lime, and granular urates (after DR. THUDICHUM).

These phosphatic salts, like the super-lithates, have the property of reddening vegetable blues, and they show an acid re-action. When the earthy bases of lime or magnesia are from any cause secreted in greater abundance than natural, they combine with the phosphates, which are thrown down in the form of insoluble phosphates, and which may be deposited either as a sediment on the urine cooling, or in the bladder or kidney before being passed, or, being retained in those cavities, may concrete into a stone or calculus.

Symptoms.—When this diathesis is a primary affection, the patient is usually of a sallow complexion, stout, but effeminate, and of great irritability of nerve. He also suffers from indigestion, flatulence, constipated or disordered bowels, his stools being either black or clay-coloured. His bladder also is highly irritable; he has pain in his back and loins; his urine is abundant and loaded with mucus, together with a copious white sediment, so that the latter portions of urine pass like so much milk.

The duration of this condition is often very long, on account of the diseased state of the bladder, with which it is connected. In cases, however, in which that viscus is healthy, it often readily yields to medical treatment; but in other instances, when all appears to be proceeding favourably, the lungs become affected, and the patient dies of phthisis.

The urine, when examined, is pale, increased in quantity, often turbid, and covered with an iridescent pellicle or film, consisting of a solution of the triple phosphate of ammonia and magnesia; much mucus is also deposited, together with a most copious precipitate of the phosphates, so that sometimes the urine appears like so much chalk and water. It is generally of low specific gravity, or 1,001, 1,002, or 1,003.

As the phosphates have little tendency to crystallize, a nucleus is necessary before the sediment can concrete into calculi; and it is in this form of disease that we find such singular substances in their centres, as a clot of blood, a piece of hardened mucus, broken ends of sounds or bougies, bits of straw, bodkins, pins, plum-stones, beans, nut-shells, and bullets.

The calculi which form on these nuclei are of three descriptions, and in the following proportions:—Out of 102 calculi examined there were of—

Phosphate of Lime, nearly pure, ...	8
Triple Phosphate, or Phosphate of Ammonia and Magnesia, . .	3
Mixed or Fusible Calculi, being a mixture of the two preceding, .	91
	102

These calculi are distinguished from all other calculi in being soluble in an excess of phosphoric acid. They are distinguished from one another by the phosphate of lime calculus being nearly infusible, by the mixed being readily fusible, while the triple phosphate is known by the minute crystals which often form between the interstices of the laminæ.

Every other form of calculus, whether the lithate or oxalate, from the irritation it occasions, constantly produces a deposit of a soft coating of the phosphates. When, however, the phosphatic deposition is once well established, it is seldom followed by the deposition of strata of any other description. Thus, of 823 calculi examined by Dr. Prout, he found only three specimens in which the phosphates had been followed or surrounded by other calculous deposits.

The physical characters of the phosphatic calculi are, that they are white, soft, and easily broken down, and are deposited in concentric laminæ like the lithic acid concretions.

Diagnosis.—The phosphatic sediments may be distinguished from the lithic by the urine, though at first acid, becoming putrescent, and giving an alkaline re-action after standing a few hours. Ammonia also added to the urine throws down a white cloud, which consists of the phosphate of lime with some of the ammoniaco-magnesian phosphates, a test which would render the lithates soluble. The best test, however, is the addition of phosphoric acid, which would re-dissolve the precipitate.

Prognosis.—When this diathesis is unaccompanied by disease of the bladder the prognosis is always favourable. When, however, it results from a morbid state of the bladder or diseased structure of the kidney, the disease is always of long duration.

Treatment.—The treatment of this affection is by some mineral acid combined with an opiate. The acid may be the *nitric*, *muriatic*, the *phosphatic*, or the *sulphuric;* but the *dilute sulphuric acid* is generally preferred, as being most pleasant to the taste. A most usual prescription is the *infusion of roses*, with an addition of ♏ v. to ♏ x. of *dilute sulphuric acid*, together with *tinct. opii*, ♏iij. to ♏ v. every six hours. This combination is generally efficient in checking the formation of phosphates, and the *sulphate of magnesia* may be added to it if the bowels should require to be regulated. *Benzoic acid* has been found of great use (gr. x. to xv.) in a mucilaginous draught. It may be given in still larger doses—twenty to forty-four times a day (GARROD).

When the phosphatic diathesis depends on vesical catarrh, or other diseased state of the bladder, *salicinæ* gr. x. thrice daily has occasionally been found successful. Others prefer the *inf. diosmæ*, and others *uva ursi*. The pure alkalies in these cases should not be used.

Dietetic Treatment.—The diet should be as nourishing as the state of the diseased viscus will allow; and acid, wines, and ripe fruits greatly assist in effecting the cure.

(4.) *Oxalate of Lime, its Forms and Pathological Relations.*

Opinion is very undecided as to the significance of deposits or *sediments composed of oxalate of lime*. It occurs in one out of every three cases, taking the cases indifferently as they occur in an hospital. It appears (fig. 66) in four forms:—(1.) As octahedra (*a*); (2.) As hourglass, contracted, or dumb-bell-like bodies (*b*); and (3.) Compound octahedra (*c*.) may also be seen; (4.) As small, flattened, bright discs, very readily mistaken for blood-discs.

Oxalate of lime, although found in the blood, is probably a result of chemical changes in the renal passages. The octahedra grow or increase in urine after it is passed; but the dumb-bell crystals are said not to do so. Dr. Beale considers that the dumb-bell crystals of oxalate of lime are mainly formed in the kidney-tubes. He found them most frequently in casts. Dr. Parkes considers that the oxalic acid of urine most probably results from uric acid; and may be a substitution for the excretion of carbonic acid of the lungs (Schmidt). In order that oxalic acid shall form in the urine, there must either be irritation from a calculus, or from some other cause, or there must be fermentive changes from mucus. If there be no evidence of any of these conditions, the oxalic acid of fresh urine may be presumed to come from the blood (Parkes). Crystals of oxalate of lime are insoluble in water—are unaltered by boiling either in *acetic acid* or in *liquor potassæ;* and are soluble in nitric acid without effervescence.

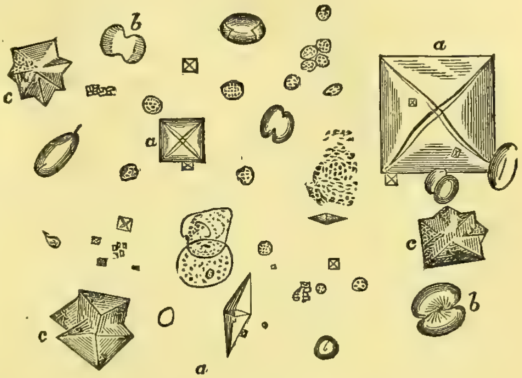

Fig. 66.*

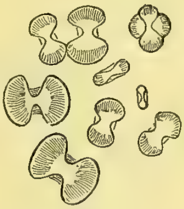

Fig. 67.†

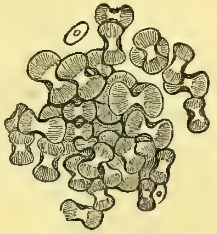

Fig. 68.‡

Pathological Relations.—Many articles of diet contain oxalic acid in abundance; but there are also certain articles of food and drink the use of which, when digestion is deranged, is followed by the formation of oxalate of lime and its presence in the urine. Sugar in excess, the use of frothy sparkling beer or wine, turnips, parsnips, carrots, cauliflower, asparagus, may all cause a "temporary oxaluria" (Prout, Robin, and Verdeil, Rose, Bird, Hassall). When the respiratory functions are impeded, oxalates appear in the urine, and during convalescence from some severe diseases as *typhus fever.*

Oxaluria is the name given to that general state of the constitution in which oxalate of lime is secreted, as first determined by Dr. Wollaston.

The remote cause of this constitutional condition is not determined, for persons who seem in the best health will often void oxalate of lime in the urine. It is supposed, however, to be most frequent among those who eat largely of common sorrel (*rumex acetosa*), or of tomata (*solanum lycopersicum*), and of the leaf stalks of the rhubarb plant, all of which many persons are passionately fond, and all of which contain oxalic acid.

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* (*a.*) Octahedra; (*b.*) Dumb-bell; (*c.*) Compound Octahedra; circular and oval crystals of oxalate of lime (after Dr. Beale, l. c.)

† Dumb-bell crystals of oxalate of lime, of very regular form, from the urine of a child two years of age, suffering from jaundice (after Dr. Beale, l. c., p. 300).

‡ Collection of dumb-bells, such as often forms the nucleus of a calculus (after Dr. Beale, l. c., p. 300).

This form of disease may exist before puberty, and from that period till sixty. It is most usual, however, between forty and fifty. It is common in both sexes, and is not incompatible with gout.

The oxalate of lime very rarely appears under the form of an amorphous sediment; still it has occurred mixed with the lithic amorphous sediments, but even this is uncommon. Its appearance is still more rare under the form of crystallized gravel, so much so that Dr. Prout mentions only two instances. Renal calculi of this formation are not very uncommon. When detained in the bladder they often acquire a considerable size, are rugged, dark-coloured, and tuberculated, and from these appearances have been termed the "mulberry calculi." Oxalate of lime enters as a constituent part into about one-fourth of all the calculi examined. The following table will show the different transitions:

Oxalate of Lime,	113
Lithic and Mulberry,	15
Mulberry and Lithic,	40
Mulberry and Phosphates,	49
Fusible and Mulberry,	2
	219

When heated before the blow-pipe the oxalic acid is decomposed, and pure lime remains, which gives a strong brown stain to moistened turmeric paper. This calculus is insoluble in the alkalies, but by digestion in carbonate of potash it is decomposed, and the insoluble carbonate of lime is left. When reduced to powder and digested in nitric or muriatic acids a perfect solution is effected. It is not dissolved by acetic acid—a circumstance which distinguishes it from the ammoniaco-magnesian phosphate. It is distinguished from the phosphate of lime by being insoluble in phosphoric acid.

Symptoms.—Oxaluria is attended with no prominent feature. The urine, which contains this substance, is acid, of a good colour and remarkably pure, and free from all sorts of sediment as well as gravel. The patient is therefore hardly sensible of any inconvenience till he is attacked by a fit of nephritic colic, caused by the passage of the calculus from the kidney or bladder, or till he is troubled, supposing it to be retained, by symptoms of stone in the bladder or kidney.

Treatment.—Respecting the medical treatment of oxaluria, Dr. Prout recommends that we should try to induce a lithic acid diathesis; but it must be questionable whether the disease substituted is not as dangerous as the one under which the patient originally laboured. He speaks, however, of having seen much advantage derived from *mineral* acids and the *sulphates of iron* or of *quina*. The mineral acids are useful in correcting the dyspepsia associated with the elimination of oxalates. *Henbane* is considered by Dr. Harley as a most valuable remedy. Water containing lime should be avoided, and distilled water used instead; and in many cases it will be necessary to empty the intestines by suitable purgatives.

Dietetic Treatment.—The patient should carefully avoid eating all substances containing oxalic acid. A gentleman who had lived as a *bon-vivant*, determined to reform his diet, but to render his new dishes more palatable, he ate every day a plateful of sorrel, and was attacked with an oxalate of lime calculus.

(5.) *Leucine, its Forms and Pathological Relations.*

Sediments of Leucine are precipitated from the urine in round corpuscles, sometimes with a concentric form, and look like heaps of fat; but when crystallized from pure solutions, it appears as fine, dark-coloured needle-like crystals. To recognize it fully, it must be separated by careful sublimation (PARKES). The suspected leucine is to be placed on platinum, carefully moistened, and then dried with nitric acid. The almost imperceptible flake which is left is to be moistened with caustic soda, and evaporated carefully over a spirit-lamp. If leucine is present, it forms an oily-looking drop (SCHERER).

Pathological Relations.—It is believed to be the result of the disintegration of certain of the nitrogenous tissues of the principal glands, and occurs particularly in *acute yellow atrophy* of the liver, where it is found in the substance of the liver itself.

(6.) *Tyrosine, its Forms and Pathological Relations.*

Sediments of Tyrosine are of a greenish-yellow colour, composed of heaps of fine needles, which can be obtained on evaporation. It ought to be treated with nitric acid, like *leucine*, and then a little *liquor soda* used. The nitric acid gives a deep orange-yellow colour, which becomes deep yellow on evaporation. The soda gives the yellow flake a red tinge; and on heat and evaporation, a black-brown residue is left (SCHERER, quoted by PARKES).

Pathological Relations.—It is found wherever leucine is met with.

(7.) *Cystin, its Forms and Pathological Relations.*

Sediments of Cystin form a white or light fawn-colour, amorphous, rather bulky precipitate, or they appear at once as six-sided plates (fig. 69). In both cases ammonia dissolves it,—so do fixed alkalies and their carbonates; and from this solution it crystallizes on spontaneous evaporation. It does not disappear when the urine is gently warmed; and it is insoluble in carbonate of ammonia, in dilute hydrochloric acid, and in acetic acid.

Pathological Relations.—It is associated with the excessive elimination of *sulphur*. Its persist-

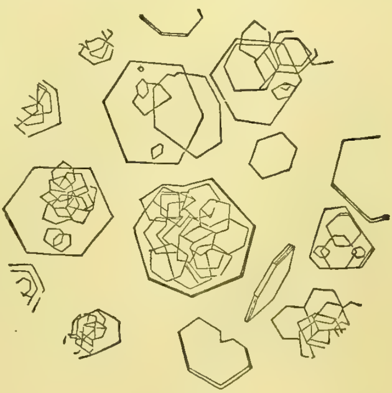

Fig. 69.

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* Cystin precipitated by acetic acid from its ammoniacal solution (after THUDICHUM).

ence in the urine is often hereditary, and is generally associated with derangement of the functions, or organic disease of the liver. It occurs also in cases of chlorosis.

Cystinuria is the name which has been given to the general state of the system in which cystine is eliminated by the urine in abnormal amount. The cystic oxide was described by its discoverer, Dr. Wollaston, in the *Philosophical Transactions* for 1810; and from the similarity which this substance bears to certain oxides in uniting both with alkalies and acids, Dr. Wollaston termed it an oxyde, and gave it the name of cystic oxyde, on the supposition of its being peculiar to the bladder. Dr. Marcet, however, has found it in the kidney.

The first calculus examined by Dr. Wollaston of this description was taken from a boy five years old. It has been found also in the adult; and Professor Stromeyer found it in two instances in one family.

An analysis of a cystic oxide concretion by Lassaigne gives—

Carbon, ...	36·2	
Hydrogen, ...	12·8	
Oxygen, ...	17	
Nitrogen, ...	34	
		——100

Its formation as a concretion appears to result from a general constitutional state; and it has been discovered in the urine in a state of solution, of mechanical suspension, and also in the solid form of a calculus, either pure or incrusted with the phosphates or lithates. The concretion, when pure, is not laminated, but appears as one uniform mass confusedly crystallized through its whole substance, having somewhat of the appearance of the ammoniaco-magnesian phosphate, though more compact. Before the blow-pipe it emits a peculiarly fœtid smell, quite distinct from that of uric acid, and is consumed. It is characterized by the great variety of re-agents in which it is soluble. It is dissolved abundantly by the *muriatic, nitric, sulphuric,* and *oxalic acids;* by *potash, soda,* and *ammonia,* and even by the neutral carbonates of *soda* and *potash.* It is insoluble in *water, alcohol, bicarbonate of ammonia,* and in the *tartaric, citric,* and *acetic acids.* The urine in which this substance has been found was copious, of a yellowish-green, of a strong peculiar smell, and of a low specific gravity; it was entirely free from uric acid, and the urea deficient in quantity. This diathesis is of unfavourable prognosis, and its mode of treatment not yet determined. Dr. Prout has seen most benefit from the *nitro-muriatic acid.* The peculiar smell of the urine abates under its use; but the complaint shows a great disposition to return when the medicine is left off.

CLASS III.—SEDIMENTS COMPOSED OF SUBSTANCES FOREIGN TO THE URINE, AND WHICH ACCUMULATE IN THE URINE ALWAYS AFTER EXPOSURE TO THE ATMOSPHERE.

1. *Fungi* in acid urine—*e. g.*, the *Penicillum glaucum,* its *spores, thullus,* and *fructification.*

2. The *Torrulœ cerevisœ* in saccharine urine begins to form in two or

three hours after emission. It forms a gelatinous mass, composed of *sporules*, which subsequently develop into *beaded threads*, and in a few days aërial fructification appears.

3. *Vibriones* and *Monads* occur in urine containing much mucus, and which is feebly acid or alkaline.

All urine should be collected in glass vessels, which are scrupulously clean; and *the quantity operated upon should embrace the whole urine passed during the twenty-four hours.* All sediments should be examined—(1.) Within a few hours after excretion; (2.) After twenty-four hours.

Of the substances dissolved in the urine, or becoming obvious only under special conditions, there are some which require more special notice, namely:—

1. *Hœmatine*, or *Blood-pigment*, occurs either as blood-corpuscles or separated from them. The corpuscles fall to the bottom of the tube or glass, and form a sediment. If the hæmatine is separate from the corpuscles, it constitutes a pigment completely dissolved in the urine, and to which it gives a brown or black colour (see scale of colour). It is always accompanied by albumen. Urine of a brown or black colour, due to blood-pigment dissolved in the urine, is to be determined by the elimination process in part—bile and vegetable pigments being absent.

Urine containing blood in obvious quantity permits it generally to coagulate into blackish masses like pieces of black currant jelly; and when it partly coagulates in the bladder, linear masses of clot, of nearly the shape of leeches, are passed from the urethra. The urine has a port-wine hue, and abundance of entire red discs of blood-corpuscles may be detected with the microscope. These sink with readiness to the bottom of the vessel. Their non-granular surface, uniform size, and yellow colour, are characters sufficient to identify them.

Blood-pigment, or hæmatine, dissolved in the urine, does not necessarily indicate local disease or rupture of vessels in part of the urinary organs. It must be regarded as indicating rather a specially morbid condition of the blood, as is associated with septic poisons, or with profound cachectic diseases (PARKES). It may be observed in *typhus fever*, *malignant variola*, *remittent fever*, *yellow fever*, *scurvy*, and *Bright's disease.* Albumen is said always to co-exist.

2. *Albumen.*—Its presence must be regarded as an indication of disease, although it has been observed to occur in small quantity, and temporarily, in the urine of some persons supposed to be at the time in a healthy state; although it is more than probable that slight disease is present, or that such grave affections as Bright's disease may be commencing, to terminate fatally perhaps two years later (Christison, *Monthly Journal*, Jan., 1851; PARKES, p. 184). Very slight disease—not more than what appears to be dyspepsia—may have albuminuria associated with it, of a temporary nature. It has been observed in *intermittent fever*, *typhoid fever*, *rubeola*, *variola*, *pneumonia*, *pleuro-pneumonia*, *scarlatina*, *bronchitis*, *pleurisy*, *peritonitis*, *intestinal catarrh*, *follicular enteritis*, *acute rheumatism*, *chlorosis*, *cardiac diseases*, *phthisis* (SOLON, FINGER, PARKES).

Hæmaturia has been found associated with albuminuria in *scarlatina*, *rheumatism*, *arthritis*, *typhus* (during the second week), *nephritis* (*calculous?*), *pneumonia*, *erysipelas*, *torpor*, *hepatitis*, *peritonitis*, *phthisis*, *chlorosis*, *intense* *oxaluria, phosphatic diathesis, last month of pregnancy, uterine disease, bladder-cancer, internal use of turpentine* (Heywood Thomson, *Lancet*, July, 1857; Parkes).

Dr. Parkes desires to draw a strong distinction between "temporary" and "permanent" albuminuria. The former ought to imply that the albuminuria, after lasting for some days, or even weeks, finally disappears entirely. The latter implies that albuminuria does not disappear. Dr. Parkes found temporary albuminuria, with the quantity of albumen large, in acute lobar pneumonic cases and acute renal cases, such as Bright's disease. He found albumen considerable in amount in *typhoid, variolous*, and *scarlatinal cases*, and small in amount in *paraplegia (spinal), hemiplegia, chronic phthisis, acute pleurisy, acute bronchitis, in hypertrophy, dilatation, and valvular affections of the heart, in acute and subacute rheumatism, in purpura hæmorrhagica, in typhoid and typhus fever, and in erysipelas* (Parkes, p. 188).

"Permanent albuminuria" he found associated with all forms of *Bright's disease*, with *encephaloid and cystic disease of the kidney*, with *leucocythæmia and presumed lardaceous disease of the kidney, in chronic phthisis, and in pleurisy, where casts and kidney-structures were seen in the urine, in hypertrophy and valvular affections of the heart, in cerebral softening of hemiplegia, in pancreatic disease, in purpura hæmorrhagica, and in typhoid fever.* The result is, that if heart diseases be excluded, "permanent albuminuria" indicates kidney disease invariably, and the distinction between "temporary" and "permanent" albuminuria is apt to be drawn with insufficient care; and in all cases where it occurs there is either congestion and increased lateral pressure on the vessels of the kidney, or there is absolute structural disease of their walls. In the Mauritius, and in Bourbon, a singular hæmaturia sometimes occurs, in which a varicose condition of the lymphatics has been supposed to cause the albumen (Parkes, p. 190).

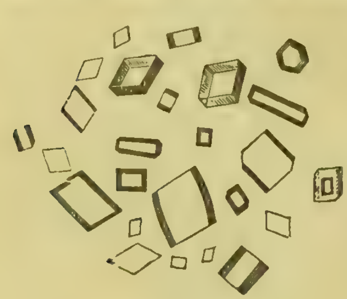

Fig. 70.*

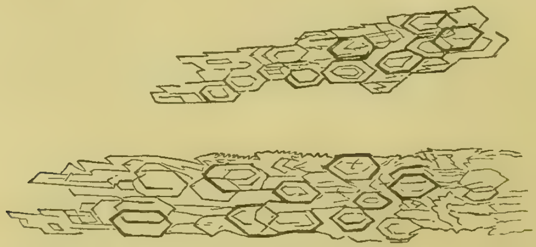

Fig. 71.†

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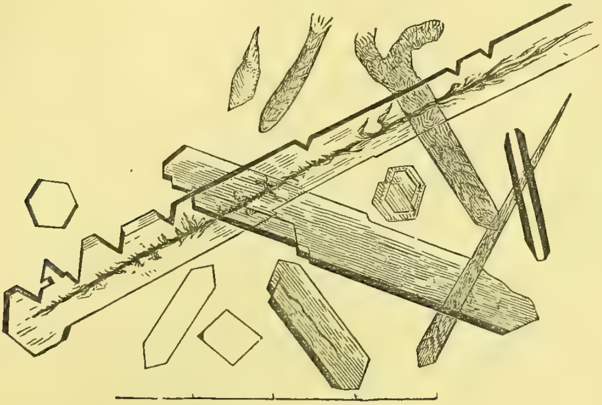
* Oxalate of urea—perfect crystals (after Beale) "Urinary Deposits," No. II., Plate IV.

† Oxalate of urea (after Beale). "Urinary Deposits," No. II., Plate IV.

3. *Urea.*—It is often of importance to determine the mere presence of urea in a small amount of fluid, as in the fluid of the ventricles of the brain. The process has been already explained at p. 1043; and, if in place of nitric acid, oxalic acid is used, we obtain crystals of *oxalate of urea* (figs. 70, 71).

The alcoholic extract of urea leaves, on spontaneous evaporation, acicular crystals of the following form (fig. 72):—



100th of an inch × 42.

Fig. 72.*

SECTION V.—DISEASES OF THE KIDNEY.

BRIGHT'S DISEASE—Syn., ALBUMINURIA.

LATIN EQ., *Morbus Brightii*—Idem valet, *Albuminuria;* FRENCH EQ., *Maladie de Bright*—Syn., *Albuminurie;* GERMAN EQ. *Bright'sche Krankheit*—Syn., *Albuminurie;* ITALIAN EQ., *Malattia di Bright, Albuminuria.*

Definition.—*A generic term, including several forms of acute and chronic disease of the kidney, usually associated with albumen in the urine, and frequently with dropsy, and with various secondary diseases resulting from deterioration of the blood.*

Pathology.—In previous editions of this text-book I considered that there existed a large amount of evidence, sufficient to regard Bright's disease as partaking of the characters of a general or constitutional disease; and I classified it amongst them, with gout and rheumatism. I looked upon it as a constitutional affection culminating in a variety of structural lesions of the kidneys, each of which is accompanied by the persistent separation of more or less albuminous serum from the blood, and by its presence in the urine, the connective tissue, and serous cavities of the body. The urine frequently contains blood, renal structures, exudation material, and desquamative renal products. The kidneys may be either large or small, atrophic or not, with fatty, or albuminous, or lardaceous degeneration, and altered in various ways as regards the vessels, the tubes, and the epithelium. When the constitutional state associated with Bright's disease is fully expressed there is characteristic anæmia, indicated by pallor of the countenance, a puffy face and œdematous limbs, anasarca or general dropsy, pain in the loins, with more or less albumen in the urine. Associated with these phenomena, the morbid states of the kidney may be various. There is scanty urine and frequent micturition; a sensation of heat and scalding on passing water, dryness, heat of skin, general feverishness and occasional chilliness of the surface, an irritable pulse, generally above the normal standard, emaciation, and progressive debility. Dyspeptic symptoms predominate. There is difficulty of breathing and anæmia-chlorosis. The term is generic, including several forms of acute and chronic disease of the kidney, usually associated with albumen in the urine, and frequently with dropsy, and with various secondary diseases resulting from deterioration of the blood.

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* Oxalate of urea from urine—extraction by alcohol, and an oxalate formed by the addition of oxalic acid.

In Bright's disease, as with many others, an undue share of attention has been bestowed upon the prominent local lesions in which the existence of the constitutional state has finally culminated. For example,—the anatomical characters of *tubercle* in *phthisis*—of *cancer growths* in the *cachexia* of *carcinoma*—of the *supra-renal bodies* in *morbus Addisonii*—of the *serous effusions* into the spinal and other visceral cavities in *beriberi*—of the *kidney* in *Bright's disease* and *mellituria*—of the sediments in the urine in *oxaluria, lithiasis,* and the like, have been so prominently dwelt upon that a very narrow view is apt to be taken of the important antecedents of these diseases—of the constitutional states under which they respectively become developed, and therefore, also, of the principles which ought to guide the treatment of these respective diseases, and of the means by which their development may be prevented. A vast amount of evidence has been accumulating since Drs. Wilson Philip, Tweedy Todd, Sir James Clark, Bennett, and Ancell demonstrated how prominently the general health was impaired and forms of dyspepsia prevailed long before scrofula became fully expressed with or without the deposition or growth of tubercle; and this kind of evidence tends to show that not only are constitutional phenomena characteristic of scrofula long antecedent to the expression of the disease by local lesions, but that other diseases, having similar constitutional antecedents, ought to command a more extended study from this point of view than they have hitherto obtained. *Bright's disease* and *diabetes mellitus,* I consider, belong to this group of general or constitutional diseases.

Between 1841 and 1846 Rokitansky wrote that the nature of Bright's disease is the more obscure, because the question is generally evaded. He considers that the cause of the disease is to be sought for in some anomalous constitution of the blood—to something which gives rise to a change in the blood, rather than to any specific disease of the kidney. It is this anomalous condition of the blood which must be regarded as the primary affection in Bright's disease, which, from some peculiar relation to the kidney, is followed by the secondary and visible disorganization of the renal tissue. The general or constitutional origin of the disease is also shown in the fact that both kidneys generally express the local lesion about the same time, or with a brief interval.

In 1849, Dr. Walshe, the Emeritus Professor of Medicine in University College, published a commentary on a clinical lecture, in which he taught that Bright's disease was not essentially a renal disease, but essentially and primarily a blood disease (*Lancet*, July 14, 1849). Since that time he has maintained the same view regarding the pathology of this disease; and there seems to be now an abundance of facts and arguments to establish its correctness. The conclusions at which Dr. Walshe arrived were mainly these:—That from the very commencement Bright's disease is a general disease of the blood; that the renal textural changes are not the cause of the phenomena comprehended under the name of Bright's disease; and that the local lesions which occur in the kidney do not even furnish any measure of the intensity of the characteristic phenomena of Bright's disease; that the textural changes in the kidney are in reality the local expression of the constitutional state, just as tubercle is of tuberculosis; that the primary constitutional and blood changes are probably due to a morbid condition or direction of the primary and secondary digestion processes; and that Bright's disease is thus recognized as one of the typical *diathetic* or *constitutional* diseases.

Some of the more eminent pathologists and discriminating physicians of the day take a similar view of the essential nature of Bright's disease. Mr. Simon writes that it "depends on some humoral derangement of the entire system, and (the subacute inflammation of the kidney) commences as functional excitement, manifested in an act of over-secretion. The *materies morbi* seeks to effect its discharge by means of an increased activity in the secreting functions of the kidney: it stimulates it; and the result of the stimulation is not so much an increase of the watery secretion as it is an augmented cell-growth in the tubules of the gland. This acceleration of function is incompatible with the maturity of the secreted products; the epithelial cells undergo various arrests or modifications of development, and become more or less palpably imbued with evidences of inflammation."

Dr. Johnson teaches the same doctrine. He shows that the morbid states of the kidney found in Bright's disease "are often mere local manifestations of a more general constitutional disorder;" and Sir Thomas Watson concurs with him in the belief that "it is the quality of the blood sent to the kidney, and not its quantity, that determines the renal disease."

The very interesting experiments made by Dr. Parkes (*Med. Times and Gazette*, 1854, p. 394) regarding various phenomena in Bright's disease, tend to strengthen the view of its constitutional development, more especially when studied in connection with the observations and experiments of Lehmann, Bernard, and Pavy in relation to the excretion of a substance by the liver, and the formation of sugar in the blood when that substance comes in contact with albumen. Bernard's researches have shown that the blood which leaves the hepatic vein contains a peculiar substance (saccharine?) which does not exist in the blood brought to the organ by the portal vein; and Pavy, of Guy's Hospital, has demonstrated that the liver does not form sugar, *but a substance that becomes sugar* almost immediately upon contact with albuminous matters. It is this material (as Dr. Carpenter writes) which is especially destined as a *pabulum* or fuel for the combustion process, being usually eliminated from the blood in the form of carbonic acid and water, during its passage through the lungs, so as not to pass into the systemic circulation, unless its quantity be either unusually large or its elimination be interfered with by imperfect respiration. This substance appears to be mainly elaborated by the converting power of the liver, either from materials supplied by the food or from the products of the waste of the system (*Animal Physiology*, Bohn's edition, p. 308). Now, both of these conditions obtain in many cases of Bright's disease, if not in all. The condition of the urine before and after food is a point which has been in a great measure neglected till Dr. Parkes showed its importance in cases of this affection. In many cases of persistent albuminuria with renal casts and anasarca (not dependent on heart disease) the albumen in the urine is augmented after food.

But apart from the albumen excreted, Dr. Parkes found that the solids excreted were greatly increased. A weak, thin, quiescent man gave off daily 981 grains of non-albuminous solids, whose average in health did not exceed 650 to 750 grains. It has usually been considered that in chronic Bright's disease the non-albuminous solids are diminished (CHRISTISON, FRERICHS, JOHNSON); but according to Dr. Parkes this rule has numerous exceptions. To detect the increase of the solids, ALL the urine passed during the twenty-four hours must be collected and examined. It is then found that the urine is of low specific gravity, but so copious that the total amount of solids in it is very great. Such cases are found to be constantly feverish—not so much so as to be detected by the mere heat of skin as felt by the hand; but the thermometer under the axilla will show that the temperature of the body is uniformly more than a degree and a half above the healthy standard of 98° Fahr. Thus the thermometer will delicately indicate and explain how an increased metamorphosis of tissue is going on in such cases; and observing such an increase among the urinary non-albuminous excreta, it may be concluded that they are partly due to increased disintegration of tissue; and, as Simon writes, "the morbid material which thus stimulates the kidney in its struggle for elimination will sometimes consist of products of faulty digestion (*e. g.*, lithates, oxalates); sometimes of matters cast upon the kidney in consequence of suppressed function in other organs (*e. g.*, the skin, the liver); sometimes the mysterious ferment of a fever poison (*e. g.*, typhus, scarlet fever, enteric fever)." This view of the subject and method of investigating cases of Bright's disease, as well as of other wasting diseases, is not sufficiently practised and studied by the student of medicine; and it will be found, as Dr. Parkes has shown, that many chronic cases of Bright's disease are often really slow febrile cases, just as tuberculous cases are. In all cases of Bright's disease, and constitutional diseases generally, much may be learned regarding their nature by examinations of the urinary excretion, and microscopic characters of the sediment, in connection with the temperature of the body, the number of respirations, and the state of the pulse. The following formula may be suggested as a method for daily record of the chemical composition of the whole urine passed during twenty-four hours in such cases; and if chemical and microscopical examinations of the urine are made from day to day, and carried on continuously, the particular morbid state of the kidney associated with Bright's disease may be probably or approximately ascertained:—

DATE.	Quantity in cubic Centimetres.	Specific Gravity.	Solids (in grains).	Urea.	Albumen.	Sulphuric Acid.	Phosphoric Acid.	Extractives.	Remarks.

The albuminous character of the blood after meals was long ago pointed out by Dr. Andrew Buchanan, the Professor of the Institutes of Medicine in Glasgow (*Trans. Phil. Society of Glasgow,* vol. ii.) He found a peculiar state of the blood, amounting to an appearance of leuchæmia, which immediately succeeds digestion in healthy persons, and which seems to contain numerous molecular and corpuscular elements, resembling chyle or lymph, and consisting of fat emulsionized with albumen. To this substance in the blood he gave the name of *pabulin,* and which is still further elaborated in the blood, in the glands, and in the lungs. The earliest notice of milky blood is that in the *Philosophical Transactions,* No. 6, p. 100.

During the processes of digestion in the stomach, albuminous substances are known to undergo extraordinary changes; and during the transit of the blood through the liver, the fibrin and albumen undergo still further modification, so that 30 *per cent.* of the albumen entering the liver in the portal vein disappears in that organ, and is not to be found in the blood of the hepatic vein (LEHMANN). Thus the liver is known to exert an overwhelming influence on every digested aliment which enters it, and the influence which it exercises over albuminous aliments is not the least important (PARKES). Again, if crude albumen is taken and introduced at once into the circulation, as Bernard has proved by experiment, through the jugular vein, it is incapable of assimilation, and is rapidly excreted in the urine, a temporary albuminuria being thus produced. In the very interesting and suggestive work lately published by Dr. Basham, there are cases detailed which illustrate, upon these principles, the constitutional origin of *Bright's disease;* so that, connecting all these observations together—namely, those of Andrew Buchanan, Walshe, Lehmann, Bernard, Parkes, Pavy, Carpenter, and

Basham—may it not be suggested as a topic for further inquiry (and at all events a direction which inquiry should take) as to whether or not substances usually elaborated by the liver are not arrested in Bright's disease, or that the liver is insufficient to use up the albumen carried into the blood, and which therefore comes to be eliminated by the kidney? The inference at any rate now presents itself; and, as Dr. Parkes writes, " May it not be possible that, by some failure in preparation, either by the stomach or liver, albumen enters the blood of the right side of the heart in a still crude state, and in a condition similar to that in which it would have been had it been introduced through the jugular vein? and thus, being unassimilatable, is it not excreted, as in Bernard's experiment, by that ready outlet, the kidney? In support of such a supposition," continues Dr. Parkes, " we have the facts, that many cases of kidney disease seem to be most probably of blood origin, and that among the common antecedents of Bright's disease are circumstances of diet or mode of living which would be likely to impair the processes which should go on in the stomach or liver. In how many cases of Bright's disease have dyspeptic symptoms been present for a long time before renal signs have shown themselves? In how many other cases are signs of liver diseases coincident with the renal signs? In how many *post-mortem* examinations, although no liver disease has been suspected, do we find evidence that for a length of time the structure of this organ has been seriously diseased? In the history of Bright's disease there are many reasons for believing that the nutrition of tissues is early and deeply affected."

The renal engorgement is certainly not the first in the order of morbid change : it is secondary to more remote morbific action, pervading the system throughout. An altered, defective, or contaminated state of the blood is the source, in all probability, of the morbid phenomena which follow. A careful inquiry into the antecedents of patients suffering from Bright's disease will exhibit the predisposing causes of their failing health ; and these will ever be such as exercise a marked influence over the quality of the blood, rather than such as have any special reference to the kidneys. The most frequent and patent of these predisposing causes are habits of intemperance, inducing an alcoholized state of the blood; the various febrile poisons, particularly the scarlatinal; a scrofulous habit of the body, or the tuberculous constitution (BASHAM). We see here a wide field for future exploration, especially in discriminating the forms of dyspepsia or of ill-health which precede these cases, and which are frequently to be seen among the wandering uncertain crowds who frequent the outdoor or dispensary practice of our large city hospitals. In the morbid anatomy of Bright's disease, and diseases generally of constitutional origin, the glandular structure of the stomach and intestines, as investigated by Handfield Jones, Schäpfer, Habershon, and Fox, promises to yield important results in connection with the early history of such cases.

Nomenclature.—The disease has been named after the eminent physician of Guy's Hospital, who, in 1837, first drew the attention of the profession to the connection which he observed to subsist between certain forms of anasarca and lesions in the kidneys. It has of late been most extensively investigated by Christison, Frerichs, Ringer, Walshe, Parkes, Gairdner, Wilks, Goodfellow, Basham, and others. The questions which the subject opens up in pathology are of extreme importance. The disease has been termed "granular degeneration of the kidney" by Sir Robert Christison; but looking to its pathology, and to what is now known regarding the morbid anatomy of the kidney in this disease, perhaps it is better designated by the name of the distinguished physician who first described the more prominent phenomena of its course.

The College of Physicians has not adopted the view which regards Bright's disease as a general or constitutional malady. It is therefore classed under "Diseases of the Urinary System;" and considered under two forms, namely,—(1.) *Acute Bright's disease,* of which the synonyms are "*acute albuminuria,*" "*acute desquamative nephritis,*" "*acute renal dropsy;*" (2.) *Chronic Bright's disease*—Syn., "*chronic albuminuria,*" with the following subdivisions:—(a.) *Granular kidney*—Syn., *contracted granular kidney, chronic desquamative nephritis;* (b.) *Gouty kidney;* (c.) *Fatty kidney;* (d.) *Lardaceous kidney*—Syn., *amyloid disease, waxy disease.*

I.—ACUTE BRIGHT'S DISEASE—*Syn.*, ACUTE ALBUMINURIA; ACUTE DESQAMATIVE NEPHRITIS; ACUTE RENAL DROPSY.

LATIN EQ., *Morbus Brightii acutus*—Idem valent, *Albuminuria acuta, Nephritis desquamans acuta, Anasarca renalis acuta;* FRENCH EQ., *Maladie de Bright aiguë*—Syn., *Albuminurie aiguë, Nephrite desquamative, Anasarque aiguë d'origine renale;* GERMAN EQ., *Acute Bright'sche Krankheit, Acute parenchymatöse Nephritis;* ITALIAN EQ., *Malattia di Bright acuta*—Syn., *Albuminuria acuta, Nefritide desquamativa acuta, Idropisia renale acuta.*

Definition.—*An intense febrile disease, which may come on after scarlatina, and other exanthemata, or independent of these, and which is marked by signs of intense congestion of the kidney, with exudation and hæmorrhage into the tubes, and desquamation of the epithelium. The secondary phenomena are uræmic symptoms to a greater or less degree, and in the majority of cases general dropsy.*

Pathology.—This affection seems to bear a similar relation to chronic Bright's disease that cases of acute phthisis bear to scrofula. It is described under the name of "*acute desquamative nephritis*" by Dr. George Johnson (whose investigations have especially elucidated this form of kidney disease); and corresponds with the acute inflammatory dropsy of many writers. Elements closely allied to pus form in the kidney-tubes.

In the rapid forms of Bright's disease the products in the urine occur at first in the form of casts—a catarrhal process. These casts may accumulate and block up the tubes. The kidney is then enlarged, of a white colour (the large white kidney of Bright). Acute dropsy is constant, often ascribed to cold, or as a result of scarlatina. Urine may be for a time suppressed, and the little which passes is of a red-brown colour, generally from blood. The sediment is abundant and deeply tinged with blood. It contains albumen, blood-casts, and renal epithelium. The sediment is "composed of coagulated fibrin, blood-corpuscles, cells having for the most part the character of renal epithelium, and occasionally crystals of uric acid. Some of the fibrin is coagulated in irregular masses, having no definite form; this is always the case when the hæmorrhage has been abundant and rapid, so that much of the blood has escaped from the kidneys before it has had time to coagulate; but with these masses there will be seen numerous cylindrical bodies composed of fibrin, which, having exuded from the Malpighian bodies, have coagulated in the tubes, and, escaping thence, present solid cylindrical moulds of the interior of the tubes, in which are entangled blood-corpuscles and epithelial cells, which have been shed by a process of desquamation from the surface of the tubes" (Johnson, *On the Kidney*, p. 89). To such casts, characterized by the presence of recently formed and entire epithelial cells, Dr Johnson proposed the name of "*epithelial casts*"—their average diameter being about $\frac{1}{700}$th of an inch. Death, sometimes after only a fortnight's illness, discloses a large soft kidney, with swollen cortical substance of a dark colour, on removal of the capsules, and the surface much injected. It exudes, on section, drops of blood. To the microscope the tubes are opaque, and are filled with lymph-corpuscles and granular matter. Some have lost their epithelium, and are filled with coagulated fibrin, which, if expelled, would form a fibrinous cast.

At a later period the pyramidal portions are still of a dark colour; but afterwards the cortical part becomes paler and softer, the surface not smooth when cut, and giving out a turbid fluid when squeezed. The tubes are seen to be denuded of epithelium, and filled with masses of secretion, which take on the forms of the tubes. Scanty and bloody urine are conditions associated with engorgement of the Malpighian tufts. These are visible as red specks to the naked eye, and the tubes may be found filled with blood, or containing hæmatine. The morbid process continuing, secretion collects in the tubes and fills them to an extreme degree, and the more it accumulates, the larger and whiter the kidney becomes. The material can be seen with the naked eye in the cortical part. The surface is pale and smooth, with stellate or arborescent venation, surrounding numbers of opaque white spots. A section shows similar appearances, but the white material is now seen disposed in lines. If a congeries of tubes are swollen between the blood-vessels, granulations are thus formed. The new material consists of large masses of dark, granular, and fatty matter, which distend the tubes.

A section shows, microscopically, large portions of tubes quite opaque; their natural lining is gone, and contiguous tubes become united or massed together by the new material which fills them; and it is often difficult to determine whether the partition-membranes of the tubes exist or not (WILKS).

Granular matter may also be seen in some parts of the straight tubes. It is exceptional to find this material in the capsule of the Malpighian tufts.

The *Urine* in the early stages and height of the disease presents intensely febrile characters. It is small in quantity, deeply pigmented, and deposits urates. It contains a variable but usually a large amount of albumen and blood. The sediments consist of desquamated kidney ureter, and bladder-structures, and voided renal cylinders, and sometimes large masses of coagulated fibrin, or partly decolorized clots. Urea is augmented; and when it appears to be below the normal amount, its exit is impeded, and uræmic symptoms are present, which generally increase and prove fatal. When the urine is very scanty, it becomes almost solid by heat. When the disease is about to end fatally, the quantity of water and of the solids decrease, the diminution of the solids being more considerable than that of the water. The albumen decreases least, and is very abundant till the last. When recovery is about to take place, *diuresis* usually occurs; and often an enormous quantity of water is passed, containing much *urea* and *chloride of sodium.* The albumen at the same time diminishes and disappears, and the kidneys recover perfect health (PARKES, l. c., p. 378).

Causes.—The poisons of *scarlet fever*, *cholera*, *measles*, and *erysipelas;* intemperance in alcoholic drinks; the exhausting influence of previous disease; deficiency of food, with fatigue and mental anxiety; exposure to cold and wet.

Treatment.—(1.) Relieve the kidneys as much as possible from the labour of elimination, by avoiding exposure to cold, by keeping the patient at rest in bed, in a room of moderate uniform temperature. (2.) The food should be scanty, consisting of gruel, arrow-root, milk, or weak broth; pure water is the best drink, and alcoholic fluids are not to be taken on any account. (3.) Free action of the skin and bowels must be maintained. The *hot air bath* and antimonial remedies are the best agents to effect the first of these conditions, and free perspiration is to be encouraged by bedding the patient in blankets. *Antimonial wine* may be given in doses of from fifteen to thirty drops every four or five hours. The bowels are to be kept open by the *compound jalap powder*, in doses of twenty to sixty grains, repeated daily or on alternate days. It may be alternated with *podophyllin*, or with extract of *colocynth.* Mercury is not to be given. (4.) Cupping over the loins relieves pain in the back, and the quantity of urine passed generally increases after eight or ten ounces of blood have been withdrawn in this way from an adult, or two or three ounces from a child three or four years old. (5.) When the tongue becomes clean and the general symptoms improve, mutton broth or good beef-tea may be indulged in; and, as the digestion improves, solid food may be eaten in small quantities, beginning with fish and fowl, and afterwards mutton or beef. (6.) Flannel must be worn next the skin. (7.) Iron is of great service during convalescence, for in such cases the anæmia becomes extreme. Phosphate of iron in the form of *syrup*, or *citrate* of *iron* and *quinia*, or the *ferrum reductum*, are the most digestible forms, and they ought to be given in small doses repeated after every diet. (8.) Diuretics are not to be given (see JOHNSON, *loc. cit.*, p. 125 to 138). In a case where the urine was suppressed, fomentations consisting of infusion of the leaves of digitalis were found by Professors Christison and Vogel to increase enormously the amount of urine. Dr. Parkes found the amount of the albumen to diminish markedly from the use of the tincture of the sesquichloride of iron (l. c., p. 379).

II.—CHRONIC BRIGHT'S DISEASE—*Syn.*, CHRONIC ALBUMINURIA.

LATIN EQ., *Morbus Brightii longus*—Idem valent, *Albuminuria longa;* FRENCH EQ., *Maladie de Bright chronique*— Syn., *Albuminurie chronique;* GERMAN EQ., *Chronische Bright'sche Krankheit;* ITALIAN EQ., *Malattia di Bright cronica.*

The **Definition and General Pathology** have been already indicated, —page 1072, *et seq.*

It remains now here to consider the

Morbid Anatomy of the Kidney in Chronic Bright's Disease.—There are several different forms of lesion in the kidney associated with the phenomena of chronic Bright's disease. The several forms are distinguished by certain anatomical characters, and by more or less characteristic symptoms throughout the disease. Two at least of these forms may be considered as typical and distinct; and other forms may be recognized as a commingling of these two—modifications of them—or a mixture of these typical states together, but not to be considered as a gradation from the one form into the other. The two forms are essentially different, and never pass by any pathological process from the one to the other (WILKS, GOODFELLOW). Other forms of morbid kidney in this disease have been regarded as degenerations, namely,—(1.) The fatty; and (2.) The amyloid or lardaceous kidney.

The late Dr. Bright, the distinguished physician of Guy's Hospital (who first discovered the relation subsisting between these morbid kidneys and certain cases of anasarca), described the disease, which now bears his name, as an affection of the kidney passing through *three* stages. Martin Solon describes *five* stages, and Rayer no fewer than *six*, and Rokitansky *nine*.

More recent observers — for example, Frerichs, Reinhardt, and Chambers — recognize three stages. Those who contend for the difference of stages affirm that in the first stage the kidneys are unusually large, flabby, loaded with dark venous blood, and hardly in any respect different from what is observed in diffuse inflammation, except that externally the kidney has a granular appearance, caused by the deposition of a dark, reddish-yellow matter, which is an effusion of inflammatory products.

In opposition to the views which hold that *Bright's disease* is always one morbid state passing through successive stages, the observations of Dr. George Johnson, of King's College; of Dr. Wilks, of Guy's Hospital; and of Dr. Goodfellow, of the Middlesex, lead to the belief that there are several lesions of the kidney developed independently of each other, and associated with the phenomena of Bright's disease.

The *subdivisions* of the lesions in the chronic form of Bright's disease, as adopted by the College of Physicians (which leaves the progressive development of these lesions from one into another an open question), will now be considered, namely:—

(a.) *Granular Kidney*—Syn., *Contracted Granular Kidney, Chronic Desquamative Nephritis, Gouty Kidney.*

In this form of lesion the kidney is smaller than the normal kidney, sometimes only half, or even less than half, the natural size. It is hard, contracted, red, and granular. The cortical part is much narrowed,—it is wasted, and almost allows the cones to touch each other; and the capsule, generally thickened and opaque, comes very near the margin of the pyramidal portion. The capsule is in general separated with great difficulty from the surface, so that more or less of the substance of the kidney is torn away with it. The surface of the organ is granular,—its substance is tough, coarse, and fibrous. The distribution of the veins on the surface of the kidney appears to determine the true shape or limits of the granulations and depressions seen on the surface. The granulations are composed of bundles of tubes lying between the meshes of these veins. The blood-vessels are diminished in size, and more impervious than in the large "white kidney." It is this form of kidney which shows so well the fibrous matrix between the tubules when examined by the microscope—an appearance which some attribute to the shrunken state of the kidney, while others consider this matrix to be entirely new structure. There can, I think, be no doubt of a matrix or skeleton of connective-tissue existing in the kidney, and holding its tubules together, although it may in some cases present almost little beyond a granular appearance between tubes and blood-vessels; or at all events it is extremely delicate and difficult to be seen, unless the light is well managed. This tissue becomes intensely hypertrophied in the small contracted kidney of chronic Bright's disease.

A microscopic section shows the connective-tissue greatly increased, and having a coarse appearance, passing inwards from the depressions on the surface, the tubes lying side by side, and closely packed together, shrivelled up and imbedded in the new connective-tissue. They are by no means uniform in appearance. Some are almost, if not quite solid, forming fibrous cords. Some are of very irregular shape, from contractions and dilatations, giving a sacculated or varicose appearance to the section. Many of these sacculated expansions being cut off from the rest of the tubule, form independent cavities or cysts, both in the cortex and in the pyramidal parts. They may be only capable of recognition by the microscope, although sometimes obvious to the naked eye. In the pyramids such cysts are sometimes arranged in a row, of an oval shape, lying end to end. Their walls are generally very thin; but nuclei and epithelium, as in neighbouring tubes, may still be visible. They are formed by portions of tubes, obstructed so as to enclose a space which forms the cyst, or by dropsical distension of the capsules of the Malpighian glomeruli. The large cystic kidney may be an exaggeration of the process (Clymer). These tubules, cysts, or cavities are devoid of epithelium; and if any remain, it is imperfect, shrivelled, and granular. The walls of the tubes are puckered, and present an irregular outline (Wilks, Goodfellow). The process which leads to this form of kidney generally extends through many years, and is seldom found without, at the same time, a similar process having taken place in the *liver*, or the *spleen*, or the *lungs;* and it is more than probable that these have been affected cotemporaneously from the influence of the constitutional cause which affects the body generally. The increase and alteration of the fibrous element tend to induce the atrophy and destruction of the tubes and glomeruli, and at the same time there is a degeneration and actual wasting of the whole tissues of the kidney.

In the most extreme or advanced stage of granular kidney, the size is greatly reduced. I have weighed such kidneys as low as twelve drachms. The capsule is generally thickened, opaque, and not easily separated. On removal the granulations are seen of a lighter colour than other parts—about $\frac{1}{8}$th to $\frac{1}{16}$th of an inch in diameter—marked out by irregular, livid, vascular depressions, contrasting with the pale, bloodless, granular elevations. Dr. Clymer considers that "the minute changes in advanced granular degeneration, as seen by the microscope, show that the essence of this affection consists in a slow irritation of the connective-tissue, especially that which immediately surrounds the Malpighian bodies and blood-vessels, followed by a proliferation of that tissue, so that it gradually usurps the place of the tubules, which it contracts and flattens till they become mere threads. The microscopic appearances are as follow:—The most conspicuous alterations are at the surface immediately beneath the capsule, at the points of superficial depression, little streamlets of fibrous tissue passing into the organ and embedding the Malpighian bodies and compressing the tubes. As the fibroid material extends inwards, it becomes diffused, and spreads over and between all the tubes in its neighbourhood. Or its existence may be chiefly indicated by the contraction it has caused, so that an angular space under the capsule is seen filled by the shrivelled remains of the tubes. In such cases the Malpighian bodies are aggregated, owing to the contraction of the parts between them, while they resist the compressing agency. The new fibrous tissue is sometimes seen in isolated patches of some extent in the deeper parts of the cortex. In some portions the cortical tubes have been reduced to impermeable microscopic threads, whilst in others they are irregularly dilated. The effusion which leads to increase of fibrous tissue does not take place uniformly throughout, but at points a little removed from each other. The tubes in the track of the effusion may be involved whilst others escape, and, in the earlier stages of the disease particularly, many tubes may be found perfectly natural. Those which are altered are in one or two conditions, both of which are commonly found in the same kidney: (*a.*) They are packed with epithelium, or distended with dark granular matter, probably the result of the breaking up of the cells subsequent to their detachment from the wall of the tube; (*b.*) A transparent fibrinous material may take the place of the epithelial lining, and occupy the tubes. This material may be quite uniform and glossy, or studded with oil-globules. Sometimes it is broken up into very fine fragments, and passes out with the urine as dark, coarse, granular casts, which often look opaque and granular till touched with acetic acid, when they become clear, and show the broken pieces of fibrine of which they chiefly consist. The condition of the tubes which form the cones is precisely the same."

The granular kidney has been regarded by some as a result of original productive changes or growth of new material, which first shows itself in the large white kidney seen in certain cases of Bright's disease. In this form the kidney varies in size to nearly double, and weighs from six to twelve ounces, or even more. Its external cortical secreting part is increased at the expense of the internal medullary or purely excreting parts. Between the base of the pyramids of a cut section and the investing capsule, an extreme case will measure from half an inch to an inch. The general appearance of the kidney is of a whitish or yellowish-white colour; it is anæmic, partly from the obliteration of the vessels, partly from more or less abundant serosity, and partly from the diminution in the amount of red blood-discs. In some parts the veins may be enlarged and turgid; in the interior of the kidney such veins are tortuous, on the surface they have an arborescent form. The medullary portion may present various degrees of engorgement, or it may be as exsanguine as the cortical part; but such extreme bloodlessness is rare. Such a kidney is flaccid.

The morbid development of this large white kidney may be traced through two stages; and if the patient lives through these two, it may be recognized even in a third stage.

If death ensues early, the kidney is found to be of tolerably firm consistence; its surface smooth, and of a more or less deeply suffused redness, very numerously interspersed with minute points of a deeper red. The capsule, a little more vascular than natural, may be easily detached. On section, the increase in size is found to be mainly in the cortical substance, which is minutely injected with blood, and presents a uniform redness, with numerous dots of a deeper red—engorged Malpighian bodies. Other red spots may be visible—due to ecchymoses. Sometimes there may be seen portions of a lighter shade of colour, from the growth of new material. With this general state of engorgement there may be actual extravasation into the Malpighian capsules, into the tubules, and even into the intertubular substance. The pyramids are redder than natural, and the mucous membrane of the calyces and pelvis is injected. The tubes are filled with epithelium, mixed with fibrinous matter and blood, and sometimes holding together minute crystals of oxalate of lime or lithic acid. The entire parenchyma, but especially the cortical substance, is peculiarly pulpy and friable; and the surface, on section or on fracture, yields a reddish-brown slightly viscid fluid, more or less bloody, and delicately flocculent or opaque. The *materies morbi*, seeking to effect its discharge, stimulates the cell-growth in the tubules of the gland—"an acceleration of growth and of function which is incompatible with maturity of the secreted products. The epithelial cells, therefore, undergo various arrests or modifications of development. The ultimate tubules are found gorged with an uneliminable excess of crude and vitiated secretion. Blood, amorphous matter, and an infinite range of cell-growth, from pus-globules to the healthy germination of the gland-cells, present themselves in various combinations. By products such as these the tubes are plugged and irregularly distended; and they not unfrequently burst and are annihilated. So close is the compaction of material, even in many of those tubes that have no shaped inflammatory products within them, that they are plainly impervious; and it is only by artificial means—by further tearing of the fragment, or by the use of chemical agents (under the microscope)—that we can satisfy ourselves that the dense plug in question consists of but agglomerated gland-cells" (SIMON). A second or more advanced stage in the morbid development of this large white kidney may be recognized in the still greater enlargement of the organ; but the blood engorgement has subsided, the cortical substance is now paler than natural—of a whitish or yellowish-white colour. It is still more thickened or enlarged relatively to the pyramidal portion, is somewhat soft and friable, and on pressure yields a turbid milky-looking fluid. The granular material of increase is to be found almost entirely in the cortical part, both in the convoluted tubes and between them, and may be found dipping down between the tubuli of the pyramids, and separating them. The tubes of the pyramids at the base thus present a radiated, striated, and frayed or unravelled appearance; and in the cortical substance numerous whitish lines are traceable from the surface inwards, some of which are composed of numerous isolated whitish *puncta*, while others are uninterrupted. The pyramids are of a reddish colour, of different shades in different cases. The vessels of the cortex are extensively obliterated, and the new material in this stage has undergone more or less of fatty metamorphosis. These whitish *puncta* are more particularly abundant in the superficial layer of the cortical part. They are composed of tense granules of a yellowish-white colour (Bright's and Christison's granulations), of the size of a poppy seed or a pin's head. In the cortical substance these granulations are chiefly due to the lesions in the Malpighian capsules.

Occasionally it happens that a patient may survive through these two stages of the kidney lesion, when a third stage of development may be recognized which this large white kidney will pass through. The fatty degeneration progresses; and becoming absorbed, a wasted, shrivelled, flaccid but tough kidney results.

Microscopically, the appearances of this large white kidney vary somewhat with its stage of development. The Malpighian bodies are irregular in size, some being excessively enlarged, others being smaller than natural. Their capsules are more or less distended with granular matter, and so also are the tubes. The tufts of capillaries composing the glomeruli seem wasted, and only a few large distended ones are visible. Fat-globules may also be detected, and granules of fat in extreme minuteness. In some places the tubes are empty, and compressed by intertubular new material; but in general they are greatly distended, with altered epithelium, mixed with granular matter more or less fine. Here and there a tolerably healthy capsule and tube may be distinguished close to one that has undergone the changes already noticed. The altered epithelium which abounds contains varying quantities of oil. Some of the cells are large, others are shrivelled up and otherwise imperfect in form, and all of them are more or less granular;

and lines of the same granular matter may be often observed between the straight tubes.

The next stage is marked by the granular matter penetrating still deeper into the cortical substance, and which gradually increases till it invades the whole of the medullary substance of the kidney. This granular substance is of a greyish-red or greyish-yellow colour, and has in many cases something of a *cheese*-like appearance. The kidney now may be larger than natural, sometimes of the natural size, and sometimes, though rarely, diminished. Its consistency also varies; for, if enlarged, it is commonly softer than the healthy kidney; but, if diminished, it is for the most part firmer. Its colour, viewed externally, is sometimes a pale tint of the natural hue, but more commonly it is of a greyish-yellow or yellowish-red colour, and mottled. Its surface is also strongly granulated, and even rough. In this state, if the kidney be injected, the matter of the injection does not, according to Dr. Bright, penetrate the cortical portion. This is the stage in which it is considered that a complete and general metamorphosis of the inflammatory products into fat takes place.

The last stage is marked by the morbid granular deposit, which, besides invading the medullary substance, attacks the tubular portions of the kidney, so that the tubuli are often to a very considerable extent obliterated, and perhaps, with the exception of a single pencil of that structure, is entirely converted into one homogeneous degeneration. The kidneys are now, in some instances, of their natural size, but more generally they are contracted, and are smaller than usual. Their surface is lobulated, pale, and granular, resembling the roe of a salmon or the vitelarium of a bird. Their consistency also is sometimes softer and sometimes harder than natural; and Dr. Bright speaks of some instances in which they cut like cartilage. This is the stage which leads to final atrophy and wasting of the kidney.

(b.) *Fatty Kidney.*

As a degenerative change, similar to its occurrence in other organs, this lesion may not always be associated with the phenomena of Bright's disease.

The presence of fat in the kidneys of cases of Bright's disease is altogether a secondary process, and due to a change or degeneration of the inflammatory products. The renal secreting cells are found to contain distinct globules of oil or fat-granules, which obliterate all appearance of a nucleus. Little, however, is positively known regarding the formation of this degeneration as an independent form of Bright's disease; but it is a degeneration which supervenes to a great extent upon the "large white kidney," and is found more or less associated with it in every instance. The fatty kidney is large and flaccid, full of yellowish or whitish striæ and marks, combined or not with the characters of the parenchymatous inflammation just described. The calibre of the urinary canals are diminished, and are separated by fatty masses.

(c.) *Lardaceous Kidney*—Syn., *Amyloid Disease, Waxy Kidney.*

The lardaceous disease of the kidney, associated with the phenomena of Bright's disease, constitutes the eighth form of kidney lesion which Rokitansky describes in connection with that disease. It is the form most commonly found in patients who have suffered from constitutional syphilis or mercurialism; also in scrofulous or ricketty persons. It is generally associated with similar lesions of the spleen and liver.

The kidney in this form of disease presents but a slight increase of size, and is always considerably indurated. It is generally of a brownish-yellow colour, and the cortical substance on section has a glossy waxy look, with a glistening appearance of the glomeruli when the light is allowed to fall upon them in an oblique direction. The substance is usually hard and brittle, and infiltrated with the albuminoid material.

Gairdner, Sanders, and Virchow were the first to discover the true nature of this condition of the kidney. It is the Malpighian bodies and arteries of the kidney where the disease seems first to commence; but it is not limited to these structures, for ultimately the tubes, epithelium, and the intertubular textures become similarly affected—so that in extreme forms the organ looks "as if it had been soaked in a substance like glycerine or balsam, which had subsequently hardened, and so enveloped all the tissues." The lesion seems to commence—(1.) In the loops of the Malpighian capillaries; and (2.) In their afferent vessels; and after advancing to a certain stage, the injection of the cortical substance by means of the finest artificial injection becomes impracticable. The diseased arteries therefore become impervious to blood; and ultimately the Malpighian glomeruli become converted into solid, homogeneous, translucent globules; and the extension of the disease to the tubes and other tissues produces at length the condition known as waxy kidney. Sometimes the disease commences in the capillary vessels of the *vasa recti.*

It is thus to be distinguished from the large white and soft kidney already described. It is much harder, tougher, and firmer. It is not easily torn; and its surface is uniform and smooth. The cortical substance, having a dim waxy appearance, is increased in extent, and anæmia predominates. It is this translucency, hardness, and uniformity of appearance which are sufficient characters by which to identify extreme cases; and in the more common and slighter cases the Malpighian glomeruli can be seen shining prominently like glistening grains on the cut surface. The iodine test, combined with microscopic examination, will alone, however, clench the recognition of the lesion (vol. i., p. 104). But without the application of any re-agent, the Malpighian glomeruli appear as glistening bodies, having a clearness resembling the appearance of "frosted" glass or a globule of rough ice. An excellent wood-cut of this appearance is given by Dr. Bennett in his work on the *Principles and Practice of Medicine*, p. 807, fig. 445. The capsule of the kidney is generally easily torn off; and there may be considerable loss of substance or atrophy present, indicated by depressions or hollows on the surface of the kidney beneath the capsule. Sometimes the degeneration is so slight that a microscopic examination is necessary to detect its commencement.

Lardaceous disease of the kidney begins with the arterial capillaries; especially—(1.) The loops of the *Malpighian* tufts; (2.) Their afferent vessels; (3.) The *vasa efferentia* and capillaries in the cortical part; (4.) The "*arteriolæ rectæ;*" and after the lesion has advanced to a certain point the injection of the cortical substance becomes impracticable; the arteries become impervious, the cortical substance anæmic, while hyperæmia increases in the pyramids; and hæmorrhage is apt to occur at times, owing to the increased pressure on the inelastic vascular walls, giving rise to reddish-brown streaks or spots. Ultimately the Malpighian glomeruli become converted into solid, homogeneous, translucent globules; and the extension of the disease to the tubes and other tissue produces at length the condition known as waxy kidney. Sometimes the degeneration begins in the capillaries of the *arteriolæ rectæ;* but rarely are the large vessels affected, and it is long before the epithelial tissues become affected.

The lardaceous kidney must be distinguished from the large white and soft kidney described by Dr. Bright. It is much harder, more tough, and firmer than the white kidney of Bright. It is not easily torn, which is the case with Bright's white kidney. The surface appears uniform and smooth. The cortical substance is greatly increased in extent, pale, anæmic, and of a dim waxy appearance. The translucency, hardness, and uniformity are sufficient to identify the extreme cases; and in the more common and slighter cases the Malpighian glomeruli can be seen shining prominently, like glistening grains of a whitish-grey appearance, on the cut surface. The iodine test is absolutely necessary; and, unless a microscope has been used, it cannot be declared that the lardaceous lesion is not present. Without any re-agent, the Malpighian glomeruli appear under the microscope as globular transparent bodies, with a glistening aspect.

The capsule of the lardaceous kidney is generally easily torn off, and loss of substance or atrophy may be present, as indicated by depressions or hollows of the surface beneath the capsules. The lesion is most frequently combined with parenchymatous or interstitial nephritis, or the interstitial fatty kidney, especially in syphilitic cases.

With regard to the lardaceous kidney, Dr. T. Grainger Stewart, Pathologist to the Royal Infirmary, Edinburgh, has published recently (August, 1864, in *Edin. Med. Journal*) further observations, confirming those already referred to in the previous volume of this edition. He has found great amelioration of the symptoms from the adoption of the following rules:—(1.) To attend to the nutrition of the patient, giving good nutritive food in the form best suited to the individual tastes and powers of digestion; (2.) To give such tonic medicines as may improve the appetite; (3.) To give such hæmatic medicines as control the tendency to anæmia; and among these pre-eminently, the syrup of the *iodide of iron.* The *syrup of the phosphate,* as well as the *syrup of the phosphates of iron, quinia,* and *strychnia* (the formula for which is given at page 86, *ante*), are also, I think, most useful remedies; (4.) In all cases in which a syphilitic infection has been traced, and even in many others, *iodide of potassium* is to be given in moderate and sustained doses. It diminishes the bulk of the liver in a remarkable manner (*Edin. Med. Journal*, August, 1864).

(d.) *Mixed Forms of Kidney Lesions in Bright's Disease.*

There are undoubtedly cases, when the patient has been ill for a long time, in which the kidney is found presenting the characters of more than one of the lesions now described. The organs may be of ordinary size, granular, white, degenerating, and containing deposits visible to the naked eye, the processes leading to the lesions now described being engrafted, so to speak, the one upon the other. The kidneys are less pale than those which constitute the "large white kidney." The vessels are much more numerous, and more or less gorged with blood. The Malpighian tufts are red and solid, and the organ presents a very coarse and granular appearance (GOODFELLOW).

Symptoms.—It is necessary to inquire into the history of each particular case of Bright's disease, so as to ascertain the precise period, if possible, when the general health began to be impaired; and in order to determine the particular morbid condition in which the kidney exists, it is necessary to make a chemical and microscopical examination of the urine from day to day, determining especially those points which have been enumerated in the preceding section.

The general symptoms which are indicative of the several diseases or states of the kidney comprised under the general term "Bright's disease" have been recently analyzed and admirably described with great care by Dr. Goodfellow, Lecturer on Medicine at the Middlesex Hospital. They may be comprised under the following heads:—

1. Although the anasarca or general dropsy, either or both of which are usually prominent symptoms when a patient with "Bright's disease" seeks medical advice, yet in nearly every instance they have been preceded by other phenomena more or less definite—*e. g.*, febrile excitement, a dry and harsh state of the skin, a quick and hard pulse. It is only in the acute cases that the prominent and characteristic phenomena of anasarca come on with great rapidity, and commence generally with puffiness of the eyelids, or of the whole face, rapidly extending over the rest of the body. In the more chronic cases the œdema generally commences in the lower limbs; and it is at the same time associated with a pallid condition of the eyelids, and of the looser connective-tissue of the face.

2. Anæmia is another prominent phenomenon. It is indicated by the pallor of the surface of the body and of the countenance. Its progressive appearance may even suggest a suspicion of renal disease before the anasarca sets in, especially in patients above the age of from thirty-five to forty, and whose urine ought therefore to be at once examined.

3. Pain in the loins may or may not be a sign of significance. Lumbar pains may be considerable in amount, and yet no renal disease

may be capable of detection at the time. In most cases no unpleasant sensation is felt in the lumbar region till the anasarca becomes considerable.

4. In the early stages of the affection there is always a very considerable diminution in the quantity of urine passed. But there are many exceptions to the rule, and in some cases the urine is more abundant than usual—*e. g.*, Parkes has measured more than 100 ounces; Rosenstein (quoted by Parkes) relates a case where an increase of 174 ounces was passed daily during eight days. Various circumstances concur in determining the greater or less amount of water passed. (See under "Lardaceous Disease," vol. i., p. 99).

(*a.*) The anatomical condition of the kidneys influences the amount, and especially with or without anæmic symptoms. The mean of *six* cases without anæmic symptoms gave 61·5 ounces daily. The mean of *four* cases with uræmic symptoms gave thirty-eight ounces daily, the specific gravity in both sets of cases being very nearly the same (PARKES). There is some reason to believe that, in the highly atrophic kidney, when many vessels are obliterated, the urinary water is on an average lessened; but yet on some particular days a large quantity may be execreted; for example, Dr. Parkes has known as much as *sixty ounces* passed in one day by a kidney under such a condition.

(*b.*) The co-existence of dropsy with anæmia in an advanced period of the disease appears to be associated with lessened water; while, on the other hand, an improvement in dropsical symptoms is attended by more or less profuse diuresis.

(*c.*) In a few cases the amount of urine is influenced by the presence or absence of fever. The quantity is very much smaller in amount on fever days as compared with fever-free days.

(*d.*) Organic lesions of the liver, heart, and lungs may either cause lessened rapidity of flow through the renal vessels, or give rise to great variations in lateral pressure.

(*e.*) One of the main causes of a variable amount of water is the spontaneous purging or vomiting which sometimes takes place, and which would lessen the amount of urine; while, on the other hand, lessened skin exhalations will increase the flow.

Until the metamorphosis of the nitrogenous tissues in Bright's disease is better understood, the question as to the possible lessening of *formation of urea* and *uric acid* must remain undecided. Hitherto lessening of these products seems to have been the rule, to which, however, there are numerous exceptions (PARKES); and on the whole it appears probable that the effect of *a febrile* Bright's disease on the ordinary urinary constituents is chiefly owing to the physical condition of the kidney. There is no doubt that urea in the blood is increased; and it seems pretty clear that the urea in Bright's disease is still formed to a considerable amount, and that its lessening in the urine is in part owing to *retention* from simple impediment to diffusion through the diseased renal vessels and tubes; but it is yet undecided whether or not there is at the same time a *lessened formation* of urea (PARKES).

The principal abnormal constituent of the urine in Bright's disease is dissolved albumen, which exists in various conditions, and gives different re-actions with the usual tests, heat and nitric acid. Sometimes it entirely coagulates, and is precipitated by heat and a moderate quantity of nitric acid; at other times, after precipitation by heat *from an acid urine*, it is entirely re-soluble in a moderate amount of nitric acid. A very minute quantity of nitric acid will also sometimes prevent its precipitation by heat, while on the other hand it is sometimes precipitated by nitric acid, and not by heat (various authorities quoted by PARKES). Its most usual condition, however, is that in which it is precipitated by heat from a urine which is naturally sharply acid, or which has been made so by a moderate quantity of nitric or acetic acid.

Of the apparently anomalous phenomena just described, the following explanation is given by Dr. Parkes:—"When albumen is experimented with, it can be made to pass through various phases of solubility and insolubility, from the actions of acids, alkalies, and salts, without its real nature or composition being in any way altered; and as such influences will act on it more or less in every urine, it by no means follows, when the albumen in the urine of Bright's disease presents modifications in its re-actions with re-agents, that it is in any very peculiar or unusual condition" (*On the Urine*, p. 390).

The amount of albumen varies much, ranging from 5 to 545 grains in the twenty-four hours (PARKES); and in any particular case the amount varies greatly from day to day. It is often increased during the day, from movement or from food and Dr. Parkes has noticed it very greatly increased in the second and third hours after dinner. The albumen, however, is not constant, nor does it occur in acute cases of Bright's disease; though passed, it is often in a peculiar condition, being less perfectly coagulable by heat, and sometimes approaching in character the *albuminose* of Miahle. The albumen does not always increase as the disease advances; on the contrary, while there is a tendency to a larger excretion of albumen in the early stages of the disease than in the later ones, and while sometimes even at the last there is much albumen, yet it is sometimes entirely absent in the later period of the disease. In a certain number of cases the albumen may quite disappear from the urine; but it is important to notice that the detachment of fibrinous casts goes on even when the albumen has disappeared.

In small quantities fat is very common in the urine, derived directly from the kidneys. It is usually in the form of *oleine*, mixed up with albuminous substances, or contained in the epithelial or other cells, and microscopically visible; and it must be remembered, as Dr. Johnson was the first to determine, that some of the secreting or epithelial cells of the kidney enclose a minute quantity of fat, just as in the secreting cells of a healthy liver, as shown by Bowman.

In the more advanced stage of chronic Bright's disease, instead of the urine being scanty in quantity, and having a tolerably high specific gravity, the urine passed in twenty-four hours may amount to from *thirty-five* to *fifty* ounces or more, being equal to, and in some cases greater than, the average in health. The specific gravity is nearly always below the healthy average, the urine pale, and in very advanced cases almost colourless. Occasionally it may be red, reddish-brown, pale smoke-brown, or "smoky," as it is commonly described. If the urine is highly fatty, it may have a milky appearance.

Besides the albumen and other constituents of the serum of the blood, the urine may contain blood-corpuscles, casts containing blood, fibrinous filmy matters, and casts of tubes (granular, fatty, waxy, or hyaline), or simply of epithelium. A perfect acquaintance with these casts and with urinary deposits is essentially necessary; for, by the characters of these casts, when taken in connection with the history and symptoms, the physician is able approximatively to discriminate the morbid condition of the kidney in each particular case (BASHAM, GOODFELLOW).

The specific gravity of the urine may range from 1,020 to 1,025, from two causes, namely—(1.) The presence of serum, which is heavier than urine; (2.) From the small amount of water (relatively) (GOODFELLOW).

5. There is nearly always a frequent desire to micturate, especially at night, when the patient is in the horizontal posture (WATSON), with a sensation of heat or scalding on passing water, accompanied with a discharge of mucus from the urethra, which possesses a more or less puriform character, and appears in the urine in the form of slight thin shreds (GOODFELLOW).

6. Dryness of the skin prevails, and the power of eliminating water by the skin seems impaired. Heat of skin and general feverishness, with occasional chilliness of the surface, and a pulse generally above the natural standard, are common phenomena throughout the later stages of the disease, and when emaciation and debility are progressive.

7. *The Dyspeptic symptoms* which prevail at an early period indicate irritation of the gastro-intestinal mucous membrane. Loss of appetite, sometimes amounting to actual loathing of food, or a capricious and uncertain appetite, are amongst the most prominent phenomena. The food taken rests uneasily in the stomach, giving rise to stomachal and intestinal *pneumatosis* and acid eructations, the explosive force of the gas so generated being sometimes so great as to cause partial regurgitation of the food. Gastralgia and pyrosis may prevail; and there is very often nausea, retching, and sometimes vomiting, at occasional intervals. Attacks of diarrhœa are frequent, alternating with costiveness.

When such phenomena are discovered to exist, the urine ought to be examined at once.

8. *Symptoms referable to the State of the Blood.*—The blood in Bright's disease being decidedly watery, and the red corpuscles deficient, the stimulus of healthy blood is not experienced by the heart; on the contrary, the morbid condition of the blood impairs the action of the heart and its circulation through the capillary blood-vessels. Palpitation is caused by very slight exertion, or by any mental emotion, and the heart beats *irritably*—the sounds being preternaturally sharp and abrupt, the rhythm disturbed, and the action irregular and intermittent. With the advance of the constitutional cachexia, the nervous and muscular structures of the heart are ill-nourished, and may become temporarily or permanently damaged; signs of pericardial effusion may occur, or even of pericardial or endocardial inflammation. The condition of the blood varies; but (1.) There is an excess of serum, the clot constituting not more than one-fourth part of the blood. (2.) The density of the serum

returns to its normal standard, or even exceeds it; sometimes, however, it remains low, even in the advanced stages. (3.) The urea disappears as the disease advances, but re-appears towards the fatal termination of the case, even in a larger amount than before. (4.) The fibrin, increased in the first stage, returns to its normal amount as the disease advances, and only becomes considerable again towards the close, especially if some of the intercurrent attacks of inflammation supervene, so common in Bright's disease. (5.) The most remarkable change the blood undergoes is the great decrease of the red blood-globules. They are frequently reduced to *one-third* of the normal proportions; and this diminution progressively advances with the disease, and, as Sir Robert Christison observes, "there is no disease of a chronic nature which so closely approaches hæmorrhage in its power of impoverishing the red particles of the blood." (6.) There are also present in variable, but always in considerable quantities, highly stimulating, irritating, perhaps toxic matters—pure excrementitious material—which can never be retained in the blood without more or less disastrous effects upon several great vital processes, and leading in some cases rapidly to death (GOODFELLOW). Sir Thomas Watson is of opinion "that the renal malady has a direct tendency, by its effect upon the blood, to generate disease of the heart. It induces anæmia, and thereby debility of the muscular texture of the heart, and leads to dilatation of its cavities; and the weak muscle, becoming irritable also, grows thicker as it labours more."

The experiments of Dr. Hammond, the late Director-General of the Medical Department of the Federal Army, have proved that urea, when retained in the blood, either by disease or extirpation of the kidneys, is sure sooner or later to kill.

The blood, therefore, in Bright's disease, being poor, thin, watery, containing much less albumen, and fewer red corpuscles, than in health, and containing, moreover, extraneous offensive matters in the form of urea and the extractives—more or less urinous excrement—is ill adapted to facilitate circulation, but, on the contrary, tends to retards its progress. Ultimately the blood is charged with further impurities. It begins to abound in fatty matter, especially cholesterine, which becomes deposited in several tissues, taking the place of their own proper elements of nutrition, and interfering with their function, as in the heart, the arteries, and the capillaries. "The body is poisoned in detail by the retention of its own excrements."

9. *Dyspnœa* is one of the earliest and most pathognomonic phenomena of chronic Bright's disease; and this shortness of breath is quite apart from, and independent of, any bronchial catarrh or disturbance in the first instance. The patient observes every now and then how short his breath has become. In an ordinary walk he finds he must stop to recover his breath. Occasionally the attacks occur at night, but chiefly during the day; and there is more or less palpitation during the paroxysms; while some wheezing at the chest may also attract the patient's notice. Weeks are thus apt to pass before the patient considers the symptoms sufficiently grave to seek medical advice (BASHAM). If the urine be examined at this early period, the presence of small quantities of albumen may be detected, and the sediment will contain granu-

lar casts, with more or less decayed cell-structures. Another cause of dyspnœa is the œdema which is apt to set in and pervade the base or more dependent parts of the lungs. It must also be remembered that the lungs, the pericardium, and pleura are organs especially liable to the intercurrent attacks of inflammation in Bright's disease.

10. *Symptoms referable to the Nervous System.*—In the absence of anasarca these symptoms are of great importance when they occur coincidently with such other general symptoms as follow anæmia or puffiness of the face. Most of the phenomena are those present in *chloro-anœmia*—*e. g.*, noise in the ears—a blowing or a sensation of ringing noises in the ear, occasional dimness of vision, partial amaurosis, from paralysis of more or fewer of the papillæ of the retina, giving rise to the appearance of motes or small bodies floating before the eyes (*muscæ volitantes*). In some instances there may be flashes of light from irritation of the fibres of the optic nerve. Throbbing of the temporal and other arteries, a sense of fulness or weight at the back of the head and in the nape of the neck, with a disposition to frequent cramp of the muscles of the back and sides of the neck; neuralgic pains in the face and head, or in other parts of the body; headache in the form of *hemicrania* or *megrims;* frequent attacks of giddiness, drowsiness, disposition to comatose sleep, and in some cases profound coma, alternating with convulsions, are all premonitory symptoms of chronic Bright's disease, and may be also fatal ones. The occurrence of what are called *head symptoms* may be frequent during the course of the disease, and death of the patient is so generally preceded by *coma,* with or without convulsions, that Sir Robert Christison considers "death by coma" to be the "natural termination" of Bright's disease, or the mode in which it proves fatal when life is not cut short by some other incidental or secondary affection. Sir Thomas Watson is impressed with the idea that the pale and watery condition to which the blood is at last reduced may have something to do with the stupor and coma, as in *spurious hydrocephalus* similar phenomena are seen. The disposition to giddiness is often a remarkable and characteristic phenomenon. A disposition to erysipelas has also been noticed. Anasarca is one of the earliest symptoms; in ninety-nine out of every one hundred cases it is the first intimation which the physician has of the disease; but if he could see the patient regularly before this symptom sets in, there are no doubt other phenomena, perhaps less definite, which he would have no difficulty in setting down as the probable antecedents of Bright's disease.

Pallor and emaciation, if combined with anasarca, are almost pathognomonic of the disease.

Causes.—The exciting causes of Bright's disease may be enumerated under the following heads,—namely, hereditary tendency or constitution of body, age, sex, climate, occupation, habit of life, intemperance, exposure to cold and moisture, influence of disease poisons.

Intemperance and gout are the circumstances which seem to bring about the disease most frequently in advanced life.

In youth and childhood the hereditary constitution of body, exposure to cold, or the effects of general diseases, such as *scarlatina, erysipelas, measles, variola, syphilis, gout, rheumatism, tuberculosis;* the indiscriminate and excessive indulgence in fermented liquors, such as *alcohol, ale, stout;* the ingestion of *turpentine, æther, naphtha, chloroform,* and the like, are the circumstances and agents which tend to that morbid state of the constitution, and of the digestive functions which lead to Bright's disease. Excessive devotion to mental work ought also to be regarded as a cause; especially if it leads to dyspepsia.

A cold, humid, and variable climate is another powerful predisposing cause; although the disease is also by no means infrequent in tropical climates. These are causes which act directly upon the blood and nervous system, through the digestive and secretory functions; and which tend to interfere especially with the circulation and secretion of the kidney. The hereditary constitution of the body is of great importance. There may be such inherited weakness of constitution that impaired digestion may at once lead to the development of Bright's disease, rather than to any other constitutional disease.

There are causes, also, of Bright's disease which may be regarded as mechanical causes of irritation, but which so secondarily affect the constitution of a person predisposed to the disease that Bright's disease rather than any other is the result—*e. g.*, the *irritation of blows,* of *cantharides,* or other irritants; the presence of calculi in the kidney, the practice of *onanism,* or excessive *venery,* the existence of *amenorrhœa* or *uterine irritation.*

Dr. Goodfellow points out that the influence of spirits and of beer tends each to induce a distinct variety of morbid kidney. From the action of spirits the kidneys do not enlarge, nor vary much from the normal weight; but they become extensively granular, and full of small cysts. The cortical portion is greatly reduced in thickness, and there is much fatty matter in them. A tendency to fibroid proliferation is engendered, a tendency which the kidney shares with such other organs as the *liver* and *lungs.*

The granular form is also frequently associated with gout as a primary morbid condition.

It is a form of kidney lesion produced by lead poisoning. A man with indications of lead poisoning, and who at the same time has albuminuria, is almost certain to become the subject of granular kidney. It is therefore common amongst painters, plumbers, compositors, tin workers, and gas-fitters.

In persons who become subjects of Bright's disease from the excessive consumption of beer, the lesions of the kidney are generally of a mixed nature, something between the large white kidney seen after scarlatina and the true granular kidney, with more or less fatty deposit both in the tubes and in the interstitial tissue.

But while intemperance in drinking is a well-recognized cause of Bright's disease, it is not unknown among children and other persons whose manner of life has been strictly sober. Sir Thomas Watson mentions the example of a young girl, fifteen years old, who had never menstruated, and who became affected with Bright's disease; and he remarks that the disorder has been known in many instances to follow a sudden check or suppression of the catamenia.

Diagnosis.—The specific gravity of the urine, the albumen it contains when persistent, and the amount of the solid constituents, are the first indications of the real nature of the disease. To determine the albumen, a small quantity of the urine in a test-tube must be slowly and gently heated to the *boiling* point by the flame of a spirit-lamp, when, if albumen is present, it will appear in the form of a whitish cloud, of which the constituent particles multiply and collect, in proportion as the quantity is considerable, into small curdy fragments or flakes. These will gradually subside to the lower part of the tube when permitted to rest, leaving the supernatant liquor clear, and so indicate approximatively the amount of albumen present. A second specimen of the urine should be taken in another tube, and, after it has been thus boiled, an excess of *nitric acid* may be poured into the tube, when the albumen present will be precipitated in a flaky or pulpy form. This latter method is the best where the urine to be tested is alkaline; but both methods should be employed in every case; and they are sufficient to determine the presence of albumen.

The following table, calculated by Dr. G. Bird, according to Sir Robert Christison's formula, affords results sufficiently accurate for the guidance of the student or practitioner, and shows at a glance the number of grains of solids, and the weight of a fluid ounce of urine, of every density from 1,010 to 1,040.

Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.	Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.
1010	441·8	10·283	1025	448·4	26·119
1011	442·3	11·337	1026	448·8	27·188
1012	442·7	12·377	1027	449·3	28·265
1013	443·1	13·421	1028	449·7	29·338
1014	443·6	14·470	1029	450·1	30·413
1015	444·0	15·517	1030	450·6	31·496
1016	444·5	16·570	1031	451·0	32·575
1017	444·9	17·622	1032	451·5	33·663
1018	445·2	18·671	1033	451·9	35·746
1019	445·8	19·735	1034	452·3	36·831
1020	446·2	20·792	1035	452·8	37·925
1021	446·6	21·852	1036	453·2	38·014
1022	447·1	22·918	1037	453·6	39·104
1023	447·5	23·981	1038	454·1	40·206
1024	448·0	24·051	1039	454·5	41·300

The amount of solid constituents may be determined approximatively at the bedside by the following formula:—If $D =$ the density or specific gravity of the urine, and $\Delta =$ the difference between 1,000 and its density, the quantity of solids in 1,000 grains will be $= \Delta \times 2{\cdot}33$ for diabetic urine, and by 2 for most cases. Supposing the specific gravity to be 10·20, then $20 \times 2{\cdot}33 = 46{\cdot}60$, which is the amount of solids in 1,000 grains of urine; or simply multiplying the last two figures of the specific gravity by 2 for most cases, and by 2·33 for diabetic urine, will give the amount *in grammes* of solids in 1,000 cubic centimetres of urine (PARKES).

The formula of Trapp (the error of which, according to Vogel, cannot exceed one-tenth in healthy and one-fifth in morbid urine) is the best for urine *not diabetic,* and is as follows:—If Δ represents the excess of the specific gravity of urine above that of water, the amount of the solid constituents of 1,000 parts of urine will be represented by $2\ \Delta$.

A sufficient approximation to the truth for clinical purposes is gained by such formulæ; but they can never supersede the exact processes necessary for scientific investigation (CHRISTISON).

The hydræmia is indicated by the general paleness or anæmic appearance of the surface of the body; and even when no albumen can be discovered in the urine these phenomena are of great value; and when they are observed in persons above the age of from thirty-five to forty-five the physician should at once examine the patient and his urine closely from day to day, in order that he may detect as early as possible the other symptoms and conditions which are characteristic of Bright's disease. The specific gravity of the urine, the quantity passed daily, the amount of urea and of other solids contained in the daily discharge, should all be closely observed, and whether there be not slight puffiness of the eyelids in the morning, or indications of serum beneath the conjunctiva. The paleness is not a dry waxy paleness, as in chlorosis or anæmia-chlorosis. It it a paleness characteristic of dropsy.

When the disease is established, and its diagnosis confirmed, much information will be obtained as a guide to treatment, and as an aid to the diagnosis of the peculiar morbid condition in which the kidney may be, by a daily microscopic examination of the urinary sediments, as recommended by Dr. Basham in his excellent treatise on *Dropsy connected with Disease of the Kidney.*

The specific cell-characters of the sediments have been found by Dr. Basham to be a more certain guide in prognosis than can be furnished by any other property of the urine, or by any other symptom exhibited by the patient. The cell-elements associated with albuminous urine undergo marked alteration and change as the renal disease advances. Under all circumstances it is difficult to form an opinion as to the rate of progress or advance of the lesion in the kidney; and hitherto the physician has been usually guided in his prognosis by the subsidence of the dropsy, or by the diminution of the amount of albumen in the urine. The microscopic examination of the cell-elements is now recognized as yielding more certainty to the opinion which may be formed as to the progress of the renal disease.

The quantity of albumen present is an important point to be considered, along with the characters of the tubular or organic elements contained in the urine; and in private practice the amount of albumen passed is approximatively judged of by noting the *space* which the *coagulum* of albumen occupies in the tube after being allowed to rest. The phraseology recommended by Sir Robert Christison to express the proportions observed by the eye is as follows:—

1. *Gelatinous* by heat. 2. *Very strongly coagulable* by heat—nearly the whole tube. 3. *Strongly coagulable*—half the tube. 4. *Moderately coagulable*—one quarter of the tube. 5. *Slightly coagulable*—one eighth. 6. *Feebly coagulable*—less than one eighth. 7. *Hazy* by heat—no visible flakes of albumen.

The epithelial cells thrown off from the renal tubes, as well as the casts which accompany them, suffer very material and obvious alteration as the disease of the kidney advances. These changes consist in palpable deviations from the standard structure of healthy epithelium. The cells lining the straight tubes are the last to exhibit any character of degeneration; and in no case do the cells of the kidney-tubes appear in the urine except under the influence of disease. Any one single examination of the urine is not sufficient to determine the stage to which the disease has advanced. It is only by comparing the character of the deposit of one period with another, and noting carefully the altered appearance of the casts and cells, and the direction of whatever change is taking place, that an approximation of more or less certainty is made regarding the progress of the disease.

The sediment found in albuminous urine consists principally of cylindrical forms moulded in the uriniferous tubes of the kidney. These present different characters according to the stage of the disease or the intensity of the morbid process. They may be either coarsely granular, finely granular, or partly granular, and transparent, or completely hyaline. Associated with these casts are various cell-structures — blood-cell, epithelium-cell, compound granule-cell, and various modifications of cell-growth, as may occur during the advance of acute or chronic disease. When hæmorrhage takes place in the uriniferous tubes from rupture of the Malpighian capillaries, the coagulum formed is washed away, and appears in the urine as a cylindrical cast of a granular appearance, entangling a number of blood-cells within its mould. These casts are usually stained of a red colour by the hæmatin of the effused blood. As the hæmorrhagic state subsides, epithelial cells appear in the cast, mixed with blood-corpuscles, which gradually become less numerous, and ultimately disappear. In favourable cases the casts become more and more finely granular, and daily more transparent, and the epithelium-cells less and less abundant. In other less favourable cases various modifications of cell-development occur; resplendent granules appear, and free nuclei accompany the cast; compound granule-cells are discharged, abortive epithelial cells appear, with disintegrated granule-cells in the shape of grape-like clusters of nuclei; and other evidence of cell-transformation indicates the nature and direction which the renal disease may be taking. It is the *progressive* and day by day, or week by week, alteration of cell-formation which accompanies the casts of albuminous urine which must be watched and examined, day by day, to ascertain the progress of the disease.

The first departure from the healthy type of the secreting kidney-cell is evinced by its becoming more granular, the single nucleus being either obscured or accompanied by other nuclear granules. The cells become larger and larger, and present a cloudy appearance; for a larger quantity of material than usual has been taken up by the cells. The entire uriniferous tubules are thereby rendered broader, and even to the naked eye such a tube looks white and opaque. The individual cells are difficult to isolate, because they adhere closely together, in consequence of the alteration of cohesion they have undergone. The few granules normally present in the cells have accumulated and multiplied in greater and greater numbers the greater the energy with which the morbid process is being carried on, so that even the nucleus gradually grows indistinct. This is the condition described by Virchow as that of "*cloudy swelling*" (trübe Schwellung), fig. 73. It is the progressive and constant increase of such compound granule-cells in the urine, or of cells becoming more degenerate and compound, with a proportionate decrease of epithelium, which is the index of advancing disease. The occurrence of such cells is usually preceded by a state of inflammatory congestion; and in proportion to the quantity of these cells, and the degree of degeneration to which they have advanced, so is the degenerative process in the kidney indicated. When groups of grape-like (botryoidal) clusters of resplendent granules appear in the urine, they are generally the nuclei of various cells, and indicate a deteriorated or atrophic state of cell-development, which eventually becomes so powerless that nothing remains but a heap of granules. These accumulating in the tubes and Malpighian bodies, eventually constitute a fatty degeneration of the whole kidney. The occurrence of such clusters in the urine is generally associated with a subacute process of disease; and when the clusters become daily more numerous, they furnish very unfavourable indications. The large compound cell or inflammation-globule of Gluge never makes its appearance except as a sequel to a state of hyperæmia. There is a period in favourable cases where little or no epithelium is visible, and where transparent hyaline or waxy casts are alone seen. They are more abundant and more frequent in the chronic form of Bright's disease than in the acute; and are then present in the urine of the mildest and most tractable cases equally with the severest and most fatal. Their particular import depends on the cell-structures with which they are associated, and likewise on the relations as to time which other kinds of casts bear towards them. Although they have been named hyaline, yet occasionally they contain traces of granular matter or cell-elements. If they appear in the urine very shortly after hæmaturia, they may be accepted as indicating a state of catarrh of the renal tubes, which in all probability will prove remediable and tractable. In such cases an epithelium-cell is seen here and there in the urine, and sometimes a compound granule-cell; but these are not numerous, and do not increase. In other cases the glairy moulds of the tubes contain numerous abortive cells in almost daily increasing numbers. Compound granule-cells are then always present, clusters of granules (botryoidal or crescentic), and free nuclei are seen. These appearances are characteristic of a chronic subacute process, which progresses slowly, very insidiously, and is too often irremediable.

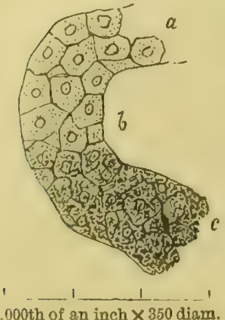

Fig. 73.*

In a third variety of Bright's disease where these hyaline casts are seen, few or no epithelial cells are present, but the appearances are characteristic of broken-up cell structures; clusters of granules are seen, having more and more the appearance of fat-granules. Many acquire a large size, and contain oil in abundance. In the more advanced cases the casts seem made up of these fatty and oily materials, and hence they have acquired the name of fatty or oily casts. They indicate the highest state of fatty degeneration.

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* Convoluted urinary tubule from the cortex of a kidney in Bright's disease (after Virchow). (*a.*) Tolerably normal epithelium; (*b.*) State of "*cloudy swelling;*" (*c.*) Commencing fatty degeneration and disintegration.

There is still a fourth variety of the hyaline or transparent cast, and which, from the absence of all structure visible in it, has received the name of waxy cast. This cast may be formed either in the smaller convoluted tubes or in the larger straight tubes of Bellini. They seem to be composed of some viscid material, and become faintly granular on the addition of dilute acetic acid.

The following may be stated as a general summary of the results relative to casts in the urine in Bright's disease:—

The blood casts represent more or less active hyperæmia and hæmorrhage from the kidney.

The coarsely granular epithelial cast, with its compound inflammation-corpuscle, and accompanied by amorphous granular flakes stained with hæmatin, represent the period of inflammatory exudation.

The finely granular semi-transparent casts, with scattered epithelium and granule-cells, represent the period of subsidence of the inflammatory process.

The transparent casts, with compound cells, or with isolated transparent molecules and grape-like clusters of granules, represent a stage of chronic subacute disease of very grave import; and if these casts become more and more loaded with large and gradually increasing fat-granules and oil-drops, the progress of fatal fatty degeneration is clearly marked (BASHAM).

Deteriorating conditions such as have been here described are not limited to the kidney in Bright's disease. There is reason to believe that the nutrition of most of the textures and organs of the body proportionally fails; and although not so apparent, because not manifested in a manner capable of being demonstrated during life, yet the tissues of every organ become more or less degenerate and inefficient for the purposes of life. The cells of the liver are invariably loaded with an abnormal amount of fat in all fatal cases of Bright's disease; and the heart-fibre and arterial textures exhibit the microscopic characters of atrophy and granular or fatty degeneration. Inflammation of the serous surfaces is a common complication.

The origin of chronic Bright's disease cannot be traced with certainty in all cases. It is one of those diseases, like phthisis, in which the patient rarely applies to the physician till the disease has made considerable advances, often beyond any remedy or means of cure. The commencement of it cannot be recalled or described by any other terms than "a gradual failure of the general health," which is usually designated as "a breaking-up of the constitution." If the urine be examined at the earliest stage, the presence of small quantities of albumen may be detected, and the sediment shows examples of granular casts, with more or less decayed and broken-up cell-structure. These phenomena often manifest themselves long before any dropsical symptoms point to the existence of renal lesions. There are also exceptional cases in which the urine at this early stage is non-albuminous.

It is important to bear in mind the relation of the hyaline, transparent, and waxy casts to the early and remediable forms of Bright's disease. In favourable cases such casts become more and more translucent; but if the disease advances, numerous cell-forms occur, and are passed along with it; and when the casts continue to present week after week a great number of free nuclei, associated with hyaline cylinders—numerous compound granular cells, some with, some without, cell-walls, —the progress of renal degeneration in the most obstinate and intractable form is fatally progressing.

Without a microscopic examination of the urine from day to day it is impossible to distinguish between a case likely to improve under treatment, and one which may be viewed as hopeless; and without the daily use of the miscroscope the treatment becomes at the best but merely guess-work.

Treatment.—It is only the general principles which can be indicated, inasmuch as every case requires a special study, and a line of treatment in detail peculiar to itself. Whatever treatment be adopted, a long time is necessary before any appreciable results are obtained, and therefore it is necessary to persist in one line of treatment steadily from week to week, and even from month to month. It is obviously of great importance, therefore, to be as accurate as possible in diagnosis as to the probable state of the kidney, so as to define the line of treatment from the first which may seem best adapted for the individual case.

It is a question of very grave importance how far vomiting or diarrhœa ought to be checked. If either of these occurrences are suddenly stopped, the gastric and intestinal membrane acting at the time as an emunctory for the urea and other excreta of the urine, the patient may be suddenly cut off by convulsions, apoplexy, or effusion into some of the serous cavities, such as the pericardium, or the pleuræ, or the ventricles of the brain. It is necessary, therefore, in the first instance, to determine in all chronic cases the particular organ or tissue which seems in each case to be acting vicariously. The perspirations are often spontaneously profuse; and the skin is by far the safest emunctory for the vicarious elimination of urinary constituents. Therefore it is important to promote the action of the skin if it be deficient, and to encourage it even if it is already considerable. Diaphoretics are always of essential service. The best are Dover's powder, the warm bath, warm clothing, and, for convalescents especially, a moderately warm climate. In Sir Robert Christison's experience they have always appeared most serviceable when they are so given as to excite a gentle perspiration during a part of the night. So it is also safe to promote the discharge of secretions from the intestinal canal, with due caution that they do not become excessive, so as to pass into permanent diarrhœa. Urea and other constituents of the urine are found in such discharges in large proportions. When general anasarca prevails, absorption may be promoted by gentle pressure, which at all times must be very cautiously applied, and the effects closely watched, for such effusions afford great temporary relief to important symptoms which indicate the involvement of vital organs. Bandaging to promote absorption is not justifiable so long as the anasarca is increasing. Patients ought to be encouraged to go about as long as they are able, care being taken that they are clothed with flannel and woollen garments, and otherwise well protected from chills or draughts of cold air.

The quantity of urea passed by the urine should be determined daily, to ascertain how far the kidneys are capable of secreting and eliminating these excrementitious products. According to the results obtained, the diet must be regulated, and such measures taken as are calculated to reduce the quantity of urea, and other constituents formed daily, to the capacity of the diseased kidneys for the work they are able to do.

In the subacute forms of the disease the action of the skin is especially to be promoted by such saline remedies as the *acetate* or *citrate of ammonia*, to which may be added *one, two*, or *three drachms of the infusion of digitalis*, and *ten* or *fifteen minims of antimonial wine.*

In such cases, also, *ten* or *fifteen minims* of the *tincture of the perchloride of iron*, or from *five* to *ten* grains of the *citrate of iron and ammonia* may be given *every day* with one of the meals. *Every second day* a dose of the *compound jalap powder* may be given; and if hyperæmia of the kidneys prevail, it may be necessary to take from four to six ounces of blood from the loins by cupping or by leeches. As the urine becomes more free from blood-corpuscles and albumen, the iron medicines may be more frequently given, and the compound jalap powder less frequently.

A very good formula for the administration of salines and iron in the subacute and chronic cases is that given by Drs. Basham and Goodfellow. It is as follows:—

Liquor Ammoniæ Acetatis, ʒi. – ʒii.; Acidi Acetici diluti, ♏ xx. – ♏ xxx. – ♏ xl.; Tinct. Ferri Perchloridi., ♏ x. – ♏ xv.; Aquæ, ℥i. – ℥iss.; *misce*, fiat haustus.

Small doses frequently repeated seem to do better than larger ones given at longer intervals. These remedies tend to lessen the watery state of the blood; and the action of the chalybeate medicines is of no avail till after purgation has been free, and when the hot air or warm baths have caused the skin to act freely. A nutritious diet is then to be given, combined with the chalybeate remedy.

The further treatment of the chronic forms of Bright's disease is influenced by the amount of the anasarca present. If considerable anasarca is present, neither cupping nor leeches can be had recourse to, on account of the bruising and erysipelatous inflammation they are apt to induce. The chalybeate mixture already mentioned is still found of service, taken at least three times daily, and to it may be added from time to time *fifteen* or *twenty* minims of the *spiritus ætherus nitrici* (GOODFELLOW). The *citrate of iron and ammonia* in from *five* to *ten* grain doses, with the *sulphuric* or *chloric ether*, are also good and useful remedies; and if the iron medicines are found too stimulating, *sulphate of zinc*, or *tannic* and *gallic acids*, may be used instead. The *sulphate of zinc* is to be given in doses of *one* to *three* grains three times a day, in the form of a pill, made with *extract of hop* and with or without a grain of the *extract of nux vomica*, or the same dose of *sulphate of zinc* may be given in a draught combined with *sulphuric* or *chloric ether* (Goodfellow). These latter medicines relieve considerably the flatulence and sensation of coldness in the stomach and bowels, so much complained of in such cases.

If the urine is "smoky," or if blood is seen on microscopic examination, the *gallic acid* in *five* or *ten grain* doses may be given, with a few drops of *diluted sulphuric acid* and a few drops of the *tincture of hops*, or other aromatic vegetable tincture, in an infusion of the same. The objection to these remedies is the constipation they are apt to induce. The bowels should always be kept relaxed, two or three loose evacuations being secured daily. The medicine most generally useful is the *compound jalap powder* of the London Pharmacopœia, which should be taken in the morning fasting, in half drachm or drachm doses, in a wine-glass of water. It does not induce prostration; but by repetition it is apt to lose its effect, when *elaterium* may be necessary in small but repeated doses; *two grains of elaterium* being dissolved in *sulphuric ether*, and *the eighth part of a grain* given every six hours till the desired effect is produced. Very abundant watery purgation is thus obtained, and a sensible impression is made upon the distended dropsical parts (Basham).

If *elaterium* may not be deemed advisable, a saline draught of the following composition may prove efficient:—

℞. Magnes. Sulph. vel Sodæ Sulph. ʒi. to ʒij.; Ætheris Sulph., ♏ x.; Acid. Sulph. dil., ♏ x.; Ferri Sulph., gr. i. to gr. ii.; Aq. Menthæ Vir., ℥iii. to ℥iv.

This draught is to be taken the first thing in the morning once or twice a week. It ought to produce two or three loose and watery evacuations (Goodfellow).

Diuretics are a most certain and speedy remedy when the *dropsical* effusions are considerable; and they are sometimes advisable when the urine is very scanty, although they are seldom necessary in the treatment of the fundamental disease. Sir Robert Christison is convinced that the fears entertained by some of injury being produced by the stimulus of diuretics on the kidneys are visionary. In his experience, which now extends over more than forty years, the best diuretics are *digitalis, squill,* and *bitartrate of potash,* taken simultaneously; and, if these fail, Hollands sometimes speedily establishes the diuresis.

When dyspeptic symptoms predominate, and there is considerable flatulence, the following pill is recommended by Dr. Goodfellow to be taken twice or thrice daily:—

℞. Ferri Sulphatis, gr. i.; Ext. Nucis Vomicæ, gr. ½ to gr. i.; Mas. Pil. Galb. Co., gr. ii. to gr. iii.; *misce,* fiat pilula.

And if there be co-existent bronchitis, a draught of the *acetate of ammonia,* with *ten, fifteen,* or *twenty* drops of the *spiritus ætheris nitrici* and half a drachm of the *oxymel of squills,* is to be taken intermediately with the pills. If much nausea prevail, *three minims* of *dilute hydrocyanic* *acid* may be added to the draught, with the occasional application of mustard to the stomach.

Want of sleep is often complained of, but opiates are inadvisable, because they are apt to check the secretions and to occasion constipation. *Henbane* may be given in place of *opium.*

The patient should select a residence where the soil is sandy or chalky, where the air is mild and dry, so that as much open air exercise may be taken as possible.

Diet should be light, but nutritious; no pastry should be eaten; and stimulant liquors must be used with caution, although, when exhaustion is great, their use may be unavoidable. The principal meal should be in the middle of the day, and not later than three o'clock; and the last meal should be taken two or three hours before bedtime, retiring early to bed, and rising early. Light but warm woollen clothing is to be always worn in summer as well as in winter.

The complications of Bright's disease are extremely difficult to manage.

The *diarrhœa* must not be suddenly checked. Thirty to sixty minims in water of the *spiritus ammoniæ aromaticus,* with half a drachm of the *tincture of kino* or of *catechu,* after every loose stool, will in general be all that is necessary. If there be much griping, the application of a lintseed poultice over the abdomen, with two drachms or half an ounce of *tincture of opium* sprinkled over it, will give relief (GOODFELLOW). When it is found necessary to use measures for checking the diarrhœa, means should be taken that the skin acts freely, or that the urine flows increased in quantity. Lead and opium pill (*pilula plumbi cum opio*), in doses of five or ten grains twice or even thrice a day, is the best remedy for checking diarrhœa. These remedies may be aided by a *suppository* of the *hydrochlorate of morphia* occassionally, each suppository containing a quarter of a grain of the hydrochlorate of morphia (*morphiæ hydrochlor. suppositoriæ*). Vomiting may be controlled to some extent by *bismuth,* but more frequently by *morphia, hydrocyanic acid, creosote, rectified pyroxylic spirit, chloroform,* or *chlorodyne,* or little fragments of ice; and if these fail, a blister over the epigastrium has sometimes succeeded (SIR ROBERT CHRISTISON).

Intercurrent inflammatory attacks and effusions into cavities are still more difficult complications to manage, and are very dangerous to life. The application of a few leeches, or the abstraction of a few ounces of blood by cupping, may be borne well in the cases with head complications; but the greatest care and caution is required in the employment of similar remedies in the complication of *pericarditis,* on account of the great danger of death by syncope.

Bronchial complications are serious, and more or less constantly present; and they are frequently the immediate cause of death in chronic Bright's disease. The least stimulating expectorants may be administered under such circumstances. The following formula is recommended by Dr. Goodfellow:—

℞. Liquor Ammoniæ Acetatis, ʒii. to ʒiii.; Spiritus Ætheris Nitrici, ♏xx. to ʒss.; Oxymellis Scillæ (*Lond.*), ʒss.; *Aquæ* Camphoræ et Aquæ, ā ā ʒv.; *misce,* fiat haustus. To be taken every four, six, or eight hours.

If the expectoration be viscid, and difficult to discharge, a few drops of antimonial wine may be added to the draught; or if there be much spasm of the bronchial tubes, as indicated by the asthmatic breathing, a few drops of *sulphuric* or *chloric ether* may be given. If, on the other hand, the expectoration be purulent and difficult, a *few grains of carbonate of ammonia* may be given with the *oxymel of squills.*

Every precaution ought to be taken to prevent a fresh attack of bronchitis. So long as the patient is able to bear it, the *habit* of cold sponging and dry rubbing of the surface of the body every morning with a flesh brush or coarse towel is the best preventive, and being clad in warm woollen clothes. Flannel next the skin must be invariably insisted on.

Mercurial preparations are contra-indicated in Bright's disease—so great is the tendency to salivation and the otherwise injurious effects they produce. *Podophyllin* (the active principle of the May apple) may be used instead. Its dose is from $\frac{1}{4}$ to 1 grain; and it ought not to be given in doses larger than one grain. It is best given in the form of a pill combined with soap and hyoscyamus. As an occasional mild aperient pill, the following is also found to answer well:—

℞. Mas. Pil. Rhei. Comp., gr. ii. to gr. iii.; vel. Ext. Aloes Aquosi, gr. i.; Ext. Nucis Vomicæ, gr. i.; Mas. Pil. Galb. Co., gr. ii.; *misce* fiat pilula (GOODFELLOW).

SUPPURATIVE NEPHRITIS.

LATIN EQ., *Nephritis suppurans;* FRENCH EQ., *Néphrite supurée;* GERMAN EQ., *Interstitielle Nephritis;* ITALIAN EQ., *Nephritide suppurativa.*

Definition.—"*Inflammation, with suppuration of the substance of the kidney.*"

Pathology.—The general nature of inflammation having been fully considered in volume first, it is of importance, in studying the inflammations of the kidney, to determine—(1.) The tissue or tissues affected —vessels, parenchyma, or interstitial connective-tissue; (2.) Whether only the cortical substance, or the pyramidal, or the pelvis of the kidney, is affected, or all parts together; (3.) Whether the affection is partial or diffused.

In the commencement of the disease a definite tissue can generally be indicated by microscopic examination after death, as the starting-point of the affection; but later it is not so easy. The several tissues are often consecutively attacked.

There are two essentially different affections,—(1.) Parenchymatous nephritis; (2.) Interstitial nephritis; and these may be complicated with the fatty degeneration or the lardaceous disease already noticed. All forms of disease may be present at once, sometimes one and sometimes another occurring first; but *parenchymatous nephritis* is most generally the primary affection (Rasmussen, *Med.-Chir. Review,* July, 1863).

The cells of the convoluted tubes appear to be larger and richer in albumen than those in the straight ones; and any disease of the former, rendering them inactive, is of far more serious import than disease of the latter, producing an actual change in the urine. Affections of the pyramidal portion of the kidney only are usually of a catarrhal nature, and are described by Rasmussen as "*papillary catarrh*," or "*catarrhal nephritis*." The catarrh affects principally the straight canals and papillæ, and is often continued from the bladder and urethra. Its exciting causes are stimulant, alcoholic, or terebinthine drinks, the use of cantharides, or of acid diuretics. It may be the starting-point of parenchymatous nephritis, and often complicates it. Such cases are generally suppurative, especially when secondary to bladder inflammation, or paralysis of the bladder in spinal affections. The suppuration is then generally most obvious and active in the cortical portion of the organ. The kidney is enlarged, and its surface covered with minute points of pus or small abscesses. A section of the whole kidney shows that these abscesses are diffused throughout the cortical, to the exclusion of the pyramidal part. The mucous membrane of the pelvis is inflamed, and is described under the name of pyelitis (WILKS). When the capsule is highly vascular and involved in the process, it indicates a chronic affection, or is a result of a former inflammation. In cases of uncomplicated catarrhal nephritis, where suppuration does not occur in the cortical portion, the lesions are confined to the canals and papillæ. They present a whitish or yellowish striation, with hyperæmia of the intervening vessels, and bloody ecchymosis may be present over the whole kidney, especially if acid diuretics have been largely given. If the disease continues long, the distended urinary canals press on the blood-vessels, when the hyperæmia generally ceases. The productive effects of the inflammation are limited to cell-growth (nucleated, club-shaped, fusiform, or ramifying), mixed with mucous catarrhal products (mucin), sometimes combined with fatty metamorphosis or other degeneration of epithelium.

True parenchymatous nephritis is a hypertrophy or "cloudy swelling" (see p. 1098) of the large cells of the convoluted tubes. The cells take up large quantities of the *albuminates*, becoming distended, turbid, granular, and closely adherent to each other. Subsequently all the cell-forms vanish, and a large granular fatty mass is set free, which generally forms into "inflammatory globules." It often co-exists with "interstitial nephritis." When catarrh exists, it is usually associated with the symptomatic phenomena of Bright's disease. Retardation of venous blood in the kidney is a common event in parenchymatous nephritis. Thrombi are then apt to form in the veins of the kidney, and being transported to the *vena cava* and heart, may find their way into the lungs. In the advanced stage of the disease, resolution or recovery corresponds to fatty degeneration of the cells, sometimes with loss of substance apparent on the surface, indurations, granulations, cysts, in connection with or separated from the urinary canals. The interspaces between the granular elevations consist of empty collapsed tubes. The granular cirrhotic appearance of the kidney is not similar in pathological origin to that of the liver. In the liver it is the inter-acinous connective-tissue which is first affected and causes the cirrhotic granulations; in the kidney, on the contrary, the parenchymatous cells of the tubes become first affected, and the lesion is afterwards complicated with *interstitial nephritis.* The Malpighian glanduli ultimately become small, corrugated, and surrounded by thickened capsules of connective-tissue. These capsules, with the epithelium, sometimes eventually become fatty, lardaceous, or calcareous. With regard to *interstitial nephritis,* it has been doubted whether there is a connective-tissue or fibrous matrix to the kidney. I have never yet seen a human kidney in which it is not demonstrable. It becomes immensely hypertrophied, and minute cells grow up in its substance in large numbers, even to the extent of suppuration. Atrophy is also a usual result, first of all at the expense of the cortical part. On section it is seen to be diminished between the pyramids and the surface. The edge is irregular, and the surface is granular. The granulations are composed of bundles of tubes lying between veins, and are not to be confounded with white specks of deposit. The capsule separates with great difficulty, tearing the tissue which adheres to it. The glomeruli appear to be much closer together than natural (Dickenson, *Med.-Chir. Trans.*, 1863). Cysts are frequently met with on the surface.

Sometimes the whole of the new interstitial material undergoes the fatty degeneration. Granulations are also formed by the connective-tissue contracting round the canals and glomeruli, and circulation is more or less obstructed. The tubes become constricted in a bead-like manner, and the *tunica propria* is thickened and streaked. The glomeruli become small, homogeneous, and more or less fatty.

Circumscribed interstitial nephritis often affects the pyramids only, as a result of syphilis; nodes are the consequence, which, passing away or diminishing, leave cicatrix-like depressions not unlike those from hæmorrhagic infarctions (RASMUSSEN, WILKS, VIRCHOW, DICKENSON).

The kidneys may suppurate—(1.) From morbid conditions of the blood; (2.) From external violence; (3.) From retention of urine; (4.) From calculi in the kidney (JOHNSON, *op. cit.*, p. 417).

Symptoms.—Uncomplicated acute nephritis has hitherto been considered so rare that its symptoms have not been well described. Those mentioned by Baillie are as follow:—“When the kidneys are inflamed, more or less pain is felt in the region of these glands, and the pain commonly shoots along the ureters. There is a sense of numbness down the thigh, and in the male there is often retraction of the testicle, or a feeling of pain in it. When one kidney is affected, these symptoms are only felt on that side. The urine is voided frequently, and is sometimes of a pale, but more commonly of a deep-red colour. There is sickness and vomiting. The bowels are at the same time often costive, and subject to collicky pains. These symptoms are accompanied by more or less fever.” “When pus is formed, the event may be known by the pus being mixed with the urine.” Cases related by Mr. Stanley, however, by no means bear out this description. He gives the case of a man who had retention of urine in consequence of a gonorrhœal discharge being stopped by injections. In this instance the kidneys were found extremely vascular and soft, with numerous minute depositions of pus throughout the cortical and tubular parts, and the infundibula and the pelvis were likewise filled with pus. The principal symptom was severe pain at the fifth lumbar vertebra. In another similar case, but not quite so acute, the kidneys were found so dark coloured as to be almost black, and at the same time remarkably flaccid. This patient died paraplegic, the loss of motion being complete, and that of sensation nearly so.

If nephritis passes to a chronic suppurative state, the pain in the loins is often severe and the appetite impaired, while pus is found often to a considerable amount in the urine, and if a calculus or gravel be the immediate cause, the urine may contain large portions of those substances mixed with blood. Dr. Johnson has clearly shown that the desquamative and suppurative processes in the kidney are much more closely allied than is generally supposed. They pass into each other by almost imperceptible gradations; the products appearing in the urine as purulent casts of the tubes.

Prognosis.—Acute affections of the kidney are in all cases of grave prognosis. The chronic forms of these affections are perhaps consistent with life, but in every case they greatly impair it, and are ultimately the cause of premature death. When pus-corpuscles take the place of renal epithelium, the prognosis must always be unfavourable; and the suppurative process in the kidney is the most rapidly destructive of the lesions. Dr. Johnson has not met with a case of suppurative nephritis which has terminated in recovery (*op. cit.*, p. 440).

Treatment.—The treatment of acute nephritis must be according to the ordinary principles of the treatment of inflammation by evacuants and opiates. Blisters in such cases are dangerous, and ought to be avoided. The neutral salts, with opiates, are admissible in some cases. Castor oil or other purgative substances which do not act so immediately on the kidneys are more useful, such as compound jalap powder or podophyllin. The general plan of treatment is similar to that which has been already stated.

HÆMATURIA RENALIS.

Latin Eq., *Hæmaturia renalis;* French Eq., *Hématurie renalis;* German Eq., *Blutharnen,*—Syn., *Nierenblutung;* Italian Eq., *Ematuria renale.*

Definition.—*Hæmorrhages in which blood is mixed with the urine, whether it proceeds from the kidney, ureter, or bladder.*

Pathology.—We are often unable to trace whether the blood passed with the urine has flowed from the *kidney, ureter,* or *bladder.* In some cases, however, when hæmorrhage has taken place from the kidney, a small clot remains after death, to mark the seat of the disease; also when it proceeds from the bladder, the coats of that viscus, though often pale, are in a few instances red, congested, and some blood exudes from them on pressure. The most usual organic diseases with which hæmaturia is associated are, *Bright's kidney,* especially *acute desquamative nephritis, fungus hæmatodes,* either of the kidney or bladder, *nephritic* and *vesical* calculi, and cancer of the bladder.

Intermittent hæmaturia is now also a recognized morbid condition (Rayer, Elliotson), generally associated with malaria.

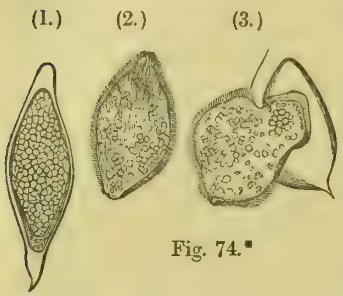

Fig. 74.*

Dr. Harley has described a form of hæmaturia already referred to in connection with the occurrence of the *distoma hæmatobium.* Its ova are then found in the urine; and its occurrence is endemic in Egypt, South Africa, and the Mauritius. (Fig. 74.)

Symptoms.—The hæmaturia may take place suddenly, or it may be preceded for a short time by pains in the loins, epigastrium, or bladder; and a burning pain is experienced on passing urine, which contains more or less blood. Sometimes the blood is deposited in clots in the bottom of the vessel, but more frequently it throws down small portions of fibrine like the sediment from beef-tea. The urine is always of a smoky hue, or of a black colour, and albuminous; so that the blood is equally mixed in such cases, and presumed to come from the kidney. Casts of renal tubes also indicate kidney lesion (blood casts). In grave cases, especially in old persons with disease of the prostate, the blood may coagulate in the bladder and render it both necessary and difficult to pass the catheter.

The hæmaturia often continues for many days, and even weeks together, and the quantity of blood passed, though often trifling, yet occasionally amounts to some ounces in the course of the twenty-four hours. The general symptoms depend, as in other hæmorrhages, on the quantity of blood lost, but in general the termination is a return to health. In other cases, however, the patient sinks, as in *toxic* hæmaturia, preceded by a comatose or typhoid state.

Diagnosis.—No certain symptom has yet been observed by which we can determine the particular seat of the hæmorrhage. We should be careful not to confound urine greatly loaded with uric acid with hæmaturia.

Prognosis.—Idiopathic hæmaturia is rarely a grave disease, except it arises from disease of the kidney, such as *carcinoma,* or other structural disease of the urinary organs, when it is the precursor of a fatal event.

John Hunter regarded turpentine as one of the best styptics; although now perhaps it is superseded by *perchloride of iron, gallic* and *acetic acids.*

Causes.—The usual causes of hæmaturia are, blows on the back or loins, the existence of renal or vesical calculi; also granular degeneration of the kidney, diseases of the bladder, and some morbid poisons, as that of *small-pox* or of general blood diseases, such as *rheumatism,* or *scurvy; cantharides* and the *turpentines* are also said to act specifically in the production of hæmaturia.

Children seldom suffer from this affection.

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* (1.) Ovum of *Distoma hæmatobium* from hæmaturia of the Cape of Good Hope (Dr. John Harley); (2.) Embryo (ciliated) from ovum capsule; (3.) Embryo attached to the ovum capsule.

Treatment.—*Tincture of the perchloride of iron* is one of the most useful of remedies, whether in debilitated patients or in those suffering from Bright's disease, and whether the blood comes from the kidneys or bladder. It is best given in doses of ♏ x. to ♏ xx., three or four times daily, in combination with *glycerine ;* and if there be much *cardiac* difficulty, with arterial excitement, it may be combined with *digitalis,* as in the following prescription:—

℞ *Tinct. ferri perchloridi,* ♏ xxx.; *Tinct. digitalis,* ♏ xv.; *Aq. menth pip., f* ℥iss., repeated every four hours.

Gallic acid in combination with *sulphuric acid* may also be given; as also *ipecacuanha, tannin,* and *acetate of lead* and *alum,* as already referred to under hæmatemesis.

Quinine and *arsenic* are the remedies indicated in malarious intermittent cases. Idiopathic hæmorrhage often readily yields to *bitartrate of potash* or to the *mineral acids.* Dr. Elliotson recommends the *ol. terebinthinæ,* in doses of ♏ x., ♏ xx., or ♏ xxx., every two, or three, or four hours.

Injections of cold water, or water in which twenty to forty grains of *alum* have been dissolved, into the bladder or up the rectum, and also a cold hip-bath are useful applications.

SUPPRESSION OF URINE—*Syn.*, ISCHURIA RENALIS.

Latin Eq., *Urina suppressa*—Idem valet, *Ischuria Renalis;* French Eq., *Suppression d'urine;* German Eq., *Harnvelhaltung;* Italian Eq., *Soppressione dell'orina*—Sin., *Iscuria renale.*

Definition.—*A complete or partial suspension of the functions of the kidney, by which the quantity of urine is greatly in defect, or its secretion entirely suppressed.*

Pathology and Symptoms.—There may be some pain in the back, or some irritability of the bladder; the patient becomes anxious and restless, till at last the brain is oppressed, and he dies comatose. In other cases there is nausea, with hiccough, and the body exhales an urinous odour. When the suppression is less complete, and depends on an affection of the bladder, the local sufferings of the patient, the forcing of the bladder, the tenesmus, and the general irritation of the poor sufferer are most severe and distressing.

When the composition of the urine is considered, the mortal effects of its suppression are easily understood. (See under Composition of Urine, p. 1041, *ante.*)

The time during which the urine may be suppressed, and yet the patient recover, is various. Children when teething often void only a few drops of urine, and that at several hours' interval. The urine passed at such times is extremely high-coloured, stains the linen, and is passed with great pain, the child crying bitterly as it scalds the surface over which it flows. In hysteria the urine is often suppressed for three or four days. A case in which no urine was secreted for nine or ten days is given by Dr. Laing, and yet the patient did well. As extreme cases, Dr. Parr, in his *Medical Dictionary*, mentions a patient who made no water for twenty-two weeks, while Dr. Richardson speaks of another who up to seventeen years of age had never passed a drop of water in his life. In this case the ureters must, as in birds, have terminated in the large intestine, and the urine have been passed with the fæces. In general, however, it may be laid down as a maxim, that when suppression of urine depends on any acute or severe disease the patient seldom survives this symptom more than three or four days. It is often the final result of such diseases as *cholera, typhus, scarlet fever*, and other blood diseases.

Among the symptoms of suppression of urine is an urinous odour of the perspiration from the axilla and umbilicus.

It is certain also that some cases are feigned; some women, for instance, are said to have had a vicarious discharge of urine from the stomach, and Nysten gives the case of a girl who vomited urine, but it was at length ascertained she first swallowed it. Rayer gives a similar case of a woman at La Charitié, who had an abdominal tumor, which was supposed to be connected with the kidneys. Many persons saw her vomit urine, and Guibourt detected it chemically in the matters thrown up, but it was found that she had first drank it, though for what motive, except notoriety, nobody could discover (DR. ROBERT WILLIAMS).

Causes.—This affection may be caused by disease of the kidney itself, or it may be secondary, and arise from disease in other parts of the body. Among the latter are injuries of the head or spine, an attack of *pneumonia*, when the patient will sometimes hardly pass a few ounces of urine in twenty-four hours, or of hysteria, when it is often suppressed for several days together, and also of inflammation or high irritation of the bladder. It may depend on inflammation of the kidney, caused perhaps by some poison acting on that organ, as that of *small-pox, scarlet fever, typhus*, or *cholera*, or else by *cantharides*, or *turpentine*. The presence of a calculus also in the kidney or the ureter is also an occasional remote cause.

All ages are liable to this affection; children from teething often suffer a complete suppression, or only pass a few drops of fiery urine in the twenty-four hours; the adult from gravel or stone; and the aged from disease of the brain, or cord, or Bright's disease.

Diagnosis.—Suppression is to be distinguished from mere retention of urine in the bladder, or from *ischuria vesicalis*, by there being no fulness in the vesical region, and by no urine flowing when the catheter is passed.

Prognosis.—Many cases recover from a suppression of urine of not more than twenty-four to forty-eight hours, but, except in hysteria, few survive if the disease continues a longer period.

Treatment.—When suppression does not depend on any morbid condition of the blood, and is primary, the patient should be placed in a warm bath, and be purged by substances that act on the kidney, as the neutral salts. Indeed, if the case be slight, purging by any cathartic is sufficient. If this method should not succeed, ♏ x. to xxx. of the *tinct. cantharides* should be tried every four or six hours, according to the urgency of the case. Many physicians, however, prefer a tonic treatment, as the *camphor mixture* and *æther*, or the *tinct. ferri muriatis,* ♏ xxv. to ♏ l.

Belladonna is also a useful remedy, and so also is *digitalis* applied as a fomentation of the fresh leaves over the abdomen; or an ounce of the tincture may be added to a warm linseed poultice; or the dried leaves may be made into a poultice, to which half an ounce of the tincture may be added.

It is chiefly cases in which the pulse is rapid that digitalis is suitable; and the urine will not begin to flow till the digitalis has reduced the action of the heart (J. D. Brown in *Medical Times,* January 25, 1868). In the suppression of urine attending cholera, Dr. E. Goodeve recommends the following:—

℞. *Tinct. Digitalis,* ♏ v. to ♏ x.; *Spr. Æther nit,* ♏ xxx.; *Liq. Ammon. Acet.,* ♏ lx.; *Aq., f*℥i.; *misce.* Such a draught may be taken every three or four hours. (*Reynold's System of Medicine,* vol. i., p. 183.)

The treatment of *symptomatic anuria* resolves itself entirely into that treatment which will remove the primary disease.

SECTION VI.—DISEASES OF THE BLADDER.

CYSTITIS—*Syn.*, CATARRH OF THE BLADDER.

LATIN EQ., *Cystitis*—Idem valet, *Catarrhus vesicæ;* FRENCH EQ., *Cystite*—Syn., *Catarrhe de la vessie;* GERMAN EQ., *Blasenentzündung;* ITALIAN EQ., *Cistitide*—Sin., *Catarro della vescica.*

Definition.—*Inflammation of the urinary bladder.*

Pathology.—The mucous membrane of the bladder is liable to the diffuse, the serous, the adhesive, the suppurative, and the ulcerative inflammations, and these may be either *acute* or *chronic.*

The inflammation may extend over the whole cavity of the bladder, or be limited to some portion of it, and the part most frequently inflamed is that near and around the neck. In this respect it follows the law of all hollow organs, namely, in that it is most liable to be diseased at its orifices. There is also another reason for this part being more frequently attacked than the rest, namely, as being liable to the occasional extension of inflammation from the urethra to this part.

"It is well known," says Dr. Baillie, that "the inner membrane of the bladder in the dead body hardly shows any vessels which are large enough to carry red blood in its natural state;" but when *diffusely* inflamed, it is crowded with a prodigious number of extremely fine blood-vessels, and among them may be seen small spots of extravasated blood. This state has many degrees, and the colour is usually of a venous red, while, in addition to this, the coats of the bladder generally are thickened. It may terminate by resolution, or it may pass into *serous* inflammation, or *catarrh of the bladder.* The mucus secreted in this latter disease is at first small in quantity and extremely fluid, but is deposited as the urine cools. At a further stage of the disease it becomes abundant and thickens, equalling or surpassing the urine in quantity, and which now resembles thick gruel, and is often mixed with blood, or gravel, or both.

Andral has twice seen the internal surface of the bladder coated with lymph more than a line in thickness, and similar to the false membrane of croup. The lymph thus effused sometimes becomes organized; and in this manner calculi have become encysted and removed out of the reach of the sound.

Inflammation of the mucous membrane of the bladder often terminates in suppuration, and pus to a considerable amount may then be passed. Occasionally, instead of suppuration taking place at its free surface, an abscess forms: in either case ulceration may take place, sometimes superficially, and sometimes so burrowing as to perforate the bladder, and form a communication between it and the neighbouring parts, as the cavity of the abdomen, the rectum, or the vagina. When the communication is formed with the general cavity of the abdomen, the urine escapes into it and produces general peritoneal inflammation.

The mucous membrane of the bladder is liable to similar chronic inflammation, sometimes retaining its normal colour, and at other times being grey, brown, or black; and has often acquired a double or even triple thickness (hypertrophy of the mucous coat). One of the most ordinary changes, however, in the bladder from its natural structure is hypertrophy of its muscular coat. In a natural state the muscular coat of the bladder, when it is moderately distended, consists of thin layers of muscular fibres running in different directions, and probably less than the eighth of an inch in thickness; it may be found, however, in some cases half an inch thick, owing, for the most part, to its efforts to overcome some resistance, as an enlargement of the prostate, or the presence of a calculus or a stricture in the urethra to the passage of the urine. In some instances these efforts of the bladder to evacuate its contents have led to the mucous membrane being protruded through the intermuscular spaces, forming a pouch or hernial sac, in which a small calculus has been imbedded, but this form of disease is extremely rare.

The mucous membrane of the ureter may participate in the inflammation of the bladder, and is liable to the diffuse, to the adhesive, to the ulcerative, and to the suppurative inflammations, and these also may be acute or chronic.

The ureter is occasionally found to be highly vascular, and of a deep venous colour after the passage of a calculus. There are repeated instances of adhesive inflammation of this canal. Andral quotes a case in which all the internal surface of the ureter was covered with a layer of lymph similar to the membrane in croup; and in some rare instances the ureter has been found obliterated or transformed into a fibrous cord. There are other well-marked effects of adhesive inflammation of the ureter, as when the delicate coats of this canal are increased, as in cases of severe chronic disease, to four to six lines in thickness. Suppurative inflammation also sometimes takes place in the ureter, and without breach of surface.

The **Symptoms** of inflammation of the bladder are pain felt in the perinæum and above the pubes, accompanied with a fulness or swelling, also frequent attempts to make water, which is evacuated in small quantities and with great pain, or there is a total retention of urine, with a strong desire to void it. The rectum is affected, from its connection with the bladder, with tenesmus, and the stomach likewise takes part in this disease, being affected with nausea or vomiting. In some cases these symptoms are accompanied with much constitutional irritation and by delirium. When pus is formed, it will be seen mixed in the urine evacuated. The slighter form of the disease, or cystirrhœa, is characterized by milder symptoms, which consist principally of local pain and irritation, and by the urine being loaded with mucus, which sinks to the bottom of the vessel, mixed with a large quantity of sandy precipitates either of the phosphates, of the urates, or of both. The symptoms of the other forms of disease of this viscus vary only in degree from those which have been mentioned. The symptoms of inflammation and of the other diseases of the ureter are probably the same as those of the similar diseases of the bladder, except, perhaps, that the pain is more strictly lumbar; and when these canals are greatly enlarged, it is possible they may be felt through the walls of the abdomen.

Diagnosis.—When the kidneys and ureters are diseased the bladder very constantly sympathizes with those diseases; and the affections of the bladder being much more painful than those of the ureters and pelvis of the kidneys, the sympathetic affection of the bladder is often mistaken for the primary disease. Morgagni first pointed out this fact, and he gives a case in which, from these sympathetic pains, it was believed that the patient laboured under disease of the bladder; yet after death the bladder was found perfectly healthy, while the kidneys were extensively diseased and filled with large calculi. Lowdell and also Howship give similar instances of the kidneys being diseased, when the symptoms of the bladder were so prominent as to be mistaken for the primary disease.

Prognosis.—The result of the acute forms of inflammation of the bladder or ureter is generally favourable. The chronic forms of *cystitis*, as of chronic vesical catarrh, are more formidable, and often ultimately cause the death of the patient; for as a rule *cystitis* is a disease of long duration.

Treatment.—*Purging*, together with *opiates*, *diluents*, and the *warm bath*, are the best means of curing the acute affections of this viscus that we meet with. Chronic inflammation of the bladder, and especially catarrh, is very difficult of cure, and often our best directed efforts are unsuccessful. Opium is the remedy of the greatest value, especially aided by hot hip baths, hot fomentations over the abdomen, and linseed-meal poultices, with or without mustard or turpentine, over the hypogastric region. Suppositories of morphia are also of great use, containing half a grain to a grain of opium; but if the pain be very great, Mr. Liston gave much larger doses—as much as two to four grains of opium, with ten to fifteen grains of extract of hyoscyamus, in a suppository at the hour of sleep. Alkaline and demulcent drinks should be at the same time given. The state of the urine is perhaps one of the surest guides in our attempts to cure the patient; and if the urine be acid, the best medicines are the *neutral salts* or the *pure alkalies,* with *opiates;* while, if the urine be alkaline, or greatly loaded with mucus, the *mineral acids* are of the most service, combined with an opiate. Thus the *infusio rosæ c. acidi sulph. dilut.* ♏ij. to ♏v. *c. magnesiæ, sulphatis* ʒj. *c. tinct. opii* ♏iij. to ♏v. every sixth hour, is one of our best and most useful remedies.

The remedies which have been mentioned, though highly useful, yet frequently fail, and in such cases tonics often succeed, and of these *salicine* is one of the best. It may be given in doses of ten grains every six hours with great chances of success. It must be admitted, however, that much difference of opinion prevails as to the best tonic remedy, some preferring *uva ursi,* others *pariera,* others the *turpentines,* as the *Canadian balsam,* and others again the *infusio diosmæ.*

In chronic catarrh, when the discharge is copious, the decoction of *uva ursi,* in doses of not less than half a pint daily is very soothing. Sir Benjamin Brodie considered it of most advantage in cases of irritability rather than of inflammation. He prescribed it in large doses from one to two drachms of the extract daily; or from eight to sixteen ounces of the infusion as a drink, made as follows:—

℞. *Fol. uvæ ursi,* ℥i.; *Aq. Fervid., f* ℥xviii. Macerate for two hours, and boil down to 16 ounces, and strain.

CHAPTER XXII.

DISEASES OF THE CUTANEOUS SYSTEM.

SECTION I.—GENERAL PATHOLOGY AND CLASSIFICATION OF DISEASES OF THE SKIN.

DISEASES of the skin have been regarded too much as a specialty; and only now are they beginning to be looked upon as a class of diseases whose pathology is capable of being investigated and studied like other diseases. The expressions of skin diseases are undoubtedly various in appearance; and the same disease does not always exist in the same simple or elementary form.

The classification of skin diseases hitherto in use is that which is comprehended in the eight orders of Willan and Bateman; and the characteristics of these orders are embraced in the following definitions of terms in common use in the description of skin diseases:—

ORDER I. PIMPLES.—*Papulæ* are simple solid acuminated elevations of the cuticle, resembling an enlarged *papilla* of the skin. They commonly terminate in a scurf, and sometimes, though seldom, in slight ulceration of its summit.

ORDER II. SCALES.—*Squamæ* consist of cuticle in patches, plates, or laminæ, in which the epidermic cells are morbidly adherent, hard, thickened, whitish, and opaque. These scales cover either small papillæ, red elevations, or larger deep red and dry surfaces.

ORDER III. RUSHES.—*Exanthemata* are composed of superficial red patches of irregular size, and variously diffused. They disappear under pressure, and terminate by desquamation.

ORDER IV. BLEBS.—*Miniature Blisters—Bullæ* differ from vesicles in size, being larger. A large portion of cuticle is detached from the skin by the interposition of a watery fluid, usually transparent. The skin is red and inflamed beneath the blebs.

ORDER V. PUSTULES.—*Pustulæ* consist in circumscribed elevations of the cuticle, and contain pus. They have red and inflamed bases, and are succeeded by an elevated scab, which may or may not be followed by a cicatrix.

ORDER VI. VESICLES.—*Vesiculæ* are small acuminated or orbicular elevations of the cuticle, containing lymph, which, at first clear and colourless, may become amber-coloured, opaque, or pearl-like. Vesicles
ORDER VII. TUBERCLES.—*Tuberculæ* are small, hard, indolent eleva-
are succeeded by a scurf or a laminated scab.
tions of the skin, sometimes suppurating partially, sometimes ulcerating at their summit.

ORDER VIII. SPOTS.—*Maculæ* are permanent discolorations or stains of some portions of the skin, often with a change of structure. They may be whitish, dusky, or dark.

Clear and accurate in detail, these anatomical definitions have not yet been surpassed, and have laid the foundation of the objective study of cutaneous diseases.

Such definitions have given the key to most of the classifications of cutaneous diseases, which are mainly anatomical. They do not attempt to throw any light on the causes producing the various diseases, which are contemplated as so many distinct and individual "unities," their mutual relations being of secondary importance.

The basis of classes, as proposed by Willan and Bateman, furnishes outward marks or anatomical characters which are useful in describing the morbid anatomy of skin diseases; but it has no relation to the causes, to the pathology, nor to the treatment of such diseases. An affection which is papular to-day may be vesicular to-morrow and pustular eventually. Under the same division, or class, maladies are brought together which Nature has stamped with broad and obvious marks of distinction. Febrile diseases are associated with non-febrile; and ailments which are local and trivial are associated with diseases of grave import and deeply rooted in the system. On the other hand, distempers which Nature has plainly brought together and connected by striking analogies and resemblances, the methodical arrangement of Willan and Bateman puts widely asunder (WATSON).

Alibert conceived the idea of arranging skin diseases into natural families, of which he gives *twelve*. His classification pre-supposes a knowledge of the subject incompatible with teaching.

M. Hardy, of the Hôpital St. Louis, classifies skin diseases, according to their nature, into the following *ten* natural families:—(1.) *Macules* *and deformities;* (2.) *Local inflammations;* (3.) *Parasitic diseases;* (4.) *Eruptive fevers;* (5.) *Symptomatic eruptions;* (6.) *Dartres, or Tetters;* (7.) *Scrofulides, or strumous eruptions;* (8.) *Syphilides, or syphilitic eruptions;* (9.) *Cancers;* (10.) *Exotic diseases.*

In an admirable paper "On the Theory and Classification of Inflammations of the Skin," by the late Dr. A. B. Buchanan, Physician to the Dispensary for Skin Diseases in Glasgow, it is shown that any classification resting on one principle of division only, runs the risk of being, to a greater or less extent, *artificial* and untrue; and to secure a *natural* system, several principles must be taken into account, though greater importance may be attached to some of these than to others (*Edin. Med. Journal*, January, 1863). Skin diseases, like all other diseases, he considered, ought to be classed according to their *nature* or *pathology.* The cause of a disease, when known, gives a more accurate indication of its true nature and its means of cure than a knowledge of its anatomical appearances or symptoms, which are mere effects of the cause. So far as can be done, therefore, Dr. Buchanan proposed that skin diseases should be classified according to their causes. Not knowing the cause, some other principles must be sought for under which groups may be formed; and he found—in the pathological processes recognized as *inflammations, new formations, hæmorrhages*—that skin diseases might be arranged into three or more groups.

The inflammatory skin diseases are those which affect the "*pars papillaries*" of the corium, as well as the "*pars reticularis*" and the subjacent connective-tissue; but the process is not confined to these, for it affects even the epidermis, whose cells are changed in formation and in structure in the same way as all cells are altered by the process of inflammation.

The skin diseases characterized by new formations may be regarded as—(1.) The productive effects of inflammation; or (2.) The productive effects of such constitutional states as cancer, scrofula, and Bright's disease.

The hæmorrhagic skin diseases arise from the escape of blood in small quantities, owing to the rupture of capillary vessels, or to the escape of colouring matter along with the exudations. They are affections peculiar to the cutis; for, although blood may be effused among the epidermic cells, hæmorrhage can only originate from the cutis.

On these principles a classification somewhat of the following form has been suggested:—

CLASSIFICATION OF SKIN DISEASES (DR. A. B. BUCHANAN).

CLASS I.—Inflammations.

GROUP I.—SIMPLE INFLAMMATIONS (ALLIED TO *Simple Dermatitis*).

(1.) ERYTHEMA ([a.] *simplex;* [b.] *multiforme;* [c.] *chronicum*—comprehending *papulatum, nodosum, strophulus, squamosum, pityriasis furfuracea, membranacea, rubra*).

(2.) HERPES (*simplex* and *zoster*).

(3.) Urticaria (idiopathic, from ingestion of particular kind of food; from uterine affections; or persistent).
(4.) Dermatitis (idiopathic, as from *burns*, or from *frost-bite;* or symptomatic, as of *erysipelas;* or phlegmonodes, as *furunculus, anthrax, Aleppo tubercle*).
(5.) Pemphigus (benign, persistent, and foliaceous).

Group II.—Eczematous Inflammations (allied to *Eczema*).

(1.) Eczema (*erythematodes; E. papulosum,* comprising *lichen simplex* and *prurigo; E. vesiculare; E. rubrum; E. pustulosum,* comprising *impetigo sparsa, figurata,* and *pilaris; E. lichen; E. squamosum; E. pityriasis*).
(2.) Acne (comprising *A. simplex; A. pilaris; A. rosacea*).
(3.) Ecthyma ([a.] *simplex;* [b.] *chronicum = rupia;* [c.] *gangrenosum*).
(4.) Psoriasis (*punctata, guttata, nummularis, circinata* [lepra], *gyrata, confluens*).

Group III.—Ulcers.

(1.) Idiopathic.
(2.) Sympathetic.
(3.) Constitutional.

Class II.—New Formations.

Group I.—Homologous New Formations.

(1.) Epidermic (*epithelial growths,* comprising *callositus, clavus ichthyosis, cornu cutaneum*).
(2.) Pigmentary (*lentigo, ephelis, moles, melanosis, chloasma, silver stain, leucopathia*).
(3.) Dermic (*cicatrix normal,* or *cheloid, cutaneous tumor* [wens], *multiple tumors* [myscosis], *molluscum simplex, condylomata, verruca vulgaris, verruca mollis*).

Group II.—Heterologous New Formations.

(1.) Pseudoplasms (*lupus,* comprehending *maculosus, tuberculosis, hypertrophicus, exedens, serpiginosus;* and *lepra,* comprehending *maculosa, tuberculosa anæsthetica, exulcerans*).
(2.) Neoplasms (*epithelioma, carcinoma*).

Class III.—Hæmorrhages—e. g., *petechiæ, vibices, ecchymosis, purpura.*
Class IV.—Diseases of Accessory Organs—e. g., *hair, nails, sweat glands.*
Class V.—Diseases Defined by Uniform Causes.

Group I.—Parasitic Diseases.
Group II.—Syphilitic Eruptions.
Group III.—Eruptions of Specific Fevers.
Group IV.—Scrofulodermata.

In the following section, however, the names of skin diseases and the arrangement followed will be that which is given in the nomenclature of the College of Physicians.

Section II.—Description in Detail of the more Common Diseases of the Skin.

ERYTHEMA.

Latin Eq., *Erythema;* French Eq., *Erythème;* German Eq., *Erythema;* Italian Eq., *Eritema.*

Definition.—*Uniform redness simply, with puffiness of the skin, distributed in distinct patches of some size.*

Pathology.—It is accompanied by little constitutional disturbance; and if febrile phenomena are decided, it may betoken more severe areolar inflammation than *erythema*, and a more grave disease. It is thus apt to be mistaken for *erysipelas*.

The varieties of Erythema are,—*E. læve ; E. fugax*—Syn., *E. volaticum ; E. marginatum; E. papulatum; E. tuberculatum ; E. nodosum.* Of these varieties *erythema nodosum* is perhaps the most important. The indisposition which precedes the eruption is generally associated with a slight degree of fever. Red oval patches, considerably elevated and very tender, appear on the fore part of the legs, sometimes on the arms; and the long diameter of these patches is generally parallel to the axis of the limb. They sometimes form bumps an inch and a half long and an inch broad on the anterior aspect of the leg. After a few days the red colour changes to a blue, the patches become soft, and although something like fluctuation may be felt, yet suppuration does not occur. Thus the bumps of erythema gradually subside. Sometimes the disease is seen in feeble boys; but it is most common in young women, in whom it seems associated with disordered menstruation, or with rheumatism (RAYER, WATSON). In the chronic stage desquamation invariably occurs; and to this stage of the disease the name *pityriasis* has been given.

Treatment.—*Rest* and *quinine*, after aperients. *Carbonate of ammonia*, after gentle purgation, is also of benefit in doses of 5 to 10 grains three times a day. *Oxide of zinc*, in fine powder, dusted over the surface, will also sometimes allay the local irritation.

URTICARIA—*Syn.*, NETTLE-RASH.

LATIN EQ., *Urticaria;* FRENCH EQ., *Urticaire;* GERMAN EQ., *Nesselausschlag;* ITALIAN EQ., *Orticaria.*

Definition.—*An eruption of little solid elastic eminences, roundish or oblong, pale in the centre, and red at the circumference. These are commonly called "wheals," similar to the results from the strokes of a lash.*

Pathology.—The eruption is attended with an intense heat, a burning, tingling, or pricking sensation, very much like that produced by the stinging of a nettle.

There are several varieties of *urticaria*—(a.) *Urticaria acuta;* (b.) *Urticaria chronica.*

The acute forms are generally connected with the ingestion of some kinds of food—oatmeal, bitter almonds, shell-fish, &c. The more chronic and intermittent forms are associated with uterine or other affections.

Treatment.—Emetics and purgatives in the first instance; afterwards the correction of faulty digestion. The surface of the eruption may be dusted over with flour; or the following lotion may be used:—

℞. *Carbonatis Ammoniæ*, ʒi.; *Plumb. Acetatis*, ʒii.; *Aquæ Rosarum*, ℥viii.

In the chronic form especially associated with uterine irritation or ovarian tumors I have found *Bromide of Potassium* of much benefit. It ought to be given in a full dose of ten grains twice daily, which is to be doubled at bed-time.

Quinine is also useful in many cases of *Urticaria,* combined with *rhubarb* and *carbonate of ammonia.*

℞. *Sulphatis quiniæ*, gr. xii.; *Solve in Glycerinæ*, ♏ii.; *Pulv. Rhei.*, gr. xxiv.; *Carb. Ammon.*, gr. xviii.; *misce et divide in pil.* xii.—*Signa*, "*One three times a day.*"

LICHEN.

LATIN EQ., *Lichen;* FRENCH EQ., *Lichen;* GERMAN EQ., *Lichen;* ITALIAN EQ., *Lichene.*

Definition.—*An eruption commencing as small red papulæ, either isolated or confluent. These becoming excoriated, give vent to a serous fluid in considerable abundance, which ultimately concretes into a crust.*

Pathology.—This disease has sometimes been regarded as a form of *eczema.* It includes five forms or varieties,—(1.) *Lichen simplex;* (2.) *Lichen pilaris;* (3.) *Lichen circumscriptus;* (4.) *Lichen agrius;* (5.) *Lichen tropicus*—Syn., *Prickly heat.* If the summits of the papulæ become torn by the nails in scratching, yellow or blackish crusts form, due to the mixture of blood and serum, and abrasions of the skin occur. The disease is then known as *prurigo* (*eczema lichenoides* or *eczema pruriginosum*). It occurs more or less in all chronic cases of *scabies* or *phthiriasis,* and sometimes in *urticaria.* The disease attacks by preference the scrofulous and the debilitated, and is especially associated with improper, insufficient, or bad food. A too liberal diet, and stimulants in food or drink, similarly predispose to the disease. Exposure of the skin to the heat of the sun, or to acrid substances, also brings about the disease—*e. g.,* in cooks, grocers, bakers, and the like. Fissures of the skin are a most frequent complication, and occur in those situations where the skin is naturally thrown into folds, as at the arms, the angles of the mouth, the joints, the palms of the hands, and flexures of the fingers. They are apt to increase in depth as the disease continues, sometimes bleeding, and causing excruciating pain. When deep, the fissures are red and raw-looking, and serum or blood exudes from them, giving rise to crusts which partially fill them up (*eczema rimosum* of T. M. Anderson and Buchanan—the *eczema fendillé* of the French).

Treatment.—*Glycerine* in the following formula is recommended by Mr. Startin:—

℞. *Acid. Nit. dil.*, ʒss. to ʒi.; *Bismuth sub. nit.*, ʒss.; *Tinct. digital.* ʒi.; *Glycerini*, ℥i.; *Aq. Rosæ*, *f* ℥vii. ss.; "*To be applied as a lotion frequently to the affected parts.*"

The alkalies internally and externally are of service. *Bicarbonate of soda,* in fifteen grain doses in some bitter infusion, to be increased by eight grains daily, till ʒi. is taken in the twenty-four hours

(DIVERGIE). Alkaline, baths, and lotions are to be used at the same time—the lotions containing two to three drachms of the salt to twenty ounces of water.

PSORIASIS.

LATIN EQ., *Psoriasis;* FRENCH EQ., *Psoriasis;* GERMAN EQ., *Psoriasis;* ITALIAN EQ., *Psoriasi.*

Definition.—*An eruption characterized by the development of irregularly formed patches, slightly raised above the level of the skin, and covered with thin, dry, white scales.*

Pathology.—The affection comprehends *Psora leprosa,* and *Lepra vulgaris* or *dry tetter.* The term lepra is now quite given up on the continent, being restricted to true leprosy. The patches may be distinct, small, and scattered; or larger, confounded together, and irregular; or they may be so extended as to make a continuous surface. Hence the names of varieties of psoriasis—(a.) *Psoriasis vulgaris;* (b.) *Guttata;* (c.) *Diffusa;* (d.) *Gyrata;* (e.) *Inveterata.* The intense itchiness and the eruption are always preceded and accompanied by that form of dyspepsia or impaired digestion in which there is a superabundance of acidity, much formation of lithates, and an obvious constitutional tendency to gout. Anæmic persons, and those in whom the circulation is languid, with a dry skin, are those in whom the disease is prevalent. Sometimes it is localized in patches, as on the back, between the shoulders, the lips, the eyelids, the palms of the hands, the scrotum, or the pudenda. Sometimes it seems hereditary. Intemperance, the use of highly salted food, fish, or indigestible substances, are apt to induce an attack. The depressing passions, anxiety, and grief are also often followed by *psoriasis.* The papillæ of the true skin are enlarged from twelve to fifteen times their normal size; and the whole cuticle, including the Malpighian layer, is greatly hypertrophied—the corium, filled with abundant caudate cells arranged round the blood-vessels of the papillæ (NEWMANN).

Treatment.—Preparations of arsenic are found to be of great service in this form of eczematous inflammation. A form of arsenical remedy, known as the Asiatic pill, is recommended by Cazenave. Its composition is as follows:—

℞. *Arsenici Protoxidi, gr. i.; Pip. Nig.,* gr. xii.; *Pulv. Acac., gr. ii.; Aq. Distill., q. s.—Divide in pil. xii. vel. xvi.* One pill to be taken once, twice, or thrice daily after meals.

But it is of the greatest importance that the meals and the diet should be regulated. Small doses of *pilulæ hydrargyri,* or of *pilulæ calomelanos composita,* or *hydrargyra c. cretâ,* may be given at bedtime for a few days, and followed each morning by a *drachm of magnesia,* given in combination with a tea-spoonful of *lemon juice.* The vascular excitement of the stomach is best subdued by dilute hydrocyanic acid, in the following formula (A. T. THOMSON):—

℞. *Potassæ Carbonatis*, ʒss.; *Succi Limonis Recentis*, *f* ʒiv.; *Acid. Hydrocyanici Diluti*, ♏ iv.; *Vini Sem. Colchici*, ♏ xv.; *Aquæ Distillatæ*, *f* ʒvi.; *misce. Fiat haustus 4ta.; quâque horæ sumendus.*

The tone of the stomach is then to be restored by small doses of *bicarbonate of potassa*, with from twelve to fifteen minims of the *tincture of henbane*, in a fluid ounce and a half of the *infusion of cinchona*. The eruption and itching disappear as the mucous membrane of the stomach and bowels return to a healthy state. The diet should be absolutely free from stimulants; and tepid baths, at a temperature of 96° Fahr., used every morning for half an hour, are most serviceable. The bowels should be regularly relieved by such mild measures as the following formula for pills:—

℞. *Pilulæ Hydrargyri*, gr. vi.; *Pulveris Ipecacuanhæ*, gr. vi.; *Extracti Colocynthidis Comp.*, gr. xii.; *Ext. Hyoscyami*, gr. xviii.; *misce. Fiat pilulæ duodecem. Sum.* ij. h. s. *quotidie* (A. T. THOMSON).

Glycerine and emollient lotions are useful local applications. I have known the itchiness greatly relieved by the following lotion of bitter almonds rubbed night and morning over the parts affected:—

℞. *Amygdal. Amar. Numero*, xx. *vel* xxx.; *Aquæ Rosæ*, ʒvii. *Contunde et tere simul, dein cola, et adde Hyd. Corrosiv. Sublim.*, gr. xii.; *Ammoniæ Hydrochloratis*, gr. xii.; *Spt. Vini Rect.*, ʒi.; or *Dilute Hydrocyanic acid* may be used to the extent of two drachms to the eight ounces, omitting the *corrosive sublimate;* or let *borax* replace the *Hydrocyanic acid*, and omit the *Spirits of Wine*.

When the lotion has dried off, *oxide of zinc ointment* or *glycerine starch* ought to be applied to the parts all night.

MILIARIA.

LATIN EQ., *Miliaria;* FRENCH EQ., *Miliaria;* GERMAN EQ., *Miliariá;* ITALIAN EQ., *Miliare.*

Definition.—*An eruption of innumerable minute pimples, with white summits, occurring in successive crops upon the skin of the trunk and extremities, preceded and accompanied with fever, anxietas, oppression of respiration, copious sweats of a rank, sour, fetid odour, peculiar to the disease. The base of the pimples and the skin around are red and irritable.*

Pathology.—As to the specific nature of this disease pathologists are not agreed. All physicians are not disposed to admit that in *miliaria* a peculiar specific disease exists, with a characteristic eruption and definite course, such as the variolous pustules and course of small-pox exhibit; by many it is believed that "this affection is almost invariably symptomatic." Certain it is, however, that a peculiar epidemic disease prevailed in different parts of Europe at different periods in the world's history, the nature of which is described in the definition; and although in this country it seems to have disappeared, yet there can be no doubt that a specific disease of this description prevails epidemically in many parts of continental Europe and Asia. The disease of these epidemics has been described under the various names of "sweating sickness," "miliary fever," "sudatoria," "miliaria," and the like. Rayer has given the most accurate account of the disease; and I had an opportunity of witnessing a great number of cases of it amongst the Turks, in their military hospitals at Scutari, during the war against Russia in 1854-1856. The temperature and physical climate of that place, combined with the relaxed habits of the Turks, appear to be favourable to the development of such a disease. The best accounts of it are those of Borsieri and Rayer.

The true *sudamina* or *miliaria crystallina* are always symptomatic of some continued fever, *acute rheumatism*, or other general disease. The fluid occurs in a small cavity within the layers of dry cuticle; and as a sudoriferous gland always opens into the vesicle, the fluid is justly regarded as retained sweat (VON BARENSPRUNG). A true miliary vesicle is thus analogous as to its anatomy with the formation of a *comedo* from the obstruction of a sebaceous follicle.

Symptoms.—The fever which precedes the eruption is ushered in by chills, intense and general, shivering, anxietas, oppression of the chest, restlessness, a sense of great feebleness and imminent fainting, with pains in the head, loins, and limbs. In a few hours, nausea, flushing, and profuse sweating supervene, but without any diminution of the dyspnœa, the anxietas, or pectoral oppression, but rather with an aggravation, in the form of short, irregular, panting, and sighing breathing, as if proceeding from a sense of weight under the sternum, with a feeling of internal heat, wandering pains, and sometimes cramps of the hands and calves of the legs. The pulse is generally rapid, small, and feeble; in a few cases hard; often variable, irregular, or intermittent at every ninth, twelfth, or sixteenth beat. The tongue is coated with a white, foul, or yellow fur, indicative of a sluggish condition of the alimentary canal; and the bowels are constipated throughout the disease. The sweat which accompanies this febrile state is profuse, and emits a peculiar smell of a rank, sour, fetid odour. From the fifth or sixth day, up to the twenty-first, an itching sensation is felt in the mammary and epigastric regions, and inner surface of the arms, and the skin of those parts is found to be diffusely red, rough, and irregular, with numerous elevations not larger than pin-heads. In a short time the summits of these become pearly-white, the cuticle being elevated by a slight opaque sero-albuminous fluid,—crop after crop breaks out, and continues from three to seven days, followed by a corresponding desquamation of the cuticle. This eruption is generally confined to the neck, breast, mammary and epigastric regions, and the inner surface of the loins and legs. In severe cases, miliary vesicles appear at the junctions of the skin and mucous membranes, and there they are apt to become aphthous.

A deranged state of the gastro-enteric mucous membrane, indicated by nausea and vomiting of bilious matter, acid eructations, flatulence, and diarrhœa, frequently complicate the disease. Two forms have been described,—namely, a mild and malignant. The malignant is rendered so chiefly by the occurrence of violent inflammation in some of the internal organs, especially of the stomach, lungs, kidneys, or brain; and the danger of the disease is chiefly due to these complications. Such malignant forms have been known to prove fatal in two or three days, but more frequently in from seven to twenty-one.

The **Treatment** of the disease appears to consist in cooling drinks, purgatives, and antiphlogistics, as prescribed by the Italian medical officers who commonly attend on the sick in Turkey.

HERPES.

LATIN EQ., *Herpes;* FRENCH EQ., *Herpes;* GERMAN EQ., *Herpes;* ITALIAN EQ., *Erpete.*

Definition.—*This lesion is expressed by red patches on the skin, of irregular form and variable size, upon each of which there arises a group, cluster, or crop of extremely minute vesicles.*

Pathology.—This is one of the most interesting of cutaneous lesions; for while at present its associations, on the one hand, are with *neuroses,* and on the other with specific *exanthemata,* it seems really as much of a disease, *sui generis,* as any with which we are acquainted (HUTCHINSON). A special mystery envelops its origin. In one case it seems an instance of an inflammation of the skin produced directly by nervous influence. In another case it is not a disease of the skin, but merely a local lesion, to be regarded as a sign or symptom of disturbance, beginning at some part of a nerve-trunk or possibly in the very nerve-centres themselves.

The varieties of *herpes* mentioned by the College of Physicians are—(a.) *Herpes phlyctenodes;* (b.) *Herpes circinatus;* (c.) *Herpes iris;* (d.) *Herpes zoster,*—syn., *shingles, zona ignea, cingulum.*

The phenomena of *herpes zoster,* or *shingles,* as described by Mr. Hutchinson, are as follow:—A certain belt of skin on one side is felt to be tender and painful, but when looked at nothing is to be seen, the skin is not even red. Next day, however, or sooner or later, red points may be seen arranged in long oval groups on the painful parts, and very quickly each point shows a small clear vesicle. The vesicles at first are beautifully pellucid, and very often a number are heaped together, not positively confluent, for divisions between them may still be seen, but much in the manner that a number of hills constituting one range are piled together. At a later stage the vesicles may contain a blood-stained serum, and later still opaque pus. The groups of *herpes* are on one side of the body only. If the area of the terminal branches of the fifth nerve are affected, then the eruption appears on the one side of the forehead, or side of the temple, eyelid, side of the nose, or upper lip; if one of the intercostals, then it appears on one side of the chest or abdomen; and so of the two extremities.

Commencing from behind at the spinous processes, the groups of vesicles in *herpes zoster* arrange themselves in a covered line, passing downwards and forwards on the trunk, approaching the middle line in front. The affected parts continue very painful during the eruption, and often even for a considerable time afterwards. After the eruption has lasted for a few days it begins to fade; and in a week or ten days it will have wholly disappeared, leaving however perhaps some troublesome ulcers.

The eruption disappears at a stated time, as certainly as the eruption of measles does, and leaves the patient free of liability to another attack —as a rule—to which, however, there are rare exceptions, as in smallpox and measles. The eruption never relapses, as skin eruptions usually do. After the inflammation subsides the skin remains tender, and then numb; and the numerous little scars left show that the deep tissues of the true skin were involved (HUTCHINSON).

Morbid Anatomy.—The normal papillæ of the skin are increased in size by serous infiltration, and by multiplication of minute round exudation-corpuscles. The connective tissue-cells appear swollen, and the fusiform cells of the *rete Malpighii* increase in number, and spread through the capillary layer of cutis and the mucous layer of epidermis. These cells increase in number, forming a network throughout the papules, in the meshes of which, exudation-corpuscles appear and rapidly multiply, so as to push aside the normal epithelial cells. The papillary vessels beneath are meanwhile becoming dilated, and the contents of the network gradually assume the character of pus. The neurillemma of the affected nerves are shown to be inflamed and filled with small round nucleated cells (DANIELSSEN, VON BARENSPRUNG, NEUMANN, BIESIADECKI). A partial division of the vesicle and pustule of *herpes* into *loculi*, formed by fusiform nucleated cells has also been described and figured by Dr. Haight. The serum is enclosed in this network, between the horny layer of cuticle and the rete-mucosum. In the papillary layer of the corium are round granular cells, like white blood-corpuscles in size; and as the vesicles change to pustules, these increase in number and fill the network above (BIESIADECKI and NEUMANN, in *Brit. and For. Med.-Chir. Review*, July, 1870, p. 32).

Besides *Herpes zoster*, the other forms of *herpes* are *symptomatic*—as when it occurs on the *lips*, *nose*, *cheeks*, *prepuce*, and is then often rudely symmetrical. It goes through stages exactly like *herpes zoster*; but it may occur over and over again in the same person, and rarely or never leaves scars beyond a grazed-like mark for some time, unless ulceration from irritant applications has been deep. It may be observed in any illness in which a rigor occurs—*e. g.*, after *catheterism;* in *erysipelas;* in *ague;* in *pneumonia;* in *fever;* any inflammation of a shut sac, such as of the *pleura*, *peritoneum*, *tunica vaginalis*. It is a common lesion on the lips, associated with a common catarrh. A very common and troublesome form of the disease affects the foreskin—*herpes preputialis*. Its effects are often mistaken for chancres, and by unprincipled persons may be treated as such. It has nothing to do with syphilis, and its history is a sufficient guide to diagnosis (see vol. i., p. 880). The disease may be prolonged by ulceration of the vesicles, or by irritant applications, or when the scabs are prematurely chafed off.

Symptoms.—The constitutional disturbance is rarely more than the pain will account for; but the eruption is often preceded by considerable local pain, or by cutaneous pain over the region which is about to be the seat of eruption, or by pain resembling neuralgia in the neighbouring parts, and which may continue after the eruption has disappeared. It runs a very definite course, and when not interfered with, its development and defervescence is completed in about ten days.

Now and then the patient has a slight rigor before the attack. It occurs at almost any age, and in either sex; and no special condition of general ill health can be stated as predisposing to an attack of shingles. It is not contagious, and does not occur twice in the same person. The eruption is rarely, if ever, symmetrical; but syphilitic rashes have sometimes been known to resemble *herpes*, and so give rise to the belief that it is symmetrical (*Syphilitic Shingles* of Mr. Hutchinson). It occurs with equal frequency on the two sides of the body. The parts affected usually correspond to the cutaneous distribution of a particular nerve; but Mr. Hutchinson has in one case seen the *iris* implicated in a form of iritis.

Referring to *herpes* as symptomatic of irritation of the nerves, Dr. W. Moore, of Dublin, regards intra-thoracic tumors producing such nerve irritation as to be a probable cause of *herpes;* and hence that the occurrence of *herpes zoster* might suggest the existence of such tumors. He relates, in illustration, two cases of *herpes* eruption which occurred in patients suffering from thoracic aneurism (*Dublin Quarterly Journal of Med. Science*, Feb., 1868.)

Treatment.—Dry applications, such as enveloping the parts in cotton wool after sprinkling with *oxide of zinc* in fine powder, or with starch, I have always found better than wet applications.

Tincture of aconite and *tincture of opium* have been applied to relieve the severe intercostal pain. The intolerable burning has been relieved by painting over the vesicated patches with *collodion.* It tends to prevent the rupture of them, and so tends to prevent ulceration by promoting healing of the parts under a scab when inflammation has subsided.

An opiate application, such as Dr. Fuller recommends in *sciatica*, may be of use in some cases of shingles, namely:—

℞. *Tinct. opii., sp. Æther, Sulph. co.; Glycerin.*, ā ā ʒiii.; *Ext. Belladonna, gr.* xx.; *misce.* A strip of flannel soaked in this should be applied along the course of the deep-seated pain, and covered with oil-silk, to prevent evaporation (Waring).

The state of the stomach and digestive organs, and diet generally, should be regulated. Malt liquors should be avoided; and, if pain is severe, opiate fomentations may be applied.

Herpes preputialis requires no treatment beyond careful ablution with tepid water, and the interposition of a piece of wet lint between the prepuce and the *glans penis.* If there is much pain, the lint should be moistened with a watery solution of opium.

PEMPHIGUS—*Syn.*, POMPHOLOX.

LATIN EQ., *Pompholox;* FRENCH EQ., *Pemphigus;* GERMAN EQ., *Pemphigus*—Syn., *Pompholox;* ITALIAN EQ., *Penfigo*—Syn., *Ponfolice.*

Definition.—*An eruption of large vesicles, filled with serous fluid, known as bullæ.*

Pathology.—These *bullæ* vary in magnitude, are generally distinct, but numerous. They spring up in successive crops, generally in the forearms and legs, surrounded by redness of the skin. The vesicles, originally transparent, gradually become opaque, pearl-coloured, and ultimately of a pale red colour. It has now been established, by the researches of Dr. Haight, that the *bullæ* of pemphigus differ from the vesicles of variola and herpes. The fluid of the *bullæ* is not situated between the mucous and the horney stratum of the epidermis, but between the layers of the latter, for the floor as well as the roof of the bulla-cavity is formed of flat cells, which show no nuclei, and do not absorb carmine. The cavity is simply filled with serum, and shows no locular net-work of spindle-shaped cells (Neumann, *Med.-Chir. Review*, July, 1870).

Usually the disease is prolonged over weeks, months, or years, and is not attended by any decided febrile disturbances. A slight pricking sensation indicates a red spot, where the eruption commences. In the centre of each of these spots the cuticle begins to rise like a blister; and as the circumference of the redness increases, the blister-like vesiculæ are so rapidly formed that in a few hours each vesicle may be as big as a hazel-nut or walnut. The *blebs* are then apt to burst, when a straw-coloured serum exudes; and the epidermis contracts into folds or wrinkles, the surface underneath being red, painful, and smarting. If the vesicles do not burst, small brownish flat crusts form. The disease is usually associated with debility, intemperance, and bad or insufficient food. In one case of chronic pemphigus, quoted by Neumann, Dr. Hertz found extensive lardaceous disease of the viscera. The following varieties are to be distinguished:—(a.) *Pemphigus acutus;* (b.) *Pemphigus chronicus;* (c.) *Pemphigus solitarius;* and (d.) *Pemphigus foliaceous.*

Pemphigus foliaceous commences on the front of the chest, and when fully developed covers the whole body. It is almost always fatal.

Treatment.—Dietetic and tonic, especially by *quinine* and the mineral acids. *Arsenic* is said to be of use in obstinate cases, improving the general health. Emolient lotions, with or without opium, are useful local applications.

ECZEMA.

LATIN EQ., *Eczema;* FRENCH EQ., *Eczema;* GERMAN EQ., *Eczem*—Syn., *Nässende Flechte;* ITALIAN EQ., *Eczema.*

Definition.—*An eruption characterized by*—(1.) *Infiltration of the skin;* (2.) *Exudation on its surface;* (3.) *The formation of crusts;* (4.) *Itching.*

Pathology.—Eczematous skin feels thick when pinched up into a fold. It has a doughy feel, and on pressure the redness disappears, and shows a yellowish hue of the skin. The exudation is watery, sometimes purulent, and sometimes mixed with blood. It stains and stiffens underclothing with which it may come in contact. The exudation becoming concrete, forms crusts, and their appearances vary with the character and nature of the exudation. The itching is always aggravated by touching the part, and still, at the same time, an irresistible desire to scratch is excited. Scratching is sometimes thus persevered in till blood flows from the part. The disease is thereby greatly aggravated—a copious eruption of newly formed vesicles and fissures being apt to form.

The elementary lesions of the skin in eczema are—(1.) An erythematous state of the skin; (2.) Vesicles; (3.) Pustules; (4.) Papules; (5.) Fissures; (6.) A mixture of several or of all of these lesions; and in the fully expressed disease there may not be the merest vestige of either a vesicle, a pustule, or a papule, but the skin is red and smooth on the surface, having a brilliantly polished and shining appearance, the tissue being loaded with infiltration. The vesicles, when they occur, are usually developed at the orifices of the cutaneous follicles. They are small and closely set together; they usually rupture early, and the serosity concretes into crusts. The more the skin is infiltrated the less likely are vesicles to be seen. In the absence of vesicles, when the disease is at its height, the infiltrated patches are red and inflamed. The redness is not uniform: it is studded with innumerable points of a deeper red, giving a punctated appearance to the part (DEVERGIE). These points correspond to the orifices of the glands, like the vesicles which may have preceded them; and their distinct appearance is due to the greater congestion of the skin which surrounds the glandular orifices. Minute excoriations are apt to occur at these spots, the result of rupture of the vesicles. From these excoriated points serous fluid continues to exude, which afterwards concretes into scabs (*eczema vesiculorum*).

The *impetigo* of authors is the pustular form of *eczema,* and the pustules form upon erythematous patches of skin, just as the vesicles did before them. They are most common on the head and chin. Vesicles and pustules are often seen on the same patch (*eczema impetiginodes* or *eczema pustulosum*).

Any part of the skin may be the seat of *eczema;* but there are certain localities where it is most apt to occur. These are the head, the hairy portions of the face, the lips, the edges of the eyelids, the nostrils, the lips, the external auditory passages and ears, the hands, feet, legs, genitals, perineum, *mammæ,* umbilicus, and parts of the skin which are naturally in contact with one another.

On the head and hairy parts of the face the disease occurs most frequently in the pustular form (*impetigo capitis*); and collections of pus are apt to form beneath the crusts and scabs, attended sometimes by little cutaneous abscesses and enlargements of the lymphatic glands. Each pustule of eczema is situated at the orifice of a hair follicle; and the hair may be seen to pass through the centre of each pustule. These pustules dry up into small yellow crusts when the disease affects the hairy parts of the face; and the skin on which the pustules are developed assumes a dusky red tint, becoming gradually more thickened and infiltrated (*impetigo menti—eczema pilare faciei*). In the *eczema* of the internal auditory passages the disease usually affects the auricles, commencing on the skin of these parts, and gradually extending inwards. The calibre of the meatus is narrowed, and often so much so that it is impossible to see the *membrani tympani.* A milky or watery fluid exudes from the meatus, and moistens the pillow at night. The discharge is apt to have a very bad odour if the ear is not frequently washed out (Anderson, *Med. Times and Gazette,* June, 1863).

Morbid Anatomy.—The condition of the skin in different stages of *eczema,* as shown by five sections made perpendicular to the surface of the skin, has been investigated by Biesiadecki, and is quoted by Neumann. In the early stages it resembles the description already given under *herpes.* The papillæ are enlarged and filled with serum and exudation cells, while the normal connective tissue corpuscles of the cutis are swollen, and the same fusiform cells are seen in the *rete Malpighii.* When the formation of cells in the papillæ becomes very rapid, and fluid is also rapidly effused into the net-work of fusiform corpuscles and epidermic cells which form the eczematous papule, a vesicle is the result. The fusiform cells take on the same office as ordinary connective tissue corpuscles in conveying the liquid exudation from the inflamed papilla beneath to the epidermis, where it collects under the horney layer of cuticle. When the cuticle falls off, the same channels convey the continually secreted eczematous fluid from the papillary layer of the cutis to the *rete Malpighii,* now laid bare, and so keep up the characteristic moisture of eczematous lesions. As the disease becomes chronic, the increase in size of the papillæ becomes more marked than ever, and their solid fibrous structure is hypertrophied, until at last they become visible to the naked eye. The whole cutis is thickened; and in long standing cases bands of dense fibrous tissue stretch down among the fat cells of the subcutaneous fascia. The cutaneous glands share in the changes of the surrounding structures. Pagenstecher has shown that the fusiform cells of the epidermis, discovered by Biesiadecki, are always increased in number whenever the formation of cuticle becomes excessive —*i. e.,* in most chronic as well as in acute affections of the skin. He believes they pass by autogenic movement from the papillary layer to the *rete Malpighii,* and they may often be seen half in the *corium* and half in the *Malpighian stratum.* They are known as "*migratory*" or "*wandering cells*" (wanderzellen), suggested as the equivalent of the so-called "*basement membrane*" of English anatomists. With regard to the exudation cells first found so remarkably arranged round the vessels of the papilla, it is suggested by the able reviewer of Neumann's work, in the *British and Foreign Review,* that they may probably be corpuscles which have just made their transit as the offspring of connective tissue cells, or the result of spontaneous molecular aggregation. Like the serous fluid in which they float, they have passed into the *rete Malpighii* from the subjacent vessels of the cutis instead of being generated where we find them by the epithelium cells themselves, or the fusiform corpuscles of Biesiadecki. This explanation is derived from the remarkable observations of

Stricker and Cohnheim, already referred to in vol. i. under *inflammation,* page 30, (abundantly confirmed in Germany and England), on the passage of white corpuscles through the walls of their blood-vessels in a state of irritation.

The forms of *eczema* recognized by the College of Physicians are:—(a.) *Eczema Simplex;* (b.) *E. Rubrun;* (c.) *E. Impetiginodes;* (d.) *E. Chronicum.*

Practically, a papular, a vesicular, a pustular, a weeping, and a squamous conidtion of eczematous lesions may be recognized, sa Hebra, Willan, and Neumann describe. Every case of eczema will, at some period or other of its progress, develope *vesicles;* but the infiltration of the skin, the character of the secretion, the thickening of the texture of the skin, the formation of crusts, the itching, and the seats of election where the eruption is found, are sufficiently diagnostic and characteristic evidences of this very common disease.

The *pruritus,* or itchiness of simple cutaneous irritation, without any papular or other eruption, ought to be separated from true *prurigo,* and from the *pruritus* of eczema.

Treatment of eczema must be constitutional in the first instance; and local applications suited to the nature of the part affected are to be carefully used. Derangements of the digestive organs must be especially rectified. If the tongue is loaded, the appetite bad, the liver torpid, as indicated by light clay-coloured evacuations and costive bowels, small doses of *grey powder* in combination with *quinine* are indicated. Dr. T. M. Anderson suggests the following:—

℞. *Sulphatis Quiniæ,* gr. xii.; *Pulv. Rhei.,* gr. xxxvi.; *Hydrarg. c. Cretâ,* gr. xxx.; *Sacchari Albi,* ʒi.; *misce, et divide in Pulv.* xii.

Two of these powders are to be taken daily by an adult; but at any age the dose must be so adjusted that the patient has at least one full natural evacuation daily. If the patient is robust, but with the functions of the liver and bowels impaired, occasional doses of *calomel,* alone or in combination with *scammony,* will stimulate the torpidity of the digestive organs; and at the same time the cutaneous inflammation will diminish. If the patient is a full feeder, and will not be persuaded to live more sparingly, *one drachm to two drachms of the sulphate of magnesia* may be given twice daily, with *a sixth to half a grain of tartar emetic* added to each dose. The effect of this remedy will be to diminish desire for food, and at the same time keep the bowels freely open. If the patient is scrofulous, or debilitated from insufficient food, or food not nutritive enough, more nourishing food must be given, combined with tonics containing iron and cod-liver oil. Children a few months old, reduced to "skin and bone," recover wonderfully under the influence of twenty drops of the syrup of the iodide of iron in a tea-spoonful of cod-liver oil repeated three times daily, the dose of the oil being gradually increased to a table-spoonful (ANDERSON, l. c., June 27, 1863). *Syrup of the phosphate of iron,* or of the *phosphates of iron, quinia,* and *strychnia,* should be alternated with the *iodide of iron.*

The treatment must be steadily maintained for at least six weeks or two months. In severe cases the oil may be rubbed into the skin, in addition to giving it internally. *Cod-liver* and *iron* preparations are almost equally serviceable to eczematous adults. When there is a difficulty of taking the oil in its pure state, the emulsion of it will often be found useful;—when the appetite is very deficient, a pure tonic, such as *quinine* and *aromatic sulphuric acid* may be of service, as in the following:

℞. *Sulphatis Quiniæ*, gr. xvi.; *Acid Sulphurici Aromatici*, ʒiv.; *Syrupi Limonum*, ℥ss.; *Infus. Cascarillæ*, ad ℥viii.; *misce, et cola per chartam.* A table-spoonful to be taken three times a day half an hour before food.

If the eczematous patients are robust and plethoric, the local abstraction of blood by leeches or scarification is often beneficial; but in general the action of *calomel purgation* is sufficient to reduce the inflammatory action, combined with a regulated abstemious diet of mixed animal and vegetable food simply dressed. Dishes of pastry, pickles, spices, strong tea, and coffee, are particularly to be avoided; while the use of wine, spirits, and malted liquors must be entirely suspended.

If the patient has been much in the habit of using stimulants, the eczema will be much more difficult to subdue than if he had been an abstainer from these fluids. It is sometimes necessary to prescribe milk diet for a time, all animal food being proscribed.

Of internal medicines, *arsenic*, *sulphur*, and the *alkalies* are especially useful (STARTIN, ANDERSON). Fowler's solution (the *liquor arsenicalis* of the Edinburgh Pharmacopœia) is well suited. An adult may commence with *five* minims thrice daily; and at the end of a week the dose should be increased by *one drop* every second or third day, till the disease begins to yield, or the medicine to disagree (ANDERSON). "In order to secure the virtues of arsenic as an alterative, it will be necessary to push the medicine to the full development of the phenomena which first indicate its peculiar action on the system. Arsenic as a remedy is too often suspended, or altogether abandoned, at the very moment its curative powers are coming into play. The earliest manifestation of its physiological action is apt to be looked upon as its poisonous operation; and the patient declares that the medicine has disagreed with him. Forthwith the physician shares his fears; the prescription is changed, and another case is added to the many in which arsenic is said to have failed after a fair trial of its efficacy" (Begbie, *Contributions to Practical Medicine*, p. 270). *Arsenical solutions* should be given immediately after food; and in persons whose digestive organs are weak, a tonic infusion, such as the infusion of *cascarilla* or *gentian*, is the best vehicle for its administration; and a few drops of *morphia* may sometimes be added, as in the following:—

℞. *Solut. Fowleri; Solut. Muriatis Morphiæ*, ā ā ʒii.; *Syrupi Limonum*, ʒss.; *Tinct. Cocci Cacti*, ʒss.; *Infus. Cascarillæ*, ad ℥xii.; *misce.* A table-spoonful of this mixture to be taken thrice daily immediately after food.

The *liquor sodæ arsenias*, in doses of five to ten minims, is said to cure eczema with less gastric disturbance and with less irritability of the conjunctiva than the *liquor arsenicalis.* As the disease yields, the dose may be diminished; but the use of the remedy should not be suspended till some time after the complete removal of the eruption. In the case of infants at the breast, arsenical solutions may be given to the mother; and *mercury may be combined with the arsenic, as in Donovan's solution, of which ten minims may be given thrice daily.* Mr. Hunt lays down certain conditions for the administration of arsenic; and amongst them he forbids its commencement while signs of active cutaneous inflammation are present.

The use of the natural mineral waters which contain sulphur is the best method of securing the full effects of the alterative action of sulphur in chronic cases of eczema where there is an absence of inflammatory action. These waters are especially Harrowgate and Moffat in this country; Aix, Barèges, St. Sauveur, Cauterets, on the Continent.

Alkalies are most beneficial in those cases in which the patient has been in the habit of taking stimulants; and when there is a tendency to acidity of the stomach, and to the deposit of *lithates* in the urine, or to gout, or to rheumatism, *aqua potassæ may be given, largely diluted with water, in doses of twenty minims three times a day.* Dr. Anderson recommends *sesquicarbonate of ammonia* (now called the *carbonate of ammonia* in the *British Pharmacopœia*), in doses gradually increasing from ten to thirty grains. If gout prevails, *colchicum* ought to be given with the other remedies; and in rheumatism the *acetate* or *bicarbonate of potash* in half-drachm doses should be added to each dose. All these alkaline remedies should be largely diluted with water.

For the relief of pain and procuring sleep in acute cases, opiates must be administered; and when they fail tincture of *cannabis sativa*, in doses of ♏xxv., has been found sometimes to secure sleep, or at all events comparative ease (Christison).

With regard to local treatment, the first point is to remove all sources of irritation, and especially the crusts. A poultice composed of *crumbs of bread* and *hot almond oil* applied to the eruption at night will usually bring away the crusts in the morning. If they fail to be detached by this application, they must be again lubricated with fresh almond oil, and forcibly removed when they are thoroughly softened. If, after the crusts are removed, the surface is seen to be acutely inflamed, and if there is a *burning heat* in place of *itching*, local sedatives must be applied. For this purpose Dr. Anderson recommends a cold potato-starch poultice, a small quantity of powder containing camphor being sprinkled over the inflamed surface before the poultice is applied. The camphor powder may be composed of the following ingredients:—

℞. *Camphoræ*, ʒss. ; *Alcoholis*, q. s. ; *Pulv. Oxydi Zinci; Pulv. Amyli*, ā ā ʒiii. This powder must be kept in a stoppered bottle (Anderson).

Instead of poultices, a mixture of powdered *oxide of zinc* and *glycerine*, in the proportion of half an ounce of the one to two ounces of the other, will be found to be a soothing application; and to it a little *camphor* may also be added, as in the following:—

℞ *Camphoræ*, ℈ii.; *Pulv. Oxydi Zinci*, ℥ss.; *Glycerinæ*, ℥ii.; *Cochinillini*, gr. ii. ; *Spt. Rosmar.*, ʒi. ; *misce.* This mixture must be stirred before using it, a thin layer being then rubbed over the inflamed part twice or thrice daily (Anderson).

When the disease passes into a chronic stage, as indicated by the disappearance of the burning heat and the supervention of itching, the local applications must vary with the condition of the parts. If there be much *infiltration* of the tissues, treatment by *potash* applications is the most efficient (Hebra, Anderson). The more extensive the disease the *weaker* the solution ought to be for application over the large surface. If the infiltration is slight, common *potash soap* (soft soap, black soap, *sapo mollis, sapo viridis*), or a solution of *one part of it in two of boiling water, a little oil of rosemary or citronella being used to conceal the odour*, may be used. A piece of flannel dipped in this liniment should be rubbed as firmly as possible over the affected parts night and morning, the solution being allowed to dry upon them. It should, however, be washed off after each application. If the patient can bear it, a piece of flannel, saturated with the solution, may be left in contact with the part all the night. Another method of treatment is to paint over the eruption night and morning with a large brush, charged with the *aqua potassæ* of the *Edinburgh Pharmacopœia*, its irritant action being neutralized by means of cold water when the smarting becomes excessive. A solution of *potassa fusa* may also be employed of various strengths. If the case be mild, *two grains* to an *ounce* of water; if more severe, then *five, ten, twenty, thirty*, or *even more grains to the ounce of water* may be used. *But all these stronger solutions must be washed off very speedily, and they ought not* to be employed more than once daily. Such remedies the physician ought to apply himself, and not entrust the control of their action to the patient. As infiltration subsides, the solution ought to be gradually diluted; and great caution is necessary in using such local applications upon infants, delicate females, and old or infirm persons. The affected parts should be bathed repeatedly during the day in cold water, during the use of *potash* applications. The *cold douche* may be employed where it is practicable. If the itching is very intolerable, *prussic acid* may be mingled with the potash applications, as in the following:—

℞. *Potassæ Fusæ*, gr. x.; *Acid Hydrocyan. Dil.* (Ed. Ph.) ʒss.; *Aq. Rosarum*, ℥ii.; *misce.* A little of this solution is to be rubbed firmly over the eruption night and morning, and at any time when the itching sensation is severe.

If there is a tendency to the formation of fissures, which are apt to result from the use of potash, *cod-liver oil* or *glycerine* should be applied to the parts every night. *Glycerine of aloes* is also recommended as a healing agent by M. Chausit and Dr. Waring. It is prepared by evaporating four to eight parts of the *tincture of aloes*, and incorporating the residuum with thirty parts of *glycerine*.

Tarry applications are of great value in the local treatment of the *declining stages* of eczema, when infiltration and itching are subdued. Common tar (*pix liquida*) or oil of cade (*oleum cadini*, as manufactured at Aix-la-Chapelle) should be rubbed firmly over the eruption by means of a flannel cloth, and allowed to dry upon it. It should be applied thrice daily, and washed off with *soft soap* or *petroleum soap*. Dr. Anderson recommends that common tar should be combined with one of the potash solutions—for example, a mixture of *equal parts of common tar, methylated spirit, and soft soap*, used as above directed, will be found useful, or the following preparation, as less offensive :—

℞. *Saponis Mollis, Spt. Rectificati, Olei Cadini*, ā ā ℥i. ; *Olei Lavandulæ*, ʒiss. ; *misce*. A little quantity is to be firmly rubbed over the eruption night and morning; and it must be washed off before each re-application.

Of *mercurial applications* the *citrine ointment* (*Ung.-Hyd. nit.*) is the best, or the *ointment of the iodide of mercury*. They may be used of their full strength, or diluted with lard, according to the indications of the case, care being taken that *mercurialism* is not induced. When eczema has reached the dry stage, Mr. Milton regards the *dilute ointment of the nitrate of mercury* as the most effectual remedy we possess.

In the itching of *pruritus*—*pruritus scroti* and *prurigo pudendi muliebris*—especially the *prurigo senilis*, this dilute ointment is highly spoken of by Dr. Bowling, United States, and by Mr. Balmanno Squire in this country, as one of the best remedies for subduing the irritation and itchiness. Dr. Bowling advises that the parts be first sponged with vinegar or lemon juice previous to the application of the ointment; and states that for fifteen years he has not failed to effect a permanent cure (*Med. Times*, June 6, 1868).

In using ointments, a small quantity should be melted on the finger, and rubbed firmly into the affected part. None should be allowed to lie undissolved upon the skin; and the part should always be cleansed with soap and water before re-application. Epilation should be resorted to in cases of eczema of the hairy parts of the face. In very mild attacks, or with a view to prevent the recurrence of the disease, the skin may be washed occasionally with soft soap and water. Hendrie's "dispensary petroleum soap" is recommended by Dr. Anderson, to whose admirable lectures on "Eczema," in the *Medical Times and Gazette* for May, June, and July, 1863 (since published in a separate volume), the reader is referred for more detailed information.

ECTHYMA.

LATIN EQ., *Ecthyma;* FRENCH EQ., *Ecthyma;* GERMAN EQ., *Ecthyma;* ITALIAN EQ., *Ectima.*

Definition.—*An eruption of large round pustules, generally distinct, and seated upon a hard inflamed base. The pustules are succeeded by dark-coloured scabs, which leave superficial cicatrices behind them, or red stains, which disappear after a time.*

Pathology.—The eruption of ecthyma is common on the neck, shoulders, buttocks, extremities, and chest; and is seldom developed on the face or scalp. The typical appearance of ecthyma may be seen in the pustules produced by friction with *tartar emetic* ointment. The pustules continue for several days, and are succeeded by scabs, which begin to form in the centre. The disease may occur at any age or season, but it most frequently appears during spring and summer in young adults. The *ecthyma cachecticum* of Willan prevails in old, irritable persons much addicted to alcoholic fluids. It is classed among furuncular affections by Mr. Erasmus Wilson; and the great majority of cases appear to be either of traumatic or syphilitic origin, or due to scabies.

Treatment is chiefly by diluents, simple and emollient baths, and regulation of diet. Moderate exercise should be taken, combined with the use of alkaline or salt water baths. Mild laxatives are beneficial, and spirits, wine, and beer are to be refrained from. The food should be as nourishing as can be digested. Tonics, such as *quinine* and *iron*, are also indicated. A stimulating lotion, composed of *muriatic acid* diluted with water, is of use to brush over the parts and promote cicatrization. Specific treatment must be adopted when the disease is associated with syphilis.

ACNE.

Latin Eq., *Acne;* French Eq., *Acne;* German Eq., *Acne*—Syn., *Finnenausschlag;* Italian Eq., *Acne.*

Definition.—*Inflammation of the sebaceous glands and hair follicles.*

Pathology.—This lesion affects the sebaceous glands and hair follicles of the skin generally, or those at the roots of the hairs of the beard, and is commonly produced by local causes of irritation. The disease generally appears as pimples; but suppuration usually very slowly follows; and, there may be persistent redness after suppuration is at an end, giving a blotchy appearance to the part. The parts most frequently affected are the temples, cheeks, nose, and forehead; but it also appears on the neck, shoulders, and front of the chest. The first occurrence of the disease is indicated by the collection of morbid material in the subaceous follicles, which open on the skin by a blackish point (*comedo*), to which the vulgar give the name of "*grubs*" or "*worms*" *in the skin.* The tubercles on which the classification of this disease is sometimes based are merely the terminations of the pustules, and not an elementary lesion. The accumulation of the matter secreted by the subaceous follicles tends to keep up an eczematous inflammation in them. The lesion has been mistaken for small-pox.

The lesions of *acne* recognized by the College of Physicians are—(a.) *Acne punctata;* (b.) *Acne indurata;* (c.) *Acne rosacea;* (d.) *Acne strophulosa*—Syn., *Strophulus albidus.*

Acne rosacea should be separated from true acne, with which it seems to have nothing in common, except occasional coincidence; as well as from the other forms, which it does not even resemble in appearance. It consists in hypertrophy of the cutis and subcutaneous tissues, with enlargement of their blood-vessels, as a consequence of chronic congestion of the skin of the nose and other parts. It is thus analogous to the *œdema durum* of continued venous congestion, the hypertrophy seen in a long prolapsed rectum or uterus; or to the effects of blood-stasis in chilblains (*Med.-Chir. Rev.*, July, 1870, p. 33).

If a *demodex follicularum* should be discovered and caught in a follicle of *acne*, make a note of its presence as a probable source of irritation.

Treatment.—Local and general measures require to be combined. Diet, in the first instance, should be restricted. Wine, spirits, and coffee are to be refrained from. Milk is to be used as a drink, and as an article of diet, combined with light food, fresh vegetables, and succulent ripe fruits. Emollient applications, such as an emulsion of bitter almonds, a decoction of bran or quince seeds, and tepid milk, are useful. Dr. Anderson has found one or other of the two following lotions particularly serviceable; and generally, if one fails, the other succeeds:—

℞. *Sulphuris*, ʒi.; *Spt. Rectificati*, ℥i.; *misce.*

The mixture is to be shaken before using it; or,

℞. *Hyd. Corrosiv. Sublim.*, gr. ix.-xii.; *Hydrochl. Ammoniæ*, ʒss.; *Cochinillini*, gr. i.; Aq. Rosæ, ℥vi.; *misce.*

In that form of *acne* common to young women at the commencement of their menstrual period, Dr. Ringer recommends the following solution, applied twice or thrice daily:—

℞. *Sulphur*, ʒi.; *Glycerine*, *f*℥i.; Aq. *f*℥x.; *ft. lotio.*

Whichever of these lotions is used, let it be applied by dipping a piece of flannel into the lotion, and rubbing *very firmly* over the eruption night and morning.

To promote resolution of the induration, *iodide of sulphur*, in the proportion of *fifteen* or *twenty-four grains to an ounce of lard*, is of great service in dispelling the tumors. Drastic purgation ought to be avoided. Simple baths at a temperature of 88° or 90° Fahr. are of service.

Calomel ointment (ʒi. of calomel to ℥ii. of lard) has been found useful, care being taken against salivation. If there be much thickening of skin, Mr. Milton recommends the internal use of *liquor potassæ*, although inferior to *arsenic*, which ought also to be used.

ICHTHYOSIS.

LATIN EQ., *Ichthyosis;* FRENCH EQ., *Ichthyosis;* GERMAN EQ., *Ichthyosis;* ITALIAN EQ., *Ittiosi.*

Definition.—*A lesion which involves the whole tissue of the skin, and is characterized by the growth of thick, dry, imbricated scales of a dirty grey colour, resting upon a perfectly uninflamed surface, and never accompanied by pain, heat, or itching.*

Pathology.—It appears principally on the external parts of the limbs, round the joints, on the knee and elbow, on the upper part of the back, and generally on those regions where the skin is thick and coarse. The disease is usually congenital, and lasts during life. It may not be strongly marked at the period of birth; but the skin appears dull, thick, and fretted, and the lesion is developed as the infant grows older.

Morbid Anatomy.—The microscopic anatomy of *ichthyosis*, as demonstrated by Neumann and Rindfleisch, clearly shows that the true congenital form of this disease does not depend on collections of dried up *sebum*, but on the excessive development of the horny layer of cuticle, combined with considerable thickening of the whole skin. At the same time there is increased secretion from the sebaceous glands, which accounts for the greasy character of the scales, for their frequently dirty colour, and for the masses adhering so strongly to each other. An alcoholic extract of the scrapings of the skin, deposited on evaporation, crystals of *stearin*, and of (apparently) *hippuric acid;* while earthy phosphates, oil-globules, and cholesterine were left behind. A further treatment of the scales with *ether* showed a quantity of solid and liquid fat, as well as more cholesterine. After incineration there remained chlorides of potass and soda, with phosphates of lime and magnesia, and a certain amount of iron (SCHLÖSSBERGER).

On removing and breaking through a crust of *ichthyosis*, vertical streaks, or even actual fibrillæ may be observed on the fresh surface. When it has been macerated in a weak alkaline solution, and carefully torn to pieces with needles, it may sometimes be entirely divided into prismatic rods, which might be equally well described as short and thick fibres. Each of them consists of a number of horny laminæ, arranged concentrically round an axis like the rings of a tree (Rindfleisch, quoted in *Med.-Chir. Rev.*, July, 1870, p. 43).

Treatment.— Remedial measures are only palliative, consisting mainly of mucilaginous and glycerine lotions, with vapour baths to mollify the roughness. Potash solutions or other preparations of potash are contraindicated as local applications.

CHELOID.

LATIN EQ., *Tumor Cheloides;* FRENCH EQ., *Chéloïde;* GERMAN EQ., *Keloid;* ITALIAN EQ., *Cheloide.*

Definition.—*A tuberculated growth or swelling of the true skin occurring spontaneously or upon a cicatrix.*

Pathology.—Originally described by Alibert, subsequently by the late Dr. Addison, of Guy's Hospital, and Mr. Thomas Longmore, Professor of Military Surgery at the Army Medical School.

The keloid of Alibert is a fibrous tumor of the skin, often developed on a cicatrix; whereas true keloid, as described by Dr. Addison and Mr. Longmore, is a much more extensive lesion; but anatomically the lesions do not appear to differ, whether occurring spontaneously in the skin or limited to a cicatrix, although called genuine and spurious varieties—true and false cheloid. It begins as a few prominent red tubercles, generally over the sternum—whence it may extend gradually to the sides of the body and the back. The red tubercles gradually increase in size, and coalesce into larger growths. These growths then send forth spur-like processes on every side; a slight puckering of the healthy skin surrounds the marginal limits of the bases of the tumors, and marks the advance of the disease. The spread of the disease is very slow and gradual, extending over months and even years. Intense itching and irritation accompany its advance, more particularly when warm in bed at night, or during hot weather. Continued cold causes the growths partially to disappear, leaving an old cicatrix-like condition of the skin (*Med.-Chir. Trans.*, 1863). According to the recent investigations of Dr. Neumann, "the epidermis and papillary layer are, at least, primarily unaffected; the deep layer of the cutis is thickened by a multitude of fibres running parallel with the surface, which begin in bundles of spindle-shaped cells, first observed surrounding the external coat (*tunica adventitia*) of the arterioles of the skin. The cutaneous glands are at first unchanged, but afterwards become pressed upon so as gradually to atrophy and disappear. This account is fully confirmed by the independent researches of Dr. J. C. Warren, of Boston, who found the same cellular growth in the adventitia of the arteries, spreading to the deeper layers of the corium, in the form of dense horizontal fibres running parallel to the long axis of the cheloid nodule. The epidermis and papillary layer are unaffected, as are the cutaneous glands, until they gradually disappear in the latter stages of the disease. Dr. Warren concludes that there is no difference anatomically between true and false cheloid—that both consist of a 'fibro-plastic' tumor of the corium, and that the 'malignity' or tendency to recur after removal, which is so marked a feature of the disease, simply depends on the fact that the primary affection of the blood-vessels can be traced some distance beyond the circumference of the tumor, and can therefore reproduce a fresh nodule after the original one has been apparently completely extirpated. The chief anatomical difference between cheloid and an ordinary cicatrix appears to be, that the fibres of connective-tissue of which the latter is composed form a felted network like that of normal cutis, with no definite parallel arrangement. Virchow regards the most typical forms of cheloid, as sarcomata (a group which, it will be remembered, corresponds in great part with Lebert's fibro-plastic tumors), and distinguishes from these the fibrous growths described by Addison as true keloid, and others which he believes to be of cancerous or syphilitic origin. Rindfleisch agrees in regarding cheloid as a sarcoma, but places its seat in the papillary layer of the cutis—a supposition disproved by microscopic investigations, which were wanting when he formed this opinion" (*Med.-Chir. Rev.*, July, 1870).

Treatment is not yet defined; but the history of the disease points to cold applications and tonic remedies as the basis of a rational treatment.

SECTION III.—PARASITIC DISEASES OF THE SKIN.

TINEA TONSURANS—*Syn.*, RINGWORM.

LATIN EQ., *Tinea tondens;* FRENCH EQ., *Tinea tonsurans;* GERMAN EQ., *Tinea tonsurans*—Syn., *Ringwurm;* ITALIAN EQ., *Teigne tonsurante.*

Definition.—*An affection implicating the hairs of the skin, scalp, or chin, and usually assuming a circular form. The hairs become dry and brittle, having a tendency to crack or break across. Itching accompanies the primary eruption, which is generally at first erythematous, with slight swelling, and a parasitic fungus ultimately appears* (Achorion Lebertii—Trichophyton tonsurans), *which had been developing between the epidermis and the true skin. The fungus has a pure white colour and powdery aspect. It covers the epidermis between the hairs, and forms around them a complete white sheath. Inflammation of the hair follicles and of the surrounding tissues occurs; and when pus forms, the fungus is destroyed at the expense of obliteration of the roots of the hair, when perfect baldness ensues* (BAZIN, ANDERSON).

Pathology.—The nature of this disease is to be studied in the botany of the crpytogamic parasite called the *Achorion Lebertii* (*Trichophyton*), discovered by Malmsten in 1845. It consists of oval transparent spores or globules, about the $\frac{1}{7000}$ part of an inch in diameter. Many of these are isolated; others constitute, by their juxtaposition, articulated filaments. Comparatively few cryptogamic tubes are visible—a character which distinguishes the ringworm affections from the vegetable structures seen in the other parasitic diseases of the skin (fig. 75). Its anatomical seat is in the *interior of the roots of the hair.* The *hairs* and *fungi* simultaneously increase; the former seem larger than usual, are paler in colour, lose their elasticity, soften, and break off when they have risen some one or two lines above the surface of the scalp. In the short cylinder of hair left, the fungus grows still more rapidly, so that the normal structure of the small stump soon becomes undistinguishable. Sometimes the hair breaks off before emerging from the skin, and the fungus, epidermis, and sebaceous matter fill the ends of the piliferous conduits, and form the little prominences which can be seen by the naked eye in this disease, and which gives to the skin a rough anserine appearance. The sporules and mycelium of the plants can sometimes be seen in the form of a white powder on the roots of the broken hairs; sometimes the cutis becomes congested and thickened, and then the plant becomes mixed up with the scales of epidermis, with fatty

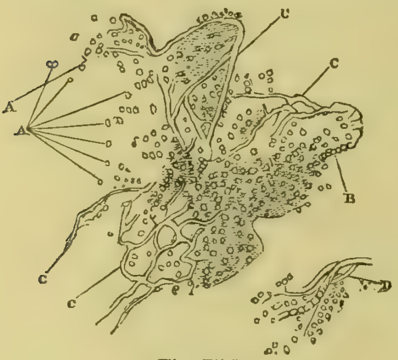

Fig. 75.*

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* Parasitic fungus from a case of *Tinea tonsurans.*—(A, A.) Isolated spores; (B.) Spores united at their ends; (C, C, C.) Empty tubes; (D.) Sporular tube (after BAZIN).

and albuminoid granules, with pus and serous exudation, and so crusts are formed of greater or less thickness in which the growth of the fungus can go on. It exists in the *Herpes tonsurans* of Cazenave, which is the *Porrigo scutulata* of Willan, the *Tinea tonsurans* of Bazin, and the *Trichosis furfuracea* of Erasmus Wilson and Dr. Wood.

The disease commonly called *Ringworm* is what is intended to be described by all those names now mentioned, and on which it seems dermatologists cannot agree.

There seems to be three varieties of this disease, which may be described under the following names:—

1. *Ringworm of the Body* (*Tinea circinatus*) commences by a little rose-coloured and slightly elevated spot, about the size of a fourpenny piece, which shortly becomes the seat of slight furfuraceous desquamation, accompanied by tingling or itching. The spot gradually increases in size, but retains its circular form; and as it extends, the healing process commences in the centre; so that in a short time the red spot is transformed into a large, prominent, erythematous ring, enclosing a portion of sound skin. This process goes on for an indefinite period, the ring gradually increasing till it may have a diameter of four or five inches. When the extent of surface affected is large, the circle is apt to become incomplete, so that various segments of circles appear. The disease may terminate spontaneously, the parasitic fungus being very superficial, the hairs small and rudimentary, so that the parasite dies for want of nourishment. It is apt to affect the face, the neck, the back, and outside of the wrist and hand (Bazin, Anderson).

2. *Ringworm of the Beard* (*Tinea sycosis*) is met with on the upper lip and hairy parts of the cheeks, as well as on the chin, when it affects men; but the hairs of the axillæ or genital organs of females are not exempt from this disease. It commences exactly like *T. circinatus;* but it is not till the deeper structures are involved, and when small indurations occur, surmounted by pustules resembling *acne,* and when the hairs can be pulled out with ease, that the attention of the patient is attracted to the affection.

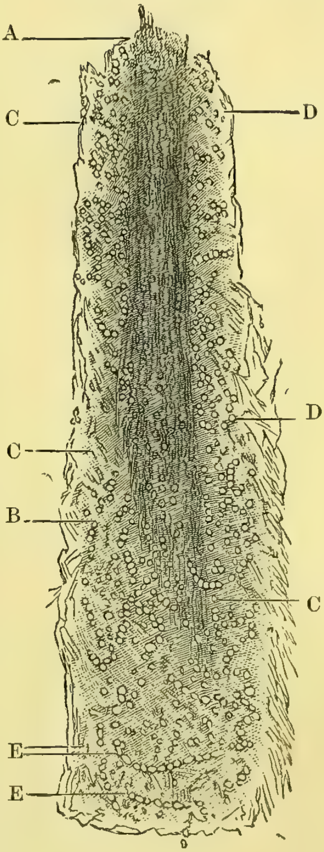

Fig. 76.*

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* Hair from a case of *Sycosis.*—(A.) Broken condition of the superior extremity of the hair; (B.) Rupture of the inferior extremity of the hair; (C, C, C.) Epidermic tunic of the hair; (D, D.) Isolated spores; (E. E.) Chains of spores (after Bazin).

The hairs are thickened, the bulbs flattened and more or less disorganized. The longitudinal fibres of the hair are separated by masses of sporules imbedded between them; and where the fungus accumulates, nodes on the hair indicate the site of such accumulation. The medullary part of the hair is quite disorganized (figs. 76 and 77), and may disappear altogether. In the advanced stage of *sycosis,* when inflammatory and suppurative phenomena prevail, the fungus is difficult to find.

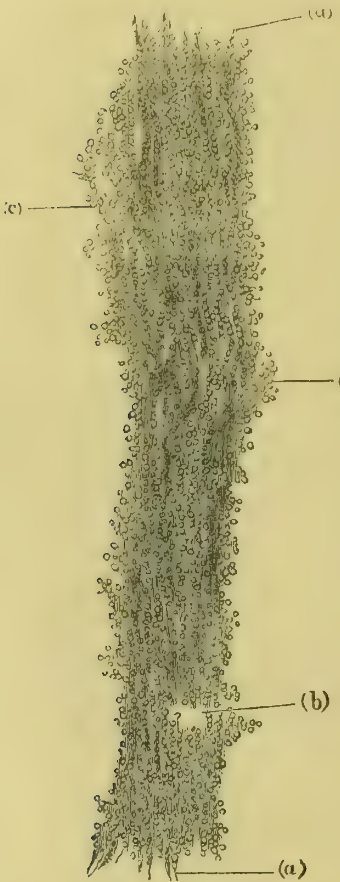

Fig. 77.*

(3.) *Ringworm of the scalp* (*Tinea tonsurans*) is for the most part met with in children. It generally makes its appearance first in the form of rounded patches on different parts of the head, of a scaly or pityriasis-like inflammation. From the irritation induced small vesicles may form. The hairs in the first instance are dull, dry, twisted, and easily extracted; but as the disease advances they become very brittle, and break on attempting to extract them; and as they become more and more friable they break off themselves within a line or two of the skin (Bazin, Anderson). The twisting of the hairs, so frequently observed, is due to—(1.) Plugging up of the follicular orifice by secretion, and detention of the upper part of the shaft of the hair, while its growth at the papilla still continues. Half an inch in length of hair may sometimes be pulled from beneath the false operculum. (2.) It may be due to the presence of *mycelium* in the follicle, clinging to the hair on one side and to the follicle on the other. It thus blocks up the follicle, and holds the hair (while still growing) to the diseased spot (Fox). The epidermis and stumps of the hair become covered over with a characteristic greyish-white powder, which ensheathes the hair. This powder consists of the *sporules* of the fungus. There is slight elevation and puffiness of the skin of the diseased parts, while its colour is bluish or slate-coloured in dark subjects, and greyish-red or yellow in fair persons (Bazin, Anderson). This elevation and puffiness of the skin is due to a granular layer or *stroma,* which, on the addition of *liquor potassæ,* is seen under the microscope to be due to *sporules* of the fungus closely packed together. The amount of fluid influences materially the size of the *sporules* (ROBIN, FOX). When pustules and yellow crusts form, the detection of the fungus is more difficult. The inflammation will last as long as the growth of *fungi* continues, but sometimes they die out spontaneously, and a perfect recovery takes place; but the hair follicles may be obliterated, when permanent *alopecia* of the affected parts is the result, with more or less atrophy of that portion of the scalp (BAZIN, ANDERSON).

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* Hair from a case of *Tinea tonsurans* loaded with *sporules.*—(*a, a.*) Broken ends of the hair; (*b.*) Rupture of the longitudinal fibres; (*c, c.*) Ragged edges of ruptured hair (Dr. T. M. Anderson).

The **Treatment** of ringworm, writes Dr. Parkes, has been long one of the most difficult points in dermatology. Its principles, however, are now well understood, and few cases resist the proper measures. The essential point is to apply to the roots of the hairs a preparation which may destroy the fungus: if this can be done, the disease is cured. It is first of all necessary to remove the hair. This is in part generally accomplished before the case comes under treatment, by the course of the disease; if it has not been sufficiently done, "epilation" can be accomplished by a chemical agent, or by extraction with pincers (fig. 78). The forceps most suitable for this little operation are those about three inches long, having a weak spring, so that the hand may not be fatigued in using them. They should be made so that the two extremities come together very exactly, and do not slide the one upon the other. Each extremity should be a couple of lines broad, so that a fasciculus of hair may be caught up at one time when required; and should be furnished on the inside with very fine, but at the same time blunt, transverse denticulations, so that they may not cut across the brittle hairs. M. Bazin recommends an ointment composed of lime and carbonate of soda, of each one part, and thirty parts of lard, as an agent to remove the hair. The *oil of cade*, however, appears to be the best depilatory known, and with this mode of treatment epilation with the pincers may be combined. If the hairs are pulled out in the proper direction, there is very little pain, especially after the sensibility of the skin has been blunted by the use of the *oil of cade.* The removal of the hairs permits a "parasiticide" solution to be applied to the hair follicles, within which are the prolific spores of the fungus. For this purpose M. Bazin recommends either a solution of *bichloride of mercury* (1 part to 250 of water) or an ointment of the *acetate of copper* (1 part to 500 of lard), about *two* grains to an ounce of water; and a little *alcohol* or *muriate of ammonia* should be used to facilitate the solution of the mineral. The *oil of cade* should be mixed with *glycerine* in the proportion of half a drachm to a drachm of the oil to an ounce of *glycerine.* Kuchenmeister's experience shows that the alcoholic solutions act most powerfully.

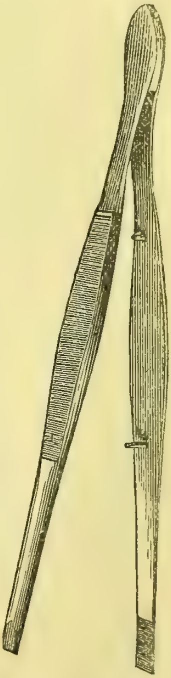

Fig. 78.*

Dr. Parkes has used, with excellent effect, a solution of the *pernitrate*

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* Forceps for epilation (DR. ANDERSON).

of mercury, about one part to thirty or forty of water. This is, however, a very powerful remedy, and is to be cautiously used, as it easily blisters the scalp; also an ointment composed of *sulphate of copper* (one part), *alum* (three parts), and *lard* (twenty to thirty parts, according to the age of the patient).

Probably, however, a better and safer parasiticide than any of these is the *sulphurous acid* as employed by Dr. Jenner, of University College, London, with astonishing results. Diluted with equal parts of *glycerine*, or with two to three parts of water, it is to be applied on a piece of lint to the affected part, and covered with oil-silk to prevent evaporation (*Med. Times*, Aug. 20, 1853).

Dr. Jenner also recommends another formula for sulphur, namely:—

℞. *Sulphur* ʒiv.; *Hydrarg., Ammon.-chloridi, Hydrarg. Sulphuret*, ā ā ʒii.; *Ol. Olivæ*, ʒi.; *Adipis*, ʒiv.; *Creosoti*, ♏v. It must be well rubbed into the patches of tinea.

Dr. Tilbury Fox recommends the following lotion:—

℞. *Sodæ Hyposulph.*, ʒi.; *Aq.*, ʒxii.; *misce.*

Dr. Bennett recommends the internal use of cod-liver oil at the same time that sulphurous applications are used.

My friend, Staff-Surgeon Dr. Davidson, has found the following method of treatment to succeed:—namely, to apply *tincture of iodine* to the affected parts twice a day for fourteen days, and afterwards ointment of the *bichloride* of mercury (*Coros. sublimat.*) After the third or fourth application of the iodine, the disease will cease to spread, and the hair (which may have been thinning rapidly) will cease to fall off. A kind of crust is formed by the application of the iodine, which will scale off in the form of a scurf when the ointment is applied. *Soft soap* (*black soap*) applied in quantity to the patches of disease at bedtime, and washed off in the morning, fits the parts for the better reception of other local remedies. Washing the head after the disease has commenced, and before medical treatment is begun, sometimes tends to spread the disease to parts of the head which had been sound before.

As a constitutional remedy, Dr. Neligan regards the *iodide of arsenic* as the best. He recommends it to be given in doses of $\frac{1}{10}$th of a grain, gradually increased to $\frac{1}{4}$th of a grain for an adult; $\frac{1}{15}$th of a grain for a child six years old; and from $\frac{1}{18}$th to $\frac{1}{20}$th for younger children. To them it may be given in sugar, to adults in the form of a pill (Neligan, *On Diseases of the Scalp*).

TINEA DECALVANS—Syn., ALOPECIA AREATA, PORRIGO DECALVANS.

LATIN EQ., *Tinea decalvans;* FRENCH EQ., *Teigne pelade;* GERMAN EQ., *Tinea decalvans*—Syn., *Alopecia areata;* ITALIAN EQ., *Tigna decalvante.*

Definition.—*A fungus disease, causing the formation of rounded or oval* *patches of baldness, sometimes solitary, more generally multiple. It affects the hairy scalp principally; but the beard, the genital organs, and hairy portions of the skin may also suffer.*

Pathology.—The fungus to which such circumscribed patches of baldness are owing has been named the *Microsporon Audouini*, detected by Gruby in 1843. It is present in the disease commonly called, after Willan, *Porrigo decalvans*, or *Alopecia circumscripta;* or, after Bateman, *Tinea decalvans;* by Bazin, *Tinea achromatosa;* by Anderson, *Alopecia.* It differs from the *Trichophyton* of *Tinea tondens* by its numerous waved filaments, and by the extremely small size of its sporules. It is not found, like the *Trichophyton,* in the interior of the root, but forms round each hair a little tube; the hair then becomes opaque, softens, and breaks off (fig. 79). The *alopecia* is rapid, with or without previous *vitiligo* of the skin; the dermis is not congested, and the epidermis is thin and smooth. In the early stage of the disease the hairs appear dull and lustreless, and more easily extracted than healthy hairs. The skin is reddened, swollen, and slightly itchy. A whitish matter may be seen on the diseased skin and hairs, which is due to the sporules of the fungus. The hairs suddenly fall off from the affected parts, and a round bald patch is left, which is perfectly white, contrasting in its whiteness with the parts of the scalp or skin provided with hairs. The fungus may also be developed in the nail, like favus (BAZIN).

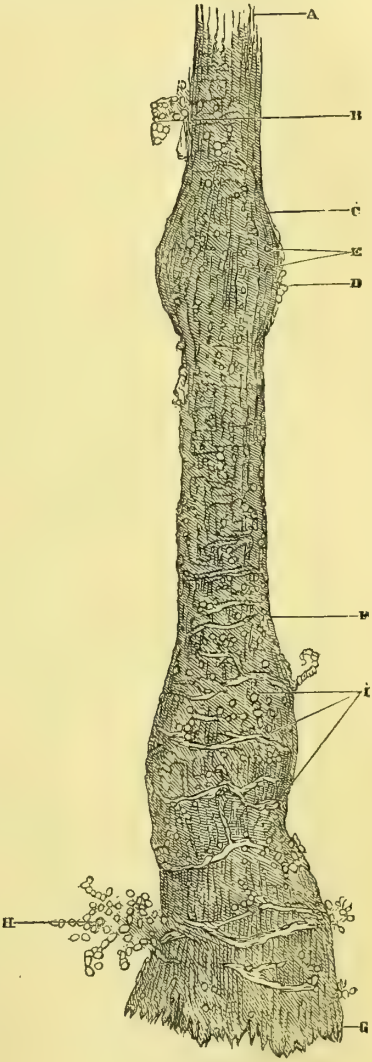

Fig. 79.*

There is an affection which should be distinguished from the *Porrigo decalvans* (or *Alopecia circumscripta*), and which is characterized by a rapid disappearance of pigment from both skin and hair, with or without *alopecia.* M. Bazin includes it in his

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* Fungus of the hairs resulting in *Alopecia.*—(A, F.) Lower part of the hair; (F, G.) Root of the hair without the capsule; (C.) Spheroidal swelling of the hair, due to the accumulation of sporules; (E.) Between the longitudinal fibres of the hair; (D.) Rupture of the longitudinal fibres; (I.) Sporules and tubes of the parasite; (H.) A group of sporules proceeding from (G.) the ruptured root (ANDERSON).

Tinea achromatosa (*Teigne achromateuse*), but does not mention the fact that *alopecia* is not constant; and states that a parasitic plant is present. It is probable, however, that something more than a fungus exists, to cause the total disappearance of pigment from a considerable portion of dermis. Besides, when the hairs return, they are at first quite white and downy, like those on children, and only gradually regain colour; whereas, if the *vitiligo* were owing to a plant, they would most likely not grow at all. The disease appears to be allied to those obscure pigmentary changes which have a much deeper seat than the surface of the body (PARKES). *Vitiligo* is sometimes a congenital affection, and seems to consist in an abnormal distribution of the pigment of the skin; so that there are irregular patches which are quite white, and altogether wanting in pigment, but are surrounded by skin provided with an excess of colouring matter. The hairs proceeding from the portion of the skin deprived of pigment are similarly colourless.

The disease is not quite so readily transmitted as *Tinea tonsurans*, but still it is capable of being transmitted from one person to another, so that children so affected should be separated from their companions (ANDERSON).

Treatment.—This consists—(1.) In preventing the spread of the disease circumferentially. All the hairs, therefore, within a quarter of an inch of the circumference of the patch ought to be carefully extracted. The head should be washed daily with soft or black soap. All the *downy hairs* within the patch must be similarly removed till healthy hairs begin to grow; and some of the parasiticide lotions or ointments must be industriously used. (2.) Stimulants, or even blisters, must be applied to the surface of the bald patch after the fungus has been destroyed. A mixture of equal parts of *collodium* and of *ether cantharidalis* (*collodium vesicans*) is the most useful stimulant. The following lotions may be found advantageous to use alternately with the *collodium* stimulant, namely:—

℞. *Liquor Ammoniæ*, ʒiss.; *Ol. Olivæ*, ʒii.; *Ol. Macidis*, ʒss.; *Spiritus Rosmarini*, ʒiv.; *Aq. Rosæ*, ℥ii.; *misce bene.* To be used night and morning applied over the bald patches.

Mr. Startin recommends a lotion nearly similar, namely:—

℞. *Spt. Ammon. co.*, *f*ʒi.; *Glycerine*, *f*℥ss.; *Tinct. Cantharidis*, *f*ʒi. to *f*ʒii.; *Aq. Rosmar.*, ℥viii.

Mr. Erasmus Wilson recommends the following:—

℞. *Ol. Amygdal; Liq. Ammoniæ*, ā ā ʒi.; *Spt. Rosmarini; Oxymel*, ā ā ℥iii.; *misce.* To use as a lotion.

Of course, none of these agents have any good result if there are no bulbs or roots of hair left whose growth can be stimulated by such application; but when hair falls off, or breaks away after debilitating illnesses, or from dryness of the scalp, they are often useful.

TINEA FAVOSA—*Syn.*, FAVUS, PORRIGO FAVOSA.

Latin Eq., *Tinea favosa*—Idem valet, *Favus;* French Eq., *Teigne favosa;* German Eq., *Tinea favosa*—Syn., *Favus;* Italian Eq., *Tigna favosa.*

Definition.—*A fungus parasitic disease, composed of cup-shaped scabs, sometimes distinct and separate, at other times indistinct or confluent. The fungi* (Achorion Schönleinii, Puccinia Favi) *are capable of being implanted by transference from one person to another. The hairy scalp is its most common site, but the disease may be developed on the face, neck, or limbs.*

Pathology.—The disease has been found to depend on a cryptogamic fungus, which has been named the *Achorion Schönleinii,* after Schönlein, who was the first to suggest that the *yellow favus* crusts in *Porrigo lupinosa* and *P. scutulata* were constituted by a vegetable parasite. The disease with which it is associated is now called indifferently *Favus, Tinea favosa,* or *Porrigo scutulata.* The primary seat of the parasite is in the depth of the hair follicle, outside the layer of the epithelium which covers the root of the hair, and which forms the "inner root sheath" of Kölliker. By using a concentrated solution of *liquor potassæ,* to make the parts transparent, the fungus may be observed with the microscope in the follicle round the hair at the place where it passes through the epidermis. A second form of the disease is that in which the plant is found in depressions on the surface of the skin, forming the yellow honeycomb-like masses which gave the specific name *favus* to the disease, and which, from their frequent buckler-like shape, suggested the term "scutulata." A cuticular elevation is seen, beneath which is a small favus. When the cuticle is raised, a drop of pus sometimes issues; hence the error of those who have considered this disease always pustular. Generally, however, there is no pus or liquid of any kind: the plant grows, and the cuticle over it (supposing it has not been forcibly detached) finally separates, leaving the favus exposed to the air. A third form of the disease is that in which the fungus attacks the nails, and occurs for the most part in those who have been long affected with the favus of the hair follicles, the fungus taking root and germinating beneath the nail (fig. 80). After the spores have commenced to germinate between the superficial and deep epidermic layers, the nail becomes thickened over the affected part, and its colour becomes gradually more and more yellow, owing to the favus matter shining through it. As the fungus increases in growth, it gradually presses on the nail, rendering its longitudinal striæ very evident, and ultimately leading to the formation of fissures in it. As the pressure on the nail increases, its substance gets thinner and thinner, till perforation occurs;

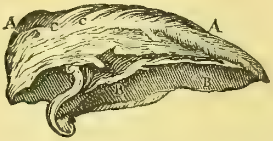

Fig. 80.*

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* (A, A.) Upper surface of nail; (B, B.) Lower surface of nail; (C, C.) *Favus* matter (white in the wood-cut, yellow in the original), running upwards and forwards between the laminæ of the nail (Anderson).

and then a favus cup makes its appearance externally, but more or less deformed (ANDERSON).

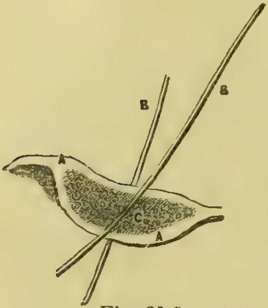

Fig. 81.*

It is important to notice that at first there is, at the point where the favus is about to form, only an increased secretion of epidermis; and sometimes the under surface of the favus is coated by cuticle, which separates it from the compressed and attenuated derma. As it increases in size, and becomes more prominent, the epidermic covering is ruptured. Each favus crust is also enveloped in a capsule of amorphous structure, within which is enclosed the true favus matter (fig. 81).

The favus consists of the *mycelium*, the *spores*, and the *receptacles* of the *Achorion*, together with a finely granular amorphous layer, which forms the external coat of the favus, and is the representative of the amorphous "stroma" which often accompanies the mycelium of *fungi*. In the favus another and distinct fungus can sometimes be found—namely, the *Puccinia favi*—which is easily recognized: it has one extremity (the body) rounded and composed of two cells of unequal size, a superior and an inferior; the other extremity is prolonged into a jointed stem or trunk.

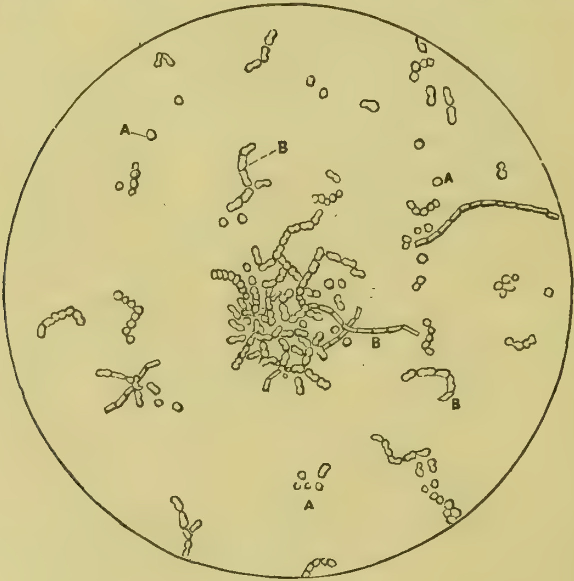

Fig. 82.†

When a little of the favus matter is broken up and examined microscopically, after being acted upon by solution of potash, it is seen to consist of numerous little oval or rounded bodies, the sporules of the fungus having a diameter of about $\frac{1}{3000}$th part of an inch (fig. 82).

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* Favus cup.—(A, A.) Amorphous envelope; (C.) Favus matter; (B, B.) Hairs traversing the Favus cup (ROBIN).

† Fungus matter from a favus crust.—(A, A, A.) The isolated sporules; (B, B, B.) Chains of sporules (DR. ANDERSON).

A number of cells united end to end form simple or jointed and branching tubes (figs. 83 and 84), developed from the sporules (fig. 82). Little granules or nuclei may be seen in the interior of the spores. The tubes vary in diameter, and hairs in the vicinity of the favus crusts are impregnated with the fungus. The disease has been communicated by inoculation from man to man, and from mice to cats, and thence to man (BAZIN, DRAPER, FOX, ANDERSON).

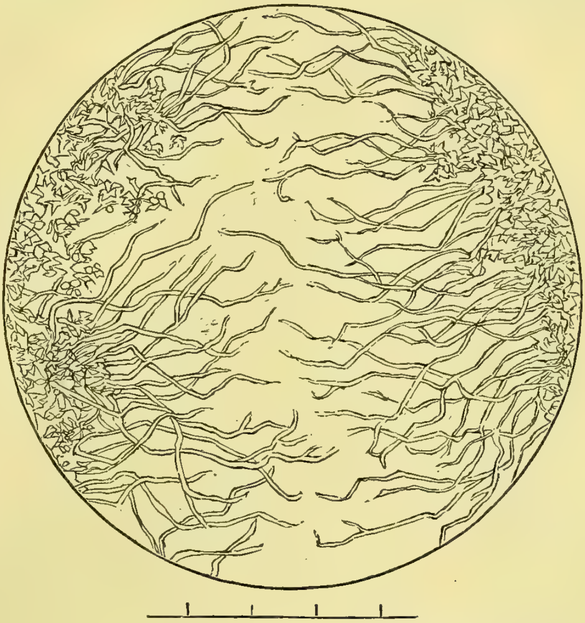

Fig. 83.*—$\frac{1}{1000}$th of an inch × 340 diameters (DR. ANDERSON).

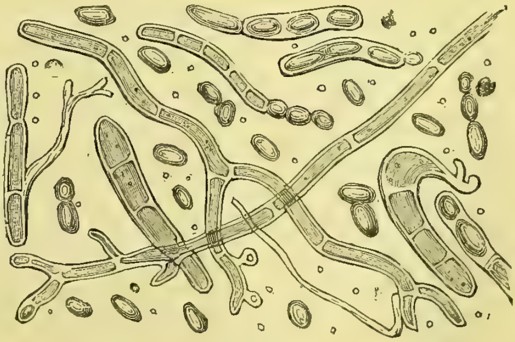

Fig. 84.†

Symptoms.—Favus is the most common and the most inveterate form of scald head. The disease commences with a slight pruritus or itching of a few hours' duration, followed by an eruption of small red *vari*, sensible to the touch and to the sight. These augment in size, and before twelve hours have passed, a yellowish point forms on each of their apices, at first so small as to be only visible under a glass of considerable power. The surface appears now as if covered with specks of a sulphur-yellow colour, and each *varus* appears as if set in the skin, with an umbilicated or depressed centre.

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* Fungus matter from a favus crust, showing branching tubes running inwards to the centre of the figure from the epithelial scabs and sporules at the edges (ANDERSON).

† Showing the mode of reproduction of the *Achorion*, or fungus of the favus (after BENNETT).

If any fluid exudation exist, it does not remain so, but concretes into a dry, brittle, candied, honeycombed-looking scab or crust, which retains the form of the pustule, is similarly cupped or depressed in the centre, covered by the epidermis, while its under surface is marked by a small mammary process which corresponds to the depression of the pustule. The honey-combed appearance of the scab gives the peculiar character of the disease, and hence the term "favus." The crust continues to increase, still preserving its circular form and depressed centre, till it occasionally attains a magnitude of five to six lines in diameter. When the crust is recent, it is of a yellow or fawn colour; as it becomes older, its hue becomes lighter; and, as it is easily reduced to a powder, it has been compared to pulverized sulphur.

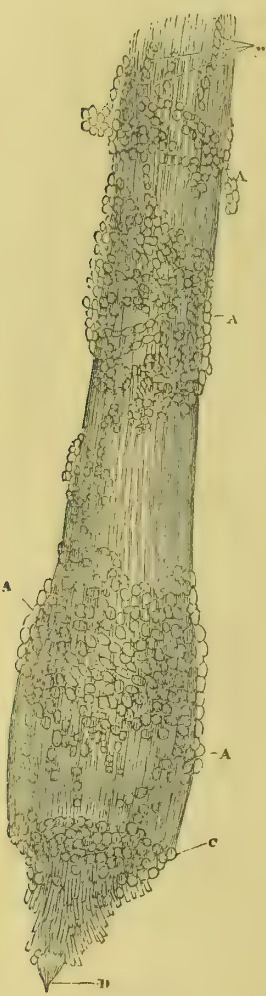

Fig. 85.*

The number of favi is considerable, and they commonly appear in crops, affecting the same or different parts of the head at distant intervals. They may be either distinct or confluent. When very numerous, they are confluent, but the cupped form of the individual crusts may still frequently be recognized; and according to Rayer, should this peculiar form be lost through the copiousness of the secretion, still, by removing the superficial layers, each particular favus, with its central depression, may in general be made out. At a more advanced stage of the disease the epidermis disappears, and a viscid fluid is secreted in such abundance as to form one entire encrustation over the whole head; hence the *Porrigo larvalis*—mask or vizor-like scald head. The smell of the scab is peculiar, and has been compared to that of the urine of a cat, or of a cage in which mice have been kept. It is probably due to a species of alcoholic fermentation (*methylamine*) in connection with the vegetable growth (Lowe).

When a crust of recent formation is removed, a circular depression, wider and deeper than the favus, is seen. At a more advanced stage the ulceration penetrates below the dermoid tissue. Indeed, Alibert says he has never been able to remove a crust, for the purpose of making a preparation, without deeply wounding the scalp, and producing considerable hæmorrhage, while in some cases a deep and extensive ulceration takes place, which has penetrated even to the bones of the cranium.

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* Hair with favus fungus.—(A, A.) Chains of sporules projecting beyond the edges of the hair; (B.) Sporules between the fibres of the hair; (C, D.) Broken-up root end of the hair, with masses of sporules between the *laminæ* (after Kuchenmeister).

The *Porrigo lupinosa* and *Porrigo scutulata* are accidental varieties, in which the scab resembles a lupine, rather than the cell of the honeycomb, and is very rarely seen; or the appearance of the scab is shield-like; and when of some extent and well-marked, the patch is soft, doughy, and painful when pressed upon. Some of the hair appears to be removed by the roots, while other portions are broken off near the scalp, the roots remaining. Those which remain are readily removed by friction, and if pulled, have scarcely any hold of the scalp.

Treatment.—The treatment of the various forms of *favus* is now very strictly determined. Some practitioners, however, still rely entirely on a constitutional treatment, such as small doses of *rhubarb* and *soda*, small doses of *mercury*, some preparation of *iron;* or on vegetable tonics, as the *infusion of cascarilla* or *compound infusion of gentian.* With such treatment, if the health improves, it is believed the fungus will spontaneously disappear. Others, again, as entirely rely on a local treatment, attempting to exterminate the disease by cauterization, or by applying some favourite ointment; and the catalogue of ointments used for this purpose includes nearly all that have at any time been admitted into the pharmacopœia.

As in the last-described disease, the cryptogamous parasite must be destroyed, and its germs eradicated.

The best method to accomplish this is, in the first instance, to shave the head and apply a poultice till all the scabs, or nearly so, are removed; and this being effected, the whole hairy scalp, or site of the favus fungus, should be anointed with some one of the following applications:—The *tar ointment* (*ungt. picis liquidæ*) has hitherto been the orthodox application. This ointment should be washed off night and morning with soft soap and water, and be as often re-applied. The head should be shaved twice or thrice a week, and where there are other children, the affected child should be isolated as much as possible, to prevent the disease from spreading. This form of porrigo, in the early stages, will sometimes yield by washing the part with the *oleum terebinthinæ* night and morning, and cutting the hair close.

Two parts of *carbolic acid* to three parts of *glycerine* and *water* used twice a day, with the daily use of carbolic acid soap, has been also found of service.

But *favus* is a disease often rebellious to every mode of treatment; taken at a favourable moment, however, a simple method of cure sometimes succeeds. Dr. Willis has seen the disease yield to fomentations, or to bread poultices. The application of the lunar caustic round the patches, about a line from their outer margin, is another favourite method of treatment. In the latter periods of the disease, Dr. Willis recommends—

A solution of *sulphate of copper*, in the proportion of seven grains to ten ounces of water; or of the *nitrate of silver* in the same proportions. The mild ointment of the *nitrate of mercury*, a salve of the black sulphuret of the same metal (*sulphuretum hydrargyri* Nigr., ʒj. ad ʒij. adipis ʒj.); the *unguentum picis*, an unguent of the *cocculus Indicus* (ʒj. to ʒij. adipis ℥j.), may be tried one after the other; and in different instances each may have the merit of the cure.

The most effectual remedy is unquestionably the eradication of the affected hairs, and the use of such parasiticides as have been already mentioned under the different forms of *Tinea*. Dr. Anderson finds that when *favus* affects the head, all treatment is absolutely useless except *epilation*. The other methods are merely palliative—the disease reappearing whenever the treatment is stopped. Dr. Bennet, of Edinburgh, places great faith in the olive oil treatment. It is very useful in many cases before proceeding to epilation. After the hair is cut short, and the favus crusts removed with poultices, the parts affected should be smeared thoroughly night and morning with fresh almond oil; and once or twice a week the head should be washed thoroughly with soft soap and warm water. After a few weeks of this treatment the hair becomes less friable, and *epilation* is much more easily and efficiently performed, and does not cause nearly so much local irritation. The hairs are to be removed *singly* with the forceps, not pulled out along with all the healthy growth in their neighbourhood, as used formerly to be done by the barbarous application of the pitch-cap.

This disease occurring on surfaces not particularly covered with hair yields at once to the application of a solution of *sulphate of copper*, or of the *nitrate of silver in water*, or to the *solution of sulphuric acid*, as recommended by Dr. Jenner. The treatment of favus recommended by Robin and Bazin is epilation, and the application of the *corrosive sublimate solution* or of *acetate of copper ointment* (1 part to 500 of lard), to kill the plant still remaining adherent to the hair follicle.

TINEA VERSICOLOR—*Syn.*, PITYRIASIS VERSICOLOR.

Latin Eq., *Tinea Versicolor;* French Eq., *Tinea Versicolor;* German Eq., *Tinea Versicolor*—Syn., *Pityriasis Versicolor;* Italian Eq., *Tigna Versicolore.*

Definition.—*A fungus affection of the skin, characterized by one or more broad irregularly shaped patches of a yellow or yellowish-brown colour, occurring most frequently on the front of the neck, breast, abdomen, and groins, having a predilection for those parts of the body covered by clothing. The patches do not generally rise above the surface of the skin; and there is usually some degree of itching.*

Pathology.—On passing the hand gently over the diseased surface, it may be found to be less smooth than the surrounding skin. It may be seen to be the seat also of a very fine desquamation, or at least of an abnormal condition of the epidermis. Thus far the surface of the affected parts may have a *dusty-like* appearance, *like bran*, and so may merit the name of *Pityriasis versicolor;* but in no other respect has it anything in common with ordinary *pityriasis*—a disease altogether unconnected with parasitic *fungi.* The scales which desquamate from chloasma have a yellowish colour when contrasted with the white scales of such scaly diseases as *Pityriasis vulgaris* or *Psoriasis.* Hence the term Chloasma—from χλοος, "a greenish-yellow colour"—appears more suitable than any of the other names by which the disease has been described.

The disease commences by little spots about the size of a pin-head, which tend to extend circumferentially; circular spots form and unite so as to produce large irregular patches, which may extend till the greater portion of the skin of the trunk is affected. The skin of the diseased parts has a peculiar brownish colour; but the depth of tint varies from the slightest increase of colour to a shade almost black. The colour has been said to resemble diluted bile.

A microscopic fungus, to which the name of *Microsporon furfur* was given by Robin, is the essential cause of the disease. It was discovered by Eichstädt in 1846. Soon afterwards it was described by Sluyter and by Sprengler, who gave a drawing of it. On putting a little of the dust from the desquamating surface under the field of the microscope, and adding a drop of *liquor potassæ,* scales of epidermis are seen mingled with the sporules and tubules of the fungus. The sporules are oval or rounded, and usually collected into large clusters like bunches of grapes, and are so characteristic as almost to be pathognomonic (ANDERSON). The tubes are short and branching.

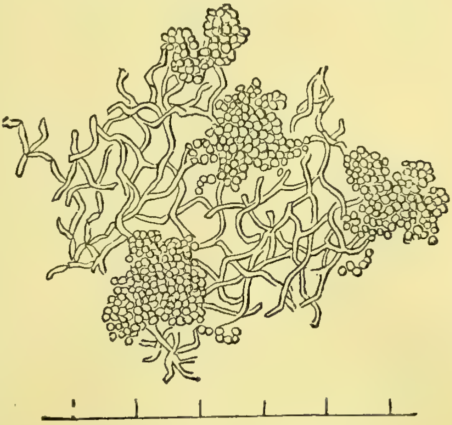

Fig. 86.*

Dr. Anderson and Mr. Startin give numerous instances which prove that chloasma is a disease capable of being propagated from one person to another. It is a common affection with scrofulous persons especially, and may not unlikely be favoured by wearing the same flannel day and night, neglecting to wash the body for fear of catching cold (ANDERSON). It is not uncommon for such people to wear the same flannel next to the skin for a week, a fortnight, three weeks, and among the poor even for a month. And it is by no means an uncommon thing for them to wear the same flannel night and day, not once removing it from the moment it is put on till the time it is considered desirable to have it washed. The consequences of such habits are an accumulation on the surface of the skin of its secretion, and of undetached epithelium, and the consequent formation of a nidus favourable to the growth of the *Microsporon furfurans* (Dr. Jenner, *Med. Times and Gazette*, 1857, p. 651).

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* Shows the grape-like arrangement of the sporules and the short branching tubes of the *Microsporon furfur* in *Chloasma* (after DR. ANDERSON).

Treatment.—Local applications constitute the principal part of the treatment. *A solution of the bichloride of mercury, in the proportion of two grains to an ounce of water,* applied over the affected parts once or twice daily, is generally effectual in destroying the progress of the fungus. Mercurial or sulphur baths have a similar effect, either singly or combined, care being taken to avoid salivation. The use of black soap night and morning is recommended by Dr. Anderson, or the use of the following mixture :—

℞ *Bichloridi., Hydrarg.*, ℈i. ; *Alcoholis*, ℥ss. ; *Saponis Veridis, et Aquæ distillatæ*, ā ā ℥iiss. ; *Ol. Lavandulæ*, ℈i. ; *misce.* To be used night and morning in the same way as the black soap; but if the gums get tender, its use must be suspended.

Great attention must be paid to cleanliness; and the patient should change his flannel clothing very often, and should not sleep in the same flannels that are worn during the day.

MYCETOMA—*Syn.*, MADURA FOOT.

LATIN EQ., *Mycetoma*—Idem valet, *Pes Madurenus;* FRENCH EQ., *Mycetoma;* GERMAN EQ., *Mycetoma*—Syn., *Madurafuss;* ITALIAN EQ., *Micetoma.*

Definition.—*A disease due to the presence of a mucedinous fungus, which eats its way into the bones of the tarsus, metatarsus, and lower ends of the tibia and fibula. In process of time it tends to cause death from exhaustion* (CARTER, BERKELEY).

Pathology.—The fungus foot associated with this disease is confined to the natives of India, who go about with naked feet, and the spores of the fungus might easily be introduced through some scratch, even were it impossible for them to penetrate by the pores of the skin. When once introduced beneath the cuticle, a single spore might soon perform the work of destruction, spreading in every direction, and, according to the peculiar condition of the secretions, the *mycelium* might put on a hundred different modes of growth. Besides, if the fungus is capable of causing the absorption of solid structures like bone, it is easy to conceive that a spore, in contact for sometime with a moist foot, might penetrate the cuticle simply by absorption. Cleanliness, in the first instance, seems to be a preventive; but when the fungus is once established, there seems to be no cure save amputation—which, happily, when resorted to in time, appears to be completely successful, as the disease never spreads beyond a certain point, though, if it be allowed to take its course, death will ensue from the exhaustion consequent on pain and the continuous discharge.

In some cases it would seem as if the foot had been in a diseased state when the fungus was introduced; at least, the history of one case, which apparently commenced with a boil on the instep (which was treated by native doctors, a thorn being used several times as a lancet), indicates a lesion such as might well encourage the growth of a fungus parasite.

It is more than twenty years since surgeons in India first took notice of this affection of the foot in their official reports; and one of the earliest to notice the disease was Dr. Colebrook, of Madras, then Zillah-surgeon at Madura, where the endemic character of the malady was first recognized by the term "Madura foot." An interesting account of the disease was afterwards published by my friend the late Dr. G. R. Ballingall, who was the first to describe the microscopic peculiarities of the disease, and he was led at once to distinguish at sight the tumor of the foot from any simple scrofulous affection, and to detect the prominent features by which he recognized the fungus foot as something *sui generis.* "Cases of diseased foot," peculiar to certain parts of the Bombay Presidency, were recorded by Assistant-Surgeon Bazunjee Rustomjee, in the fifth vol. (N.S.), p. 230, of the *Transactions of the Medical and Physical Society of Bombay*, and which Dr. Carter considers are "the most fully and carefully recorded instances of the 'fungus disease'" which had been published at the time Dr. Carter wrote his report. Most of the cases belong to the second form of fungus described in the text; and practically the disease is regarded in India as a species of *caries.*

The parasitic and fungus nature of this disease has recently been disputed.

Dr. H. V. Carter, the Professor of Anatomy and Physiology at the Grand Medical College of Bombay, made a report in March, 1860, on this formidable fungus disease, and from him the fungus has been named *Chionyphe Carteri.* It occurs in many parts of India and the north-eastern shores of the Persian Gulf. In the Bombay Presidency it has been seen at Kutch, Kattiawar, Guzerat, Scinde, the Deccan, and Lower Koncan. On the Madras side it has been seen at Gumtoor, Bellary, Madura, Cuddapak, some parts of Mysore, and at Trichinopoly. In the Bengal Presidency it prevails to a limited extent round Sirsa; but patients come from Bicaneer, Bhawalpore, and Hissar. It is known amongst Indian medical men as the "fungus foot" or "fungus disease of India;" or under the scientific names of *Podelcoma* or *Mycetoma,* and by several characteristic native names. It is a disease which has hitherto occurred among natives only, and is undoubtedly due to the presence of a *mucedinous fungus,* which eats its way into the bones of the foot and lower ends of the tibia and fibula, penetrating or tunnelling through the tissues of the entire foot by numerous fistulous canals, tending to cause death by exhaustion, unless a timely amputation is made above the diseased part. The history of this disease is now rendered still more interesting from the fact that the Rev. M. J. Berkeley, M.A., F.L.S. (whose authority on *Cryptogamia,* and especially *fungology,* is well known), has succeeded in developing a peculiar mould—the perfect condition of the species—from the black fungous masses sent to him by Dr. Carter. The nature of the disease has thus been more clearly determined; and the account here given is taken chiefly from the description of the disease by Mr. Berkeley, in the second volume of the *Intellectual Observer*, p. 248, and from the writings of Dr. Carter, in the *Bombay Medical and Physical Society's Transactions,* which he kindly sent to the author.

The fungus disease and material of the fungus assumes various forms, three of which may be considered typical:—

1. The first form is that in which the bones of the foot and the lower ends of the leg-bones, just above the ankle (for the disease never ascends higher), are perforated in every direction with roundish cavities, varying in size from that of a pea to that of a nut or pistol-bullet (fig. 87), the cavities being filled up with a dense fungous mass, of a sienna red within, but externally black, and resembling a small dark surface, from which a purulent fetid discharge is poured out, often accompanied by little pieces of the fungus. The masses and granules are imbedded in a whitish semi-opaque glairy substance of homogeneous consistence, while the walls of the canals have an opaque yellow tint, and are readily torn. The whole of the surrounding softer parts are converted into a gelatiniform substance, taking the place of muscles, the tendinous and fatty structures being less readily changed. The foot presents externally the peculiar turgid appearance which it so often assumes in bad cases of scrofula. Besides the canals, pink stains or streaks are observable on the skin, and penetrating the subjacent tissues,

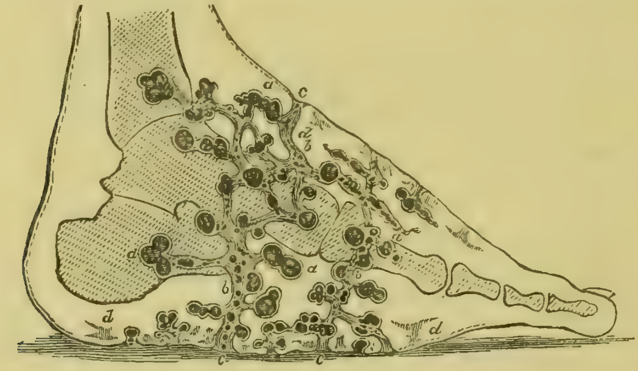

Fig. 87.*

* The figure represents the general appearance on section of the diseased foot in the fungus disease of India. It is based upon dissections, and on three sketches made immediately after amputation of the limbs (CARTER).

(*a*, *a*.) The *fungi*, some of which are globular and of large size, others smaller and more irregular, and others mere granules. The former are lodged in the spherical cavities in the bones. (*b*, *b*.) The *canals* in the soft parts and bones which lead to the free surface of the skin. They frequently communicate, and are lined by a continuous membrane: in them are contained the *fungi*. (In a diagram of this sort it is impossible to represent the soft glairy material which also occupies the canals.) (*c*, *c*.) The *apertures* on the surface where the canals terminate. They are often very numerous, and frequently in them may be seen impacted the black particles. (*d*, *d*.) The pink-coloured *stains* or *streaks* in the skin, above described. They are common to both varieties of the disease, and by them it is supposed the growth is multiplied.

It is to this variety of the affection that the term "fungus disease," which correctly expresses its nature, was, *par excellence*, originally applied. Hitherto no other instances of it have been distinguished, except those described by Dr. Carter; hence it may perhaps be regarded as comparatively unfrequent. The fungus particles or masses are of a deep black colour, and of firm consistence: they are sometimes as large and as round as a pistol-bullet.

filled with spherical or ovate groups of minute, bright orange-coloured particles, and containing occasionally a few larger cells, the nature of which has not at present been ascertained, though it is conjectured that they present the earliest appearance assumed by new attacks of the disease. (Specimens of the disease are to be seen in the Museum of the Army Medical School at Netley.)

Of the structure of these large truffle-like bodies, the figure (88) copied from the *Intellectual Observer*, from a specimen examined immediately after amputation, will give the reader an idea. The parts in which the structure is most visible present precisely the characters of a true *Oidium*, such as *O. fulvum*. Short, beaded, tawny threads arise from a common base, consisting of cylindrical articulated filaments, having at their tips large spore-like cells. These, however, do not appear to germinate *in situ*, but to become enormously dilated, their albuminous contents assuming at length a resinous consistence, while many of them burst, and nothing remains except fragments of the old cell-walls. The resinous matter is inflammable, but its exact chemical nature has not yet been ascertained.

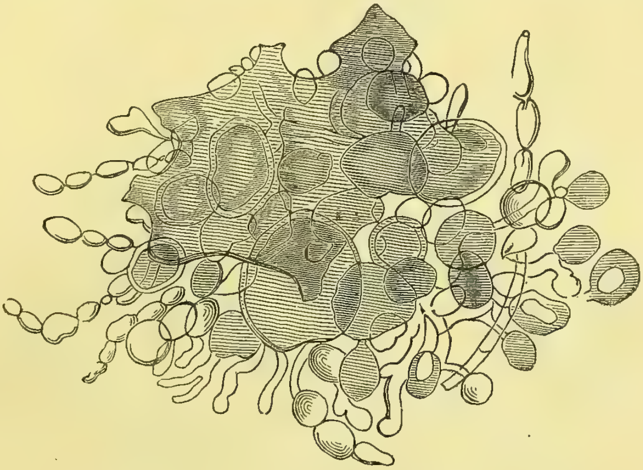

Fig. 88.*

The fungus of the foot resembles closely the genus *Mucor*, but there is no *columella* in the *sporangium*—a character which accords with *Chionyphe* rather than with *Mucor*. Indeed, there does not seem to be a single character in which the fungus of the Indian disease differs generically from the *Chionyphe*. Its mature form is seen to be composed of a thin filamentous stratum, spreading in every direction over paste, on which it may be propagated so as to form little slightly raised patches.

The species has been named by Mr. Berkeley "*Chionyphe Carteri*," the name serving to record the labours of the two Carters, "united in their love of science, though not in consanguinity." It is highly probable, as Mr. Berkeley observes, that many of our common moulds occasionally commence with a similar condition. The first indications of vegetation on tainted meat or paste assume the form of little gelatinous spots, of various colours, consisting of extremely minute distinct cells, and these seem to be an early stage of a common species of *Aspergillus* and *Penicillium*, or other genera. If there be any truth in the notion entertained by Mr. Berkeley, that hospital gangrene depends upon some vegetation of this nature, acting as a putrefactive ferment, there may be good reason for believing that the red spots in question are really the commencement of the disease under consideration.

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* Structure of the truffle-like bodies, presenting the characters of *Oidium fulvum*,—short, beaded threads arising from a common base, consisting of articulated filaments having at their tips large spore-like cells.

In the second form under which the disease appears, the black fungous masses are entirely wanting, and in their stead, masses are found of what looks like sloughing tissue. White granules, however, occur in the cavities and in the discharge, which appear to be a form of the same fungus, though the identity has not been proved. Under the microscope it wears the appearance of a congeries of large cells filled with smaller ones. Whether the perfect form of the plant be the same or not, the phases of the disease produced by it are exactly the same, and the malady admits of no other remedy than amputation of the foot.*

A third form of the disease is known under the name of the *Madura foot*, from its having occurred at Madura. In this case the foot becomes enormously enlarged about the instep, though not so much at the ankle, while the toes are hypertrophied, and almost lost or imbedded in the mass. The small bones are nearly destroyed, leaving behind a pallid or reddish tissue, while the others are more or less excavated. There are the same canals and external sanious apertures. In some parts they are filled with the same fleshy tissue, in others lined with it, where large cavities are formed by the junction of several canals, containing broken-up osseous tissue from the exposed bones around, grey fragments, and masses of pigment. The pink colour is partly owing to a general diffusion of pigment, which tinges the oil-globules, and partly to the presence of very numerous single or aggregated elliptic particles. These granules are from the $\frac{1}{50}$th to the $\frac{1}{30}$th of an inch in diameter, and occur sometimes as single ellipses, sometimes as two combined at the extremities of their major axes, and sometimes as square bodies with rounded extremities divided into four. They are quite visible to the naked eye, insomuch that, when the sawn surface is first exposed to view, it appears as if strewed with grains of red pepper; and pains were therefore taken by Dr. Carter to assure himself that they were not particles accidentally introduced through the open window. Further examination convinced him that, though different in colour, they were similar in essence to the granules described in the second form. None of the black fungus masses appeared, but there were globular opaque bodies of various sizes, which now require notice, and which, though at first apparently so different, are closely connected with the fungus of the first form. The foundation of these bodies consists of one or more large mother-cells filled with a mass of daughter-cells. These are clothed externally with a radiating growth, assuming a vast variety of forms. The structure often so exactly simulates that of minute moulds, that it is very difficult to get rid of the notion that they are really vegetable growths. Pure sulphuric ether, however, dissolves them completely, and shows that they are merely different forms assumed by stearine. Sometimes the white mass consists of straight slender threads radiating in every direction, each of which is surmounted by a globose or elliptic spore-like body, while occasionally the threads or crystals are shorter and the globe irregular. Sometimes the globules are absent, and in one case the fundamental cell budded like the

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* In the *second variety of Mycetoma* we find three or four different kinds of particles: these, however, are always light-coloured and soft, and generally very small or minute. Of them only one form, certainly the more common, has been noticed by writers: it is that in which each particle is seen to be invested by a crystalline coat. The truly fungus nature of the more common kinds of granules or particles, and of that striking instance of the disease from Madura, is as yet only matter of inference.

It seems desirable that every step in the investigation of this disease (the elucidation of which is committed, as it were, to the medical officers of India) should be based on direct and repeated observation, and that speculation be refrained from as being at least useless.

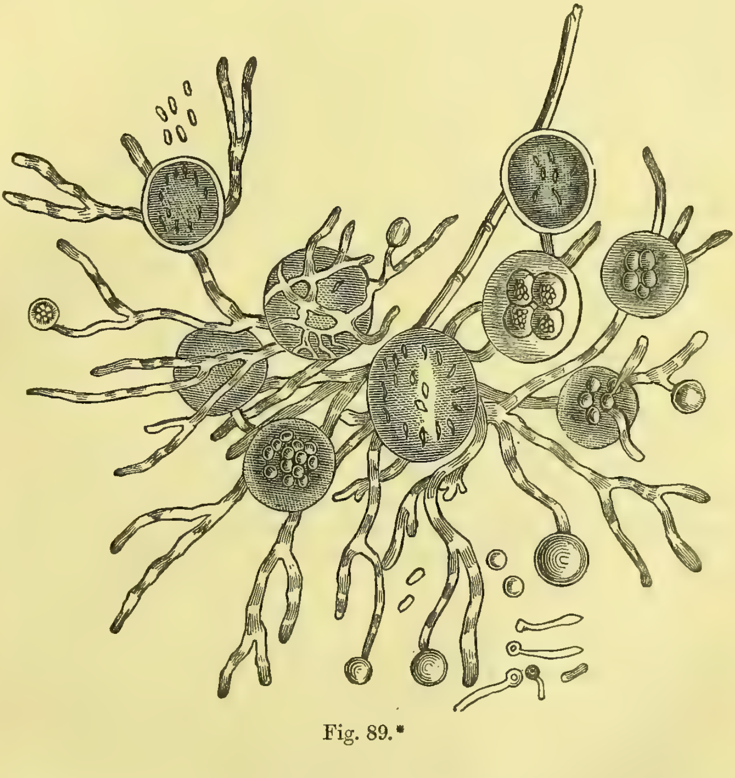

Fig. 89.*

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* Fundamental cells of the *Chionyphe Carteri* developed from the *fungus foot of India,* budding like the receptacle of an *Aspergillus* (Berkeley and Carter).

receptacle of an *Aspergillus*, each new cell being separated by an articulation and supported on a short stalk.

Sometimes the outer coat consists of regularly dichotomous or trichotomous fascicles of linear crystals, which are free above. Sometimes, on the contrary, the fascicles are dilated above with ciliary processes, or labiated; while occasionally there are straight radiating bodies surmounted by a globular mass, pierced and surrounded by cilia. Another form appears under the guise of little feathers; while a not unfrequent one consists of leaf-like, oblong strongly accuminate scales, simulating the leaves of mosses. *The foundation is, however, in every case, an organized cell, the red colour of whose daughter-cells is precisely that of the oidium-like threads of the black fungus.* Whatever may be thought of the second and third forms of vegetable growth, this at least must be considered as identical with the first, though at present the *Chionyphe* has not been propagated from its globules—so closely involved in stearine that their germination is scarcely probable. There is not the slightest ground for supposing that the disease depends on inoculation with the spores of any of the truly parasitic *fungi* belonging to the tribe of *rusts* and *mildews*, but great reason, on the contrary, for looking to the origin of the fungus of the foot amongst the *mucors*, even were there not something like direct proof. It is well known that *mucedinous fungi* make their appearance within cavities of vegetables which have no apparent connection with the outward air. Nothing, for example, is more common than to find a pink mould (*Trichatherium roseum*) in the middle of a nut, and an allied vegetable production (*Dactylium soyerium*) has been found in an unbroken egg. Even the cells of plants themselves produce *fungi* which fructify within them. How the spores are carried there is at present a mystery which may some day be cleared up, as the origin of many intestinal worms has been, which can no longer be brought forward as an argument for equivocal generation.

Symptoms.—In the first variety the general form of the foot is oval, being much enlarged about the ankle and over the instep. On either side of the ankle-joint, on the dorsum of the foot near the toes, likewise on the sole, are numerous small soft swellings or tubercles, as large as a pea or marble, having pouting, puckered apertures, leading to fistulous canals; and the skin surrounding these apertures appears lighter in colour than elsewhere. (See specimens in the Museum of the Army Medical School at Netley.) The canals sometimes lead directly to the bone; and a discoloured glairy fluid, which exudes from the canal, sometimes carries with it a few black, gritty particles. The toes are distorted and displaced upwards, and the muscles of the calf of the leg atrophied. Such a condition has been known to exist for more than twelve years; and the natural course of the disease is fatal. The external characters of the other forms are similar to those already described.

The changes produced in the bones, as shown by maceration, are of such a kind that a cursory examination of them at once suggests the conclusion that some *organic* agency has been at work to produce the changes. The cancellated tissue becomes the seat of cavities more or less spherical, and sometimes most perfectly so. These cavities vary in size from little more than that of a pin's head to that of a round bullet; and the walls of the cavities are formed by open cancellous tissue.

From being in close juxtaposition, they frequently open into each other, producing large vaulted gaps or spaces; and not only so, but every cavity, large or small, has an open communication, directly or indirectly, with the external surface. In the more superficial ones some part of the wall is wanting, so that the cavities look like mere round holes of various depths; but in the deeper cavities a regular tunnel, more generally straight than curved, serves as the channel of communication. The diameter of these passages is sometimes equal to that of the cavities themselves. In the recent state the sinuses of the soft parts are often plugged up by superficial collections of the fungus; but when cleared out, they are found to lead down to the tunnelled passages, or into the rounded holes—the peculiar *loci* of the *fungus.* The fungus has only once been seen affecting the hand (COLEBROOK).

Description of Incipient Fungus Disease.—The lesion has the appearance of an elongated flattened tumor, the rounded surface of which is marked with white patches, and presents seven circular depressed spots of one-third to one-half inch, or more, in diameter. The superficial dark layer of cuticle is cast off, leaving a very regular circular white surface, the centre of which presents a depression, closed at the bottom by a brownish layer, very thin in the middle. It was found on section that a small cavity existed beneath this depressed spot, or a tubular prolongation was detected running down through the remaining thickened cuticle and cutis into the subcutaneous cellular tissue, where it was not difficult to find the fungus particles, pink or yellowish-coloured, and also in the cavity above named, in the superficial part of the cutis, or even on the surface of the latter,—the cuticle being raised.

Another and smaller specimen most clearly showed the development of the *fungi* at the very spot where, in all probability, their germs were first produced. In other parts there is a prolongation of the growth into the subjacent tissues, and there pink-coloured particles were to be seen. The local nature of the whole affection—its very beginning—was here unmistakably displayed; and the superficial appearance of the skin gave the impression that a vesicle or blister had once existed there, not at all unlike that left after a *Guinea worm* has begun to discharge, as it is well known that the end of the worm makes its appearance in the centre of such a circular spot.

A further examination of the fungous particles showed their perfect resemblance to those of older specimens, and bodies not unlike spores were occasionally seen.

SCABIES—*Syn.*, ITCH.

LATIN EQ., *Scabies*—Idem valet, *Psora;* FRENCH EQ., *Gale;* GERMAN EQ., *Scabies*—Syn., *Krätze;* ITALIAN EQ., *Rogna.*

Definition. — *Lesions consisting of an eruption of distinct slightly acuminated papules or vesicles, accompanied with constant itching, due to* *the irritation caused by the burrowing underneath the epidermis of a female Acarus* ("Sarceptes scabei"), *for the purpose of depositing her eggs.*

Pathology.—The nature of this parasite has been described in vol. i., under "Parasites."

Symptoms.—The phenomena of the eruption of scabies are more often papular than vesicular; and the markedness of these phenomena depend partly on the length of time that the person has been affected, the number of *Acari* developed, and the degree of sensibility of the skin. It is known to infest sheep and dogs (YOUATT); and therefore hair does not preclude its existence; but it seems to prefer delicate parts of the skin—for example, the inner surfaces of the fingers, and folds of the skin between the fingers, the wrists and palms of the hands, the penis in the male and nipples in the female, as well as the hips, the feet, the umbilicus, and axillæ.

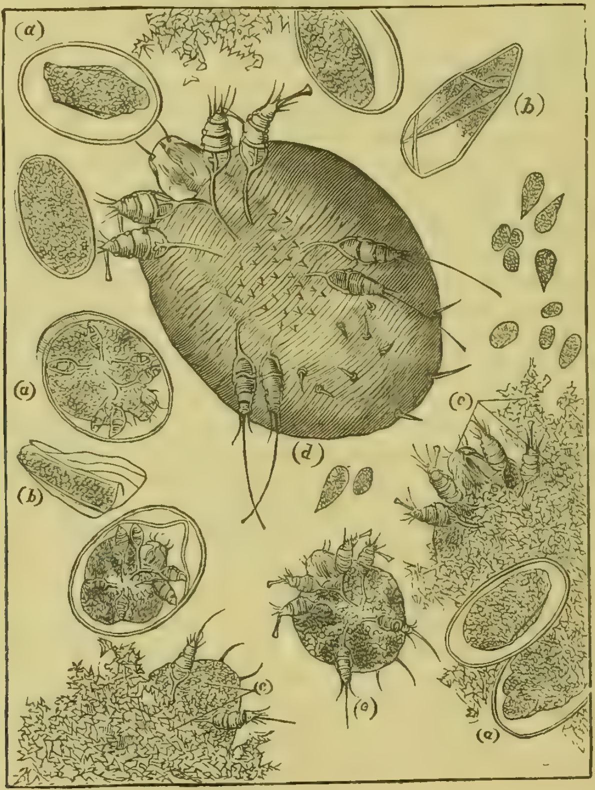

Fig. 90.*

Itching, increasing at night, first attracts attention, and is a characteristic symptom. It becomes general all over the body, and the scratching aggravates the eruption. The *prurigo* of itch is generally most expressed on the forearm, lower part of the abdomen, and the upper and inner part of the thighs. Vesicular eruption is most usual on or about the fingers and nipples of females; and pustules may be met with in children whose skin is delicate, especially on the hands, feet, and hips.

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* Crust from a case of the so-called *Scabies Norvegica* which occurred in Würtzburg.—(*a, a, a.*) Eggs of the *Acarus* in various stages of development; (*b, b.*) Egg-shells; (*c, c.*) Fragments of *Acari;* (*d, d.*) Female *Acarus;* (*e.*) Larva. The little oval irregular-shaped masses are presumed to be the excrement (after ANDERSON).

There is a severe form of scabies common in Norway and some parts of continental Europe, in which the greater portion of the skin of the body becomes thickened, the natural furrows increase in depth, the pigmentary deposit is greatly augmented, and a fine white desquamation covers the surface. Here and there papules may be seen, either with dots of coagulated blood, or with whitish crusts on their summits. In these crusts portions of *Acari* and their exuviæ, excrement, and eggs may be found. The hairy scalp, covered with a crust which adheres firmly, of a bark-like consistence and yellow colour, is studded with fine openings for the hairs, which are glued together. On the under surface, and in the furrows of this crust, multitudes of *Acari* may be seen. Such are the main features of a case recorded by Bergh, of Copenhagen, and related by Dr. Anderson in his excellent little treatise already referred to. The itching was intolerable. The crusts were principally composed of the *Acarus*, its exuviæ, excrement, eggs, and egg-husks (fig. 90). A piece of the most superficial and dense part of the crust, less than half a line square, contained 2 female *Acari*, 8 six-legged young, 21 pieces *Acari*, 6 eggs, 53 egg-shells, and about 1,030 pieces of excrement. "In the deepest and softest parts of the crusts, amongst the remains of deceased generations—partly in holes and passages, partly between particles of the crusts, partly on their free surfaces, masses of living *Acari* wallowed and tumbled about." The cases in which such a severe form of *scabies* has occurred have been characterized by extreme filth and neglect of treatment, and the irritation and course of the disease has tended to a fatal termination by pneumonia and hyperæmia of the brain.

Treatment.—Such applications are to be made to the skin that, while they tend to kill the *Acari*, they will not increase the irritation of the dermis; and if the *Acari* are thus destroyed, the eruptions will in general subside in due course.

If much irritation of the skin prevails, warm baths are to be prescribed, and opium may be given internally.

In healthy adults the whole body of the patient ought first of all to be thoroughly scrubbed over with good black (soft) soap, and the process continued for at least half an hour. The patient should then get into a warm bath, in which he should remain for another half hour. Having thus washed and dried himself thoroughly, he is to rub himself over with the following ointment:—

℞. *Subcarbonatis Potassæ*, ʒi.; *Sulphuris*, ʒii.; *Axungiæ*, ℥xii.; *misce.*

Next morning a warm bath is to be taken, to clean the surface of the body from the remains of the anointing of the previous night. The cure ought now to be complete, so far as the destruction of the *Acari* is concerned (Hardy, Helmerich, Anderson). The genuine pomade of Helmerich is one-third stronger than that which has been just quoted from Dr. Anderson, who considers it too irritant. The potash in the black soap and ointment acts as a solvent of the epidermis, and thus allows the sulphur to come into more immediate contact with the *Acarus*. A warm bath and plenty of hot water ablution completes the cleansing process.

The treatment, may, however, be inapplicable to children, females, and men with delicate skins, or constitutional affections of the skin.

In them, although the principle of treatment is the same, the process of cure must be more slowly conducted by less powerfully irritant substances. The patient having cleansed himself thoroughly in a warm bath, with ordinary yellow soap, the following lotion may be applied:—

℞. *Calcis*, ℥ss.; *Sulphuris*, ℥i.; *Aquæ*, ℥viii. These ingredients are to be boiled and stirred constantly till a homogeneous mixture is produced, which is to be strained through a sieve.

These ingredients ought to produce a quantity more than sufficient for one person, and should be rubbed into the skin, not too roughly, every night for several evenings. The cases of scabies in the Belgian army are treated by this lotion (Velminskz, Anderson).

When the person affected is predisposed to *eczematous* eruptions, the following application is recommended:—

℞ *Sulphuris, Olei Fagi*, ā ā ʒvi.; *Saponis Virdis, Axungiæ*, ā ā lbj.; *Cretæ*, ʒiv.; *misce.*

This ointment should be well rubbed in, after the skin has been prepared for it by the use of the warm bath and cleaning the body with common yellow soap. The potash in the black soap of the ointment acts as already stated, the chalk tends to remove the epidermis mechanically, the tar counteracts the tendency to eczema, and the sulphur destroys the *Acari*. The ointment ought to be left on overnight (if the skin is not too irritable), and should be washed off in the morning (Wilkinson, Hebra, Anderson). Specific printed directions should be given to each patient: and cards are useful for this purpose, similar to those in use in the Dispensary for Skin Diseases in Glasgow. The following are the directions *printed* on each card, and which is given to each patient along with the quantity of ointment required:—

"1st. Scrub the *whole* of your body (except the head) as firmly as possible, without hurting yourself, with black soap and water.

"2d. Sit in a hot bath for twenty minutes, or if you cannot get a bath, wash yourself with hot water thoroughly.

"3d. Rub some of the ointment firmly into the skin of the whole body (except the head) for twenty minutes. Let the ointment remain on the body all night.

"Repeat these processes every night for three nights, and then return to the dispensary.

"Besides, put all your washing clothes into boiling water, and iron all your other clothes *thoroughly* with a hot iron."

If such methods are systematically carried out itch cases ought never to occupy hospital beds, either in civil or in military life.

The ordinary compound sulphur ointment of the pharmacopœia is also an efficient remedy. The cure being complete so far as the person is concerned, care must be taken to destroy the *Acarus* and its eggs which may be amongst the clothes of the patient. For this purpose they should be exposed to hot air at the temperature of at least 150° Fahr., or, if possible, boiled in water, or exposed to the action of sulphur by steaming them amongst sulphur vapours.

CHAPTER XXIII.

THE PERNICIOUS INFLUENCE OF SOME POISONS.

The College of Physicians indicates a consideration of poisons and their effects under the following heads:—

(1.) Metals and their Salts.
(2.) Caustic Alkalies.
(3.) Metalloids.
(4.) Acids.
(5.) Vegetable Poisons.
(6.) Animal Poisons.
(7.) Gaseous Poisons.
(8.) Mechanical Irritants.
(9.) Poisoned Wounds.

Time and space do not permit of such a detailed consideration, so that a few poisonous agents and their effects can now only be noticed here.

LEAD PALSY.

LATIN EQ., *Paralysis ex plumbo;* FRENCH EQ., *Paralysie Saturniné;* GERMAN EQ., *Beilähmung;* ITALIAN EQ., *Paralisi litargirosa.*

Definition.—*A series of morbid phenomena induced by the absorption of the salts of lead contained in solution in drinking-waters, or in various foods and drinks, or conveyed into the system through the integuments of those who are in the habit of handling the soluble salts of lead; or through the pulmonary mucous membrane of those exposed to the influence of vapours containing lead.*

Pathology.—The theory of this disease is, that the lead being absorbed, produces a peculiarly painful affection of the alimentary canal, termed *lead colic,* or *painters' colic* (eq. *colum ex plumbo*). It may also affect the muscles of the extremities, producing palsy; and finally it produces ulceration of the gums and alveolar processes, accompanied by a peculiar blue line, which was first pointed out by Dr. Burton, of St.

Thomas's Hospital. This blue line is seen along the free margin of the gums, but is absent where a tooth or stump is wanting. To this the name of "blue gum" has been given. A stain, also from lead, sometimes affects the conjunctiva.

The fact of the lead being absorbed and mingled with the blood is demonstrated by the circumstance that lead has been obtained from the coats of the stomach of a dog poisoned by lead, even as late as a month after poisoning. Again, MM. Duvergie and Guibourt have detected lead in the brain of the human subject, and Dr. Budd has detected it, not only in the human brain, but also in the muscles. Many pathologists are inclined to believe that the blue line observed in the gums of persons poisoned by lead is owing to the presence of lead in some peculiar state of combination, as with some of the constituents of the tartar of the teeth (TOMES); and from the observations of the late Dr. George Wilson, it appears that there are various tissues of the body for which lead has an affinity, and that it is more apt to be found in some organs than in others. The stomach and cœcum of a pony that died a fortnight after being removed from the sources whence lead was received into its body through the ingesta, having been carefully analyzed by this able chemist, it was found that, while the contents of these viscera did not contain the metal, the substance of their tissues yielded it in small but manifest quantity. In a mare dying under similar conditions, the following organs were examined—(1.) A part of the lungs; (2.) A part of the heart; (3.) A part of the large intestine and contents; (4.) A part of the stomach and duodenum; (5.) The spleen; (6.) One kidney; (7.) A portion of the liver. The spleen yielded the most abundant and most deeply coloured precipitates; while the intestinal canal gave the faintest indications. Next to the spleen the liver yielded most, afterwards the lungs, then the kidney, next the heart, and least the intestines.

The general result of these analyses went to show,—*First*, That in cases of slow poisoning with lead, the metal comes to be diffused through the entire body, and exerts its poisonous action, though in an unequal degree, on every organ; and *Second*, That lead having once entered the body, leaves it again very slowly, so that long after an animal has ceased to receive lead in its food or drink, or by any other medium, we may expect to find the metal in its tissues; and the restoration to health is always protracted. The lead passes off by the urine sometimes in large quantities, but very slowly (PARKES). Fletcher found in the urine of a man with lead colic no less than 4·8 grains of metallic lead in 100 grains of solid matter of the urine (*Dublin Med. Press*, 1848); and Dr. Parkes records a case in which the last exposure to the influence of lead was on the 20th December, 1852, and lead was found in the urine before treatment commenced on the 16th June, 1853, and the blue line was still perceptible below the edge of the lower gum.

Colica Pictonum rarely causes death. De Haen opened many persons that died from lead poisoning, and found in all a constriction of the colon, and in a certain number a similar affection of the cœcum. Merat opened seven cases, and observed a similar appearance. Dubois de Rochfort says that he found in two cases intussusception of the intestines; but Andrel examined five cases, Louis one case, and Martin another, without finding any morbid appearance.

Mr. Hunter had an opportunity of examining the state of the muscles of the palsied hand and arm of a painter who died of a broken thigh in St. George's Hospital, and found them all of a cream colour, being probably in a condition of fatty degeneration. In the paralysis arising from the poison of lead the tissues of the muscles and nerves are early affected, and at a subsequent period the nervous centres became implicated. The muscles seem to be first affected, and through them the nerves participate in the contamination, which gradually advances to the nervous centres in the severer forms of the disease. This is shown by the fact that local paralysis always precedes, and generally for some considerable time, those phenomena which indicate disease of the nervous centres, and which show that contamination of the system has been great. The evidence now is also abundant which proves that it is lead existing in the affected tissues which thus contaminates and impairs their function. The morbid appearances in the brain and spinal cord are generally such as denote imperfect or depraved nutrition of those centres. The brain is pale and soft, and its convolution wasted, the *sulci* between them wide, and sometimes patches of white softening are to be seen in the hemispheres; and this seems to be more particularly the case in those who have had paroxysms of an epileptic nature, and in whose brains lead has been detected (TODD).

The introduction of lead into the system has taken place in a great variety of ways. In France the pernicious effects were wont to occur from putting a lump of litharge into the *vin gâtée*, to cover its acidity and render it saleable; and from this having been practised to a great extent by the Pictones, or inhabitants of Poictou, the disease has been named *Colica Pictonum.* In the cider counties of Great Britain this disease formerly existed to a great extent, and has been termed Devonshire colic, or *Colica Damnoniensis.* The impregnation of cider with lead in this country was generally the effect of accident, and arose from the troughs in which the apples were crushed having the different pieces of stone of which they are composed clamped together with iron, and fixed by melted lead. In some districts it was the practice to line the entire press with lead, or to tip them with that metal. It was a custom, also, almost universal, to make the upper part of the boiling vessel of lead; while some growers, in managing weak ciders, put a leaden weight in the cask to sweeten the liquor. From these and perhaps other causes, Sir George Baker found the cider he examined to contain four and a half grains of lead in eighteen bottles, or a quarter of a grain in each bottle. In the West Indies lead poisoning appears to have been produced by using leaden worms to the stills, by which the rum became impregnated with this metal. There are many other minor sources of poisoning by lead; as keeping pickles or preserves in glazed earthen vessels, and colouring confections with preparations of lead. The still and other machinery used in the distillation of fermented liquors being now constructed of metals so combined as not to be acted upon by acid fruits or sugar, diseases from the action of lead are no longer so common as they were wont to be, but are confined principally to labourers in the lead manufactories and to painters. The use of paint where lead exists is the most common source of its absorption in this country; and hence house painters are those most frequently affected. The paint called "flatting" (or that which is mixed with a large amount of turpentine, so as to give a *flat, dead,* or *non-glistening* surface) is the most injurious to the workman. The turpentine, readily passing off by evaporation, carries with it a small supply of lead, which is constantly and gradually inhaled, or it is left on the skin to be absorbed, or mixing with saliva, it gets into the stomach. By one or all of these ways the system becomes affected, first through the circulation of the blood, and subsequently by the constituent tissues of the organ combining, in some form or other, with lead, which is thus deposited in them.

All ages, both sexes, and all classes are liable to the poisonous action of this metal; but the workers in lead have been at all times the greatest sufferers. Women in this country often suffer from lead colic, but it is rare to find them paralytic; men suffer both from the colic and the characteristic palsy.

Symptoms.—The quantity of lead necessary to produce its specific results, or the time it takes to accumulate in the system when introduced, is not determined; and both the dose and the time varies greatly in different individuals. Sometimes all its most pernicious effects are produced by one dose taken by the mouth; and then again, if introduced by the skin, months, and even years, may elapse before the system is laid under its influence. As a general rule, however, a much smaller dose will produce colic than is necessary to produce palsy.

When the dose is of such intensity as to produce *colica Pictonum,* the symptoms do not differ, except in being of greater intensity, from those which mark ordinary colic. There is the same dragging and twisting pain, and the same relief by pressure; the same absence of fever; the same unhurried pulse; the same constipation, only more obstinate; and, in the worst cases, the same vomiting. Andral, who treated upwards of 500 cases at La Charitié in the course of eight years, says it is not strictly true that the pain in lead colic is always diminished on pressure; for in the greater number of cases pressure neither augments nor diminishes the pain, while in some cases the sufferings of the patient are increased by it. He also says it is as common to find the abdomen distended with gas as to find it drawn in, and the *rectus* muscle strongly contracted on both sides. The symptoms peculiar to this form of colic are occasionally an attack of epilepsy, and an ulcerated state of the mouth, accompanied by a blue line on the dental edge of the gum—a discovery which the profession owes to the careful observation of Dr. Burton.

The duration of the constipation which attends *colica Pictonum* is very various. Three or four days may elapse before a stool is procured, and when the case is early submitted to medical treatment, seldom more than a few hours; but fifteen days have been known to elapse without a stool. As soon as the bowels act, the great severity of the disease is mitigated; every symptom is gradually relieved, and the disease generally terminates within a week.

But when palsy is the result of the absorption of lead, a painful state of the arms often precedes it, as well as repeated attacks of the characteristic *colic*. The nerves of the forearm and hand become first affected, and the affection is in general limited to the upper extremities, the extensor muscles of the hands and fingers become paralyzed, so that when the arms are stretched out, the hands hang down by their own weight, causing what the patients term "*wrist-drop*." The disease may be sometimes seen limited to one finger. More commonly, however, it affects the whole arm, and sometimes so completely that the patient can execute no movement with it; and when lifted up, it falls like an inert mass. Sometimes the extensor muscles of the limb are alone affected; and in this case the hand is often strongly closed by the powerful and unresisted action of the flexors. When the case continues for a few weeks, the posterior surface of the forearms (where the extensor muscles are situated) is rendered quite concave, from atrophy and consequent shrinking of the muscles. The arm loses its plumpness, and even the interosseus membrane may be felt. The muscles of the ball of the thumb are also in a similarly wasted state. In general both arms are palsied, but not equally so, one being slightly more affected than the other. Supposing both sets of muscles to be equally palsied, the patient usually recovers the use of the *flexors* before that of the *extensors*, so that he can carry a weight hanging in his hand before he can shave himself. This restoration of the lost power is usually accompanied by more or less pain. The duration of the palsy under any treatment is always long, and often lasts many months, and in some cases years. Both colic and palsy may occur an indefinite number of times. When epilepsy is produced, the fit does not differ from epilepsy due to other causes.

Diagnosis.—The colic of lead poisoning can only be distinguished from ordinary colic by the history of the case, and by the blue line on the dental edge of the gums, but which is present only where the teeth or their stumps are in the alveoli, and ceases where a tooth is completely wanting.

The palsy is to be distinguished from cerebral paraplegia by the history of the case, by the integrity of the intellect, and by the blue line on the gum. A most important means of diagnosis in paralytic affections is the electric current properly administered. The excitability of the muscles is always much diminished in paralysis from lead, as Dr. Althaus clearly shows, and often it is entirely lost. Such is the case, not only when the muscles are atrophied, but when the bulk of the muscles is only slightly diminished; and even after the voluntary movements have regained their former power, the excitability of the muscles to the electric current still remains impaired. The relation of the muscles to the stimulus of Faradisation helps in doubtful cases to establish the diagnosis, as the excitability of the muscles is always either lost or diminished in lead palsy, whilst it is normal in spontaneous paralysis. *Therefore, when the muscles of a paralytic limb move well under the influence of the electric current, we may fairly conclude that there is no lead in the system* (Althaus *On Paralysis, Neuralgia, &c.*, p. 72).

Prognosis.—The termination of lead colic, except where the dose has been in such excess as to produce death in a few hours, is always favourable; and those cases which prove fatal are generally such as have been exposed to the cumulative influence of lead for a long time, and who have been intemperate.

The palsy does not appear greatly to affect the health of the patient; but in some cases it has hitherto not been cured or relieved. In general, however, the patient recovers, although perhaps not completely. Drs. Garrod and J. W. Begbie have satisfactorily demonstrated that lead poisoning of the system exerts a remarkable influence as a predisposing cause of gout; and my friend, Dr. W. England, of Winchester, as the result of his experience, spontaneously volunteered to me a similar remark. Inveterate forms of dyspepsia may be traced in many cases to the influence of salts of lead in the drinking-water, its pernicious influence expressing itself differently in different constitutions, although never amounting either to colic or to paralysis.

Treatment.—The treatment of *colica Pictonum* is extremely simple. The objects to be obtained are to procure stools, a copious discharge of urine, and perspiration, with a view of eliminating the poison from the body. To allay pain is also an urgent necessity. For these purposes five grains of calomel, fifteen grains of jalap, and one grain of opium, should be administered as soon as the patient is seen; and at the end of two hours about two ounces of *camphor mixture*, combined with a drachm of *sulphate of magnesia* and twenty minims of *tincture of hyoscyamus* may be given every two or every four hours, till the bowels are freely evacuated, when relief more or less complete is obtained. The mixture should be continued at proper intervals for three, four, or five days, when the patient, though greatly weakened, has in general recovered.

In a few cases, however, the pain continues, and with considerable severity, after the bowels have been freely evacuated. The practice in these instances is to apply a blister to the epigastrium, and to keep the blister open for a few hours; and this additional application will generally complete the cure.

The patient is also much relieved if placed in a warm bath, and at the same time directed to inject repeated enemata of hot water, that stools may be readily obtained. In the absence of the warm bath, a large lintseed or mustard poultice should be applied over the abdomen.

With respect to the cure of lead palsy, an endless variety of treatment, both local and general, has been tried, but with so little positive result that, when the patient has recovered, it has been doubtful whether it has been owing to the great length of time that has elapsed, or to the medicines he has been taking. It has been believed that sulphur has the power of neutralizing the effects of lead by forming some innocuous compound with it. It is not known, however, whether any such compound is really formed, but a remedy of a very useful kind exists in the form of a *sulphur bath* (TODD). The ingredients of this bath consist of from *two* to *four* ounces of the *sulphuret of potassium*, mixed with from twenty to thirty gallons of water. The *ergot of rye* (*secale cornutum*) has been said to produce a considerable increase in the power of the flexor muscles of the arm in about a fortnight, and the improvement gradually extends to the flexors, till at the end of about three months the patient has recovered. This may have been the natural result of elimination of the lead. The experiments of Orfila long ago rendered it probable that lead is removed from the body by the kidneys; and *iodide of potassium* promotes the elimination of lead in this way. It may be used with advantage combined or not with the *citrate of iron*, the use of iron, in some form or other, having been found of benefit in cases of palsy from lead contamination. Dr Parkes has chemically proved that lead can be made to pass off by the urine, by the action of *iodide of potassium*, in the same way as mercury is known to be eliminated.

The principle upon which the *iodide of potassium* acts has been pointed out in a most interesting manner by Melsens. He assumes that the lead is in actual union with the affected tissues, being retained among them as an *insoluble* compound. The *iodide of potassium*, after its absorption into the blood, combines with the lead, and forms with it a new and soluble salt. The poison is thus liberated from its union with the injured part, dissolved out from the damaged fibre, and once more set afloat in the circulation. Thus the poison and the remedy are cast out together by the urine (MELSENS, WILLIAM BUDD). It is necessary, however, to notice the dangerous phenomena which may at first supervene on the administration of *iodide of potassium* in cases of lead poisoning; and great caution is necessary in the employment of this remedy in man for the first few days. At the moment when the metallic compounds fixed in the body become dissolved or transformed, the phenomena of acute poisoning may occur, caused by their liberation. So much is this the case, that the treatment may be supposed to be at first hurtful rather than beneficial. The patient should have beside him a graduated solution of the *iodide of potassium;* and should begin with a small doze (fifteen grains during the twenty-four hours), and afterwards increase or diminish it according to his pains and sensations (MELSENS).

Galvanism, in the form of Faradisation, ought to be used as a local stimulant to the nerves, with the precaution that its application is not to be continued too long each time. Ten or fifteen minutes, at three different periods of the day, or of every second day, and persevered in for not less than four weeks, will be found of great service (TODD, ALTHAUS). The beneficial influence will follow, although, in the commencement of the treatment, even a current of very high tension does not cause any movement whatever in the paralyzed muscles. In such cases the beneficial influence seems attributable to the restoration of mobility to the molecules of nerve and muscle by an induced current, and which is necessary to enable them to be physiologically active. Severe shocks, especially in the commencement of the treatment, should therefore be carefully avoided, as by such the weakened excitability of nerve and muscle may be reduced, in place of being fostered and developed (ALTHAUS, l. c., p. 112 and 119). It would be rational, however, to defer the application of galvanism till the lead has been completely eliminated.

ERGOTISM.

LATIN EQ., *Morbus cerealis;* FRENCH EQ., *Ergotisme;* GERMAN EQ., *Mutterkornbrand;* ITALIAN EQ., *Ergotismo.*

Definition.—*A train of morbid phenomena produced by the slow and cumulative action of a specific poison in a fungus peculiar to wheat and rye, and which gives rise to convulsions, gangrene of the extremities, and death, or to symptoms of ill-health.*

Historical Notice and Pathology.—Ergotism is a disease very little known in this country; but it is not unfrequent that diseased, unripe, or damaged grain of any kind, and especially *rye,* is observed to be injurious to the animal economy. *Wheat, rice,* and such-like grains, are equally injurious when similarly unsound; and the most frequent form of this unsoundness consists in the development of a fungus (the *Ergotætia abortifaciens* or *cockspur*) upon the grain, to which the name of *ergot* has been given. In this ergot fungus there is a large proportion of fixed oil (WIGGER). A morbid state is said to be produced by various other poisonous fungi, such as the *Amanita muscaria* and *citruria;* the *Hypophyllum sanguineam;* and by plants such as the *lolium temulentum* being mingled with the grain. A most characteristic phenomenon which results is a form of gangrene which follows as a specific effect of the poison in parts remote from the source of circulation. It is a species of mortification which has not been much seen in this country, but is well known, and has been frequently observed in different parts of Europe, particularly in France, in some districts of which it has been repeatedly known to prevail as an endemic disease, where *rye* forms the principal food of the inhabitants. The *ergot* or *cockspur* in *rye* is only apt to occur after very rainy or moist seasons. In this disease the grains of rye grow to a large size, acquire a black colour, and have a compact, horny consistence. The attention of the public was first called to the peculiar mortification which follows the use of this diseased rye by M. Dodard, in the *Journal des Savans* for 1676. He described the appearance of the affected parts, and mentions that fowls fed with the grain soon died. In 1694 its frequent occurrence is noticed at Sologne, and it prevailed amongst the patients of the Hôtel Dieu of Orleans. The upper and lower extremities of those suffering from the disease "grew as dry as touchwood and as emaciated as Egyptian mummies." In 1710 it was again the subject of treatment in the Hôtel Dieu at Orleans, from its extreme prevalence in the neighbourhood. About fifty people, men and children, were admitted that season into the hospital. During thirty-three years the endemic appeared three or four times, and always in those rainy or moist seasons in which the rye contained a large proportion of the cockspur—more than a fourth. The mortification always began in the toes, and extended gradually along the foot and leg, till it sometimes rose to the upper part of the thigh. In some patients the gangrened part came away of its own accord; in others it became necessary to assist nature by amputation. In some instances death succeeded to amputation, the disease having continued to extend to the trunk of the body. It is particularly mentioned in a report on the subject to the Royal Academy of Sciences at Paris, that the rye of Sologne, in the year 1709, contained fully *one-fourth* part of the cockspur. The disease generally began in one or both feet, with pain, redness, and a sensation of heat as burning as the fire. At the end of some days these symptoms ceased as quickly as they had come on, when the sensation of extreme heat was changed to cold. The part affected became black, like a piece of charcoal, and as dry as if it had been passed through the fire. A line of separation tended to form between the dead and the living parts, like that which appears in the separation of a slough produced by the application of the cautery.

The disease prevailed in Switzerland in 1709 and 1716, and is described by Langius, of Lucerne. M. Garroud, a physician of Dauphiny, where the disease prevailed in 1709, makes some very important observations on the different symptoms apt to predominate in different individuals. Some patients suffered very violent pain, with an insufferable sensation of heat, although the part felt cold to the touch. In other patients redness, with much swelling, supervened, attended by fever and delirium. The separation of the dead parts from the living took place with excruciating pain. The gangrene was not in every instance dry. Animals were found to die of the specific gangrene when forced to swallow the diseased *rye;* and they refused to eat food containing it.

The experiments and observations of Tessier show that a given quantity is required to produce the specific effects, and that the action of the poison is cumulative. Do the fungi continue to be developed in the blood?

The history of some cases of mortification of the limbs related by Dr. Charlton Woolaston, in the *Philosophical Transactions* for 1762, shows that it may occur in this country from eating *wheat* diseased similarly to the *rye;* and Sir William Wilde, of Dublin, has recorded its occasional occurrence in Ireland.

Symptoms.—The gangrenous form of ergotism is ushered in by excessive lassitude, more or less protracted, and accompanied with fever; the extremities become painful, cold, and rigid, benumbed, and almost insensible, and are with difficulty capable of movement. Severe internal pains of the limbs prevail (acrodynia), greatly aggravated by heat. It extends by degrees from the toes to the legs and thighs, and from the fingers to the arms and shoulders, when sphacelus supervenes. With the exception of slight febrile heat, the constitutional disturbance appears to be slight, and in this respect resembles scurvy. Ricker has recently described the early symptoms of a case of poisoning by bread containing *ergot.* A family of six persons partook of the bread, and all suffered from the same symptoms—namely, dryness of the throat, epigastric oppression, nauseous taste, mucous and biliary vomiting, vertigo, stupor, and diarrhœa (*New Syden. Society Year-Book*, 1861). Seeing that symptoms vary greatly in severity, probably in proportion to the amount of diseased food taken, and the poisonous nature of the particular fungus which affects it, we may have the expression of some phenomena more fully than others—for example, *acrodynia.* Under this name Chomel described a painful affection of the wrists and ankles which prevailed in Paris in 1827, 1828, and 1829. It was then so prevalent among the soldiers in the barracks of Lourcine, that 560 men were affected out of 700; and in that of Comtille, 200 men out of 500. Since 1828 no case has been recorded in the barracks or military hospitals (PARKES). In 1859 M. Barudel observed three cases in the military prison at Lyons; and in spite of negative results of the examination of the quality of the food, Dr. Parkes is inclined to believe that the cause lay there, and probably in ergot of the flour (*Army Medical Department—Sanitary Report* for 1860, p. 358).

The general train of symptoms produced by the use of diseased grain assumes two forms—namely, the spasmodic or the gangrenous. The spasmodic form commences with a sense of tingling or itching in the feet, followed by cardialgia and similar tingling sensations in the hands and head. Violent contractions of the hands and feet follow, which seem to affect each particular joint, and the pain is said to resemble that of a dislocation. The sensations are also sometimes described as of a bruising kind; and the body is bathed in copious sweats. The symptoms intermit during intervals of two or three days of a remission at one time. Drowsiness, giddiness, indistinctness of vision, and an irregular gait are constant phenomena. Coma and epileptic convulsions are apt to supervene, which generally indicate a fatal result. An enormous appetite accompanies this train of evils. Spots like those of purpura appear on the face, and the disease rarely abates before the third week.

Treatment.—Considerable differences of opinion prevail regarding the treatment of this *dietic* disease. The cause, in the first instance, must be ascertained and removed. To obviate the effects it has already produced, the constitutional treatment must be directed to improve the state of the blood. Tonics and stimulants are to be administered, after a free employment of evacuant remedies, to clear out the alimentary canal completely. The chlorates of potash and of soda, with antispasmodics, tonics, and narcotics, are especially indicated. *Camphor, musk, ammonia, capsicum,* may be particularly mentioned; and the strength of the patient is to be supported by light, nourishing, and wholesome food.

DELIRIUM TREMENS.

LATIN EQ., *Delirium alcoholicum;* FRENCH EQ., *Delirium tremens;* GERMAN EQ., *Delirium tremens*—Syn., *Säuferwahn sinn;* ITALIAN EQ., *Delirium tremens.*

Definition.—*A train of morbid phenomena, produced by the slow and cumulative action of alcohol, in the various forms in which it is used as a drink. Delirium is one of the most prominent features of the morbid state, which is otherwise characterized by hallucinations, dread, tremors of the tendons and muscles of the hands and limbs, watchfulness, absence of sleep, great frequency of pulse. A thick, creamy fur loads the tongue, and a cool, humid, or perspiring surface prevails; while the patient gives forth a peculiar odour, of a saccharo-alcoholic description, more or less strong.*

Pathology.—This disease has only been recognized and described since the beginning of this century. The *Experimental Inquiry* of Dr. John Percy, in 1839, illustrating the physiological action of alcohol; an inquiry into the *Phyisology of Temperance*, by Dr. Carpenter; the recent *Pathological Observations on the Bodies of Known Drunkards,* by Drs. Roesch and Francis Ogston (1855); and, lastly, a most able and interesting review on the "Treatment of Delirium Tremens," in the *Brit. and For. Med.-Chir. Review* for October, 1859, are contributions which have placed on a more sure foundation our previous theoretical information regarding morbid states which follow the persistent use of alcohol.

The term *alcoholism* is used to denote various symptoms of disease attending morbid processes of various kinds which are capable of being traced to the use of stimulants containing alcohol. The immediate effects of *intemperance* in the use of alcoholic fluids, the nature of *delirium tremens,* and of *spontaneous combustion,* may be embraced under the general designation of *alcoholism.* This term is used in the sense analogous to that in which we use the terms *mercurialism, ergotism, narcotism,* and the like;—the agents inducing these specific states acting after the manner of a cumulative poison. The progress of modern science has distinctly demonstrated the poisonous action of alcohol; and an account of the nature of delirium tremens, as well as the grounds on which its treatment must be based, are now alike founded on this knowledge. Tiedemann and Gmelin in 1820, and Majendie in 1823, detected alcohol by its odour in the blood. The fluid found in the ventricles of the brain had also been observed to have the smell, the taste, and the inflammability of gin (Sir A. Carlisle). In 1828 it was theoretically advanced by Leovèille that *delirium tremens* consisted in an exalted state of the vital powers of the brain, excited by molecules saturated with alcohol absorbed from the surface of the stomach and bowels, and carried into the current of the circulation. Now it is a matter of fact, determined by direct experiment, as well as by casual observation, that alcohol is absorbed directly into the circulation, and is capable of acting as a direct poison upon the nervous tissue through which the infected blood circulates. Alcohol has been found in the blood, in the urine, in the bile, in the fluid of the serous membranes, in the brain matter, and in the liver (Percy, Ogston). Its odours can be easily detected in the breath, and the habitual immoderate drinker exhales a distinct alcoholic and saccharine odour more or less strong. His clothes at last acquire a spirituous aroma, every part of his body being long thoroughly imbued with alcohol (Craigie). This odour is generally so well expressed in cases of *delirium tremens* that a place has been given to a statement of the fact amongst the characters embodied in the definition. Dr. Percy's experiments directly support these statements, and prove at the same time the great rapidity with which alcohol passes into the current of the circulation. He injected strong alcohol into the stomachs of dogs; and within two minutes after completing the injection, their respiratory and cardiac movements ceased; the stomach was found nearly empty after death, whilst the blood was highly charged with alcohol.

I once had an opportunity of examining the body of a person who for many years had been in the habit of drinking daily a large quantity of brandy. He died of *typhoid asthenia,* with characteristic degeneration of nearly every important organ of the body and of the blood-vessels. The fluid collected from the cavities of the brain, consisting of serum and some blood, contained 2·6 *per cent.* by volume, and 2·1 *per cent.* by weight, of alcohol. This quantitative analysis was made for me by my friend Dr. F. S. B. F. de Chaumont, with Giessler's vaporimeter.

The pernicious effects of the continuous use of alcoholic stimuli on the organs and tissues of the body have been deduced from a careful study of the morbid appearances, of *a chronic kind,* met with in the bodies of individuals known to have lived intemperate lives, and who *had perished suddenly* from the effects of accident, suicide, or homicide, and while apparently in ordinary health and activity. The extent of the chronic changes in the various organs of these individuals is found to have been far in excess of what could have been reasonably looked for in a like number of persons of the same age, and of temperate habits, suddenly cut off while apparently in average health and vigour. The *cumulative* effects of long-continued intemperance have been clearly proved by Dr. Ogston's observations; and the results of his *post-mortem* inspections, on the whole, support the conclusions which have been arrived at on *theoretical grounds* as to the injurious effects of alcohol in excess. The following statements contain a summary of these results:—(1.) *The nervous centres* present the greatest amount of morbid change, the morbid appearances within the head extending over 92 *per cent.* of those examined. By this observation the theoretical remarks of Leovèille, Craigie, and Carpenter are clearly established. (2.) *The changes in respiratory organs* succeed in frequency those of the nervous centres, yielding a *per centage* of 63·24 of those examined. (3.) *Morbid changes* in the liver are next in order of frequency, and are due to enlargement, granular degeneration, the nutmeg-like congestion, and, lastly, the fatty state. (4.) Next to changes in the liver come those in *the heart and large arteries.* (5.) Next are those of the kidneys. (6.) Least frequent of all are morbid changes in the *alimentary canal.*

Two orders of changes may be observed to result from intemperance in the use of alcoholic fluids—namely, one set of long duration, or which at least must have taken some considerable time before they could be completed; another set of shorter duration, and which probably are more closely connected with the immediate symptoms which precede the fatal event.

The abnormal changes in the cranium, the substance of the brain, its convolutions and cerebral ventricles, all indicate the prolonged action of a morbid poison. The prolonged action of the alcoholic poison on the cranial contents is to produce induration of the cerebral and cerebellar substance in by far the largest number of cases, coincident with an increased amount of sub-arachnoid serum; while the steatomatous degeneration of the small arteries leads to *atrophy* of the convolutions and *œdema* of the brain.

When spirituous liquors are introduced into the stomach they tend to coagulate, in the first instance, all albuminous articles of food or fluid with which they come in contact: as an irritant, they stimulate the glandular secretions from the mucous membrane, and ultimately lead to permanent congestion of the vessels, to spurious melanotic deposit in the mucous tissue, and to thickening of the gastric substance. By the veins and absorbents of the stomach the alcohol mixes with the blood, and immediately acts as a stimulant to all the viscera with which it is brought in contact. The functions of the brain are at once stimulated, and ideas follow in more rapid succession; the liver is excited to secrete an excess of sugar, by the immediate action of the stimulant on its tissue (DRS. HARLEY and BERNARD). The flow of urine is excited in a similar manner.

In these effects it is impossible not to recognize the operation of an agent most pernicious in its ultimate results, when taken in habitual excess; but most valuable as a remedial agent, when its action is understood and appreciated (consult Dr. Anstie's valuable work on *Stimulants and Narcotics: their Mutual Relations*). The mere coagulation of the albuminous articles of food and fluid is very different from that effected by the gastric fluids, and tends to render the articles more difficult of solution by the gastric juice.

In these facts it is impossible not to recognize that alcohol being absorbed, a double series of morbid results ensue. On the one hand, a train of phenomena are induced, partly of a *chemical* nature and partly *physiological* or *vital*. The general nutrition of the body suffers; and a bad state of health is at last induced, of a peculiar kind, sometimes described as the "*drunkard's cachexia* or *dyscrasia*." This state of the system is characterized by positive irritation, which very soon succeeds to the intemperate use of alcohol, and which is manifested in a variety of ways; sometimes by an unnaturally voracious appetite; but those who over-indulge in the use of such stimuli subsequently suffer a total disrelish for food; they become unable to eat, and dyspeptic symptoms of various kinds betray the irritable state of the alimentary canal, such as stomach-ache, the frequent generation of gases, water-brash, heartburn, squeamishness, vomiting, and palpitations of the heart. A constipated condition of the intestines, attended with deficiency in the power to expel their contents, is very soon established, and sometimes ascribed to the deficient secretion of bile, which is known not to be secreted in due quantity; and there is every reason to believe, although the fact is not proved, that its quality is deteriorated. Its functional agency on the food and fluids in the intestines is therefore diminished.

If we follow the course of alcoholic absorption through the vascular and pulmonary system, it is found unquestionably to *retard* the motion of the blood, while it produces a temporary increase in the action of the heart, and a congestion of the whole system of the pulmonary capillary vessels. Respiration is thus, in the first instance, rendered from four to six times more frequent per minute than it otherwise is; and various symptoms of accumulation of blood within the chest, and pulmonary congestion especially, are apt to occur. A short, tickling cough is a most constant phenomenon. Dr. Craigie remarks that all the spirit-drinkers whom he has ever seen or known have been either subject to chronic cough or dyspnœa, or have laboured under chronic, dry bronchial disorder, with asthma. That the use of spirituous liquors ultimately retards the motion of the blood in the vessels is known by experiments on the lower animals, and by pathological observations. Thus far we have a morbid condition induced which is highly favourable to the accumulation of fat in the blood, and such an accumulation has been proved to take place. It has been shown by Becquerel and Rodier that fat increases in the blood, in most acute diseases, when the biliary secretion is retarded, and when a scanty amount of food is taken. These are the very conditions which obtain in alcoholism. It is in cases of undoubted drunkenness that fats have occurred in the blood in such obvious quantity as to leave no doubt of its presence. In such cases a milky character is imparted to the serum—a condition which may be recognized by simple inspection; while a microscopic examination and treatment with ether will establish the diagnosis, and distinguish the fat from colourless blood-corpuscles, or from molecules of albumen. Slighter degrees of this condition are altogether physiological, and are met with during the period of digestion, after eating substances rich in fat (A. BUCHANAN, VOGEL). But the extreme degrees of this condition have been especially met with in drunkards. J. Frank regards the white and fatty blood as having its origin in the abuse of alcoholic drinks (*Hannoversche Annalen,* 1847, p. 283, quoted by Vogel). Dr. Adams (*Trans. Med. and Phys. Society of Calcutta*) mentions the case of a sergeant at Fort-William who went to bed drunk, and was found dead in the morning. The vessels of the brain were greatly distended with blood, and oil was seen floating in it. Rayer relates the case of a man who, after drinking largely of punch, destroyed himself by the fumes of charcoal. The blood and the urine contained globules of oil. Serules, of Strasbourg, records similar phenomena. Thus, it is shown by abundant testimony that the blood becomes surcharged with unchanged and unused material, and contains at least thirty *per cent.* more of carbon than in the normal state. The order of events by which this state comes about is somewhat as follows:—Alcohol is directly absorbed by the blood-vessels without undergoing any change or decomposition. Part of it is eliminated very slowly as alcohol by the lungs, by the liver, and by the kidneys; but appears to tarry in largest amount in the liver and in the brain (PARKES). Another portion is decomposed. Its hydrogen enters into combination with oxygen to form water, which, with acetic acid, having been produced, carbonic acid and water are formed. Oxygen is thus diverted from its proper function, the exhalation of carbonic acid at the lungs is diminished both absolutely and relatively, and less urea is excreted by the kidneys than is consistent with health; but the pulmonary aqueous vapour is not lessened (BÖCKER and HAMMOND, quoted by PARKES). The water of the urine is diminished, the chlorine is greatly lessened, as well as the acids and bases. All the evidence, therefore, points to the effect of alcohol as causing the retention of substances which ought to be eliminated; and this retention of the effete matter is still more intensified by the stimulant action of alcohol increasing for a limited time the frequency of functional acts, followed as it is by a corresponding depression. In this way impaired health is soon brought about, tending to wasting of the tissues generally; and so long as any alcohol remains in the blood *as alcohol,* a certain toxic or poisonous effect continues to be produced upon the nervous system through which the poisoned blood circulates. If a constant supply of the alcohol is kept up, the phenomena of *alcoholism* become chronic or persistent; and acute paroxysms, generally in the form of *delirium tremens,* supervene, which is at once the most common and the most prominent evidence of *alcoholism.* In other instances the degeneration of several vital organs generally may become so excessive that death follows by *asthenia,* or with typhoid phenomena ending in coma.

When mixed with blood out of the body, spirituous liquors cause more or less coagulation according to their strength and concentration; and when applied to the blood-vessels in the transparent parts of animals, they can be seen to produce the same effects. The congestion that constantly exists in the mucous membranes of the lungs and stomach is evidence of the retarded motion of the blood. The fact that hæmorrhoidal swellings are always aggravated by the use of alcoholic fluids is the result of retarded motion of the blood in the hæmorrhoidal vessels. When death occurs from poisonous doses, either in animals or in man, although the dose is at first followed by increased frequency of the pulse, yet in a short time the pulse becomes rapid and small, while the extremities become cold, and the power of generating heat is suspended in proportion as the blood progresses slowly and more slowly through the pulmonary capillaries. These effects upon the lungs must be regarded as of a toxic kind; and this specific toxic action is not less obvious on the brain. Its nerve-substance becomes poisonously affected—a condition which seems to constitute one of the most necessary antecedents in the causation of *delirium tremens.*

The effects produced on the medulla oblongata tend to sustain this toxic effect upon the lungs. The brain and the lungs in this respect act and re-act on each other. Death may ensue in various ways, but generally by coma, asphyxia, syncope, exhaustion, or epilepsy.

In the case of habitual spirit-drinkers there is thus constantly going on a temporary stimulus and quickened motion of the blood through the vessels, especially manifested by cerebral, thoracic, and hæmorrhoidal phenomena, followed by a corresponding depression and tendency to stagnation of the blood in the capillaries of all the internal organs, especially in the membranes and the lax areolar tissues of dependent parts.* The essential nature of *delirium tremens* is associated with the loss of cerebral power, evinced especially in the want of control over thoughts, emotions, and muscular action, consequent on the direct influence of the alcoholic poison. Disturbances of function, depression, and debility are the attendant phenomena. The feeble but rapid action of the heart, the tremulous undecided action of the muscles, and the terror-stricken and agitated mental state, betoken the depressed condition of the living functions. The amount of phosphates in the urine, as determined by Dr. Bence Jones, is diminished, while the proportion of the sulphates and of the urea is greatly increased. The nature of the morbid lesions found in such cases has been already indicated. The disease has been variously named the *brain fever of*

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* The influence of alcoholism in causing progressively degeneration of the individual and of the race has been considered in part. I., vol. i.

drunkards, *dipsomania*, or *delirium tremens*, by which latter name it is more frequently known and described in this country.

Symptoms and Course.—The disease has been said to become developed under two sets of circumstances—namely, according as the patient has been continuing his potations, or after he has suddenly abandoned them. But its occurrence under this latter circumstance is now known to be nothing more than a coincidence; and of all the errors in popular acceptation connected with the malady, none is greater than that which affirms the exciting cause of a paroxysm of *delirium tremens* to be a sudden stopping or withdrawal of the accustomed quantity of stimulants. The ceasing to drink depends on the commencement of the disease (GAIRDNER). There are some who are constantly taking small quantities of spirits, and who, although they never get completely intoxicated, yet sometimes exceed considerably their accustomed allowance, and continue to do so for some time. The symptoms of *delirium tremens* generally appear in them from the second to the eighth or ninth day after a protracted debauch, and are by some pathologists divided into three stages. The first stage, according to Dr. Blake, is marked by a peculiar slowness of the pulse, by coldness and clamminess of the hands and feet, by general debility, by nausea and vomiting in the morning, and by frightful dreams at night. Very moderate exertion of body causes the patient to perspire profusely, and anything which suddenly affects his mind throws him into a tremulous agitation. The tongue is tremulous and furred, the hands shake, and he is greatly depressed in spirits, sighs frequently, is anxious about his affairs, and is either restless or watchful. These symptoms last from twenty-four to forty-eight hours.

The second stage commences by a hurried and anxious manner, by great excitability of temper, by a small, accelerated pulse; some heat, perhaps, of the surface of the trunk, but accompanied with the coldness and clamminess of the extremities. The tongue is sometimes clean, but often brown and dry, and the patient delirious, suffering from various mental illusions and alienations. In general the delirium is melancholy, and has reference to his usual occupation and habits, or to some difficulty in his domestic affairs. He sometimes sees flames or hears voices talking to him, or as soon as he shuts his eyes he sees people passing under the bed-clothes. In short, he sees objects and sights in situations in which they are not, and which have no real existence; or betrays the most dreadful alarm at hideous objects which he imagines are threatening him with immediate destruction. Restless and sleepless, he moves his trembling hands horizontally over the bed-clothes, as if seeking for something. In general he is harmless and easily controlled; but in some instances he is violent, mischievous, and requires to be restrained. This stage generally lasts from three or four days to a week, when the third stage commences by the patient falling into a sound sleep and gradually recovering, or a fatal collapse comes on, which finally and shortly closes the scene. Without reference to stages of the disease, the following is a general description of its symptoms:—

There is always more or less derangement in several other functions besides the brain. The patient is generally void of all appetite; or he may be squeamish, and vomit at intervals. Sometimes he is thirsty, and calls loudly for liquor of various kinds; but often he is indifferent to the sensation of thirst. In several instances great aversion, and even dread, of all food and drink has been evinced; and it has been impossible to persuade the patient to partake of either. The tongue is at first covered with moist white, grey, or slate-coloured fur, and when protruded it is tremulous. The bowels are constipated, and less sensible than in the state of health to the action of medicine. When they are acted upon by remedies, the discharges are very dark-coloured, the first generally consistent, the latter liquid, dark, and offensive. There are generally fulness and distension, and not unfrequently tenderness and pain in the epigastric, umbilical, and right hypochondriac regions; and sometimes the two hypochondriac regions give the patient the sensation as if they were drawn tightly together. The skin is bathed about the head and neck with a clammy, unctuous, cold moisture; but elsewhere, and especially at the feet, it is cold, dry, and imperspirable.

The pulse varies from 96 to 110 or 120, sometimes 130; and though sometimes small and oppressed, is often full, voluminous, and throbbing. The carotid and temporal arteries beat most violently; those of the wrist less forcibly; and the anterior and posterior tibial arteries pulsate feebly enough. The action of the heart is in general unusually violent, and the cardiac beat is diffused over the whole chest. The respiration is occasionally panting and irregular, but not otherwise morbid. Restlessness is extreme. The patient is in constant agitation of mind and body; speaks almost incessantly, yet seldom adheres above a minute to one subject, and is constantly changing place and looking for some new object. He cannot sleep, and dreads to be left alone, from the apprehension of spectral visitations. With this restlessness the upper extremities, and especially the hands, are in constant tremulous motion, such that they cannot be kept for two seconds in the same position, nor can the pulse in many cases be accurately numbered at the wrist. Though this tremulous motion of the arms, wrists, and hands is very general, it is not constant; and instances of the mental disorder, agitation, spectral illusions, and sleeplessness have been observed to take place without any such kind of tremors. They are very rarely, almost never, seen in the young or middle-aged to any very great extent, or those whose muscular motions are not otherwise unsteady; and are seldom well marked in first attacks of the disease. Such tremors are principally observed in the cases of confirmed dram-drinkers, whose motions are always unsteady in the morning and early part of the day, until they take a certain proportion of their habitual stimulus (Craigie).

After symptoms of restlessness and sleeplessness have continued for three or four days, the patient may fall into a sound, unbroken slumber, which lasts for some hours. The paroxysm thus works itself out in a definite time in uncomplicated cases; and sleep occurs simply as the natural, the favourable termination of the disorder. It occurs as the *result* of the paroxysm having run its course, and of the nervous system having lapsed into an improved condition, and must not be regarded as the *cause* of those favourable conditions (Reviewer in *Brit. and For. Med.-Chir. Review*, l. c.) On the other hand, the symptoms may pass into a state of *coma vigil*, with constant muttering delirium, *subsultus tendinum*, and picking of the bed-clothes, the pupils become contracted, the muscles of the face and jaw are moved incessantly, and death may ensue from prolonged coma or convulsions. The duration of the paroxysm varies from three, four, or seven days, and a favourable or fatal termination may be looked for in from three to five days.

Diagnosis.—*Delirium Tremens* is to be distinguished from typhus fever, and from paralysis agitans, by the previous history of the case, and by the symptoms.

Prognosis.—It is hardly determined what is the proportion of recoveries to deaths; but unquestionably three persons out of four do well. A paper in *The Indian Annals of Medical Science* for 1855, by Dr. Macpherson, notices the great discrepancy in the statistics of writers on *delirium tremens*, with regard to its frequency in both sexes, and to the mortality of the disease. He attributes this chiefly to a want of due classification. Calmeil states the rate of mortality at 5 *per cent.*, Bougard at 19 *per cent.* The most accurate records to be got are those regarding the British troops at different stations. The late Sir Alexander Tulloch, in his report for 1853, gives the following *per centages* of mortality among them:—

Great Britain, Infantry,	17·6
,, Cavalry,	13·8
Bermuda,	15·0
Canada,	7·94
Gibraltar,	13·6
Malta,	8·8
Nova Scotia,	9·1

A return of admissions and deaths from *delirium tremens* and *ebrietas* in the General Hospital in Calcutta, from 1848 to 1852, and another of admissions and deaths from the same causes in the Medical College Hospital, during 1851, 1852, and 1853, give some important results as follow:—

That *delirium tremens* occurs in women and men in the proportion of one to twenty-five; but that this difference is due to the difference of habits rather than of sex.

That in regard to age the ratio is as follows:—

	Cases.	Deaths.	Per cent of Deaths.
Ages from 20 to 25,	34	4	9·1
,, 25 to 30,	66	16	24·2
,, 30 to 35,	48	11	22·9
,, 35 to 40,	76	7	9·2
,, 40 to 45,	62	6	9·6
,, 45 to 50,	23	4	17·3
,, 50 to 60,	7	—	—
,, 60 to 65,	5	1	—

The greatest mortality is between the ages of twenty-five and forty, which is confirmed by the analysis of another series of sixty-four fatal cases. The *per centage* shows that there is no uniformity in the proportion of deaths to the number of cases.

There is no evidence to show that the season of the year exerts a definite influence on the occurrence of the disease, whereas the mortality very palpably varies with the temperature—it being more than double in the eight hot months as compared with the four cold months.

The apparent cause of death was as follows:—Thirty-three by exhaustion (often with coma); eighteen by coma; eleven by fits (probably apoplectic, called sometimes epileptic); one died on the night stool; one was found dead in bed.

Convulsions occurred in at least twenty of the above cases. One distinct case of paroxysmal *opisthotonos* occurred in a musician, who during the intervals was able to sit up and whistle tunes.

Treatment.—From the nature of the disease as now described, as well as from the dire results of experience, it is now clearly established that the indications for treatment are,—(1.) The elimination of the poison; (2.) The sustenance of the patient during this period. The two most fatal errors which can be committed in the treatment of *delirium tremens* are either to *bleed* the patient or to give him *opiates.* The greatest number of cases of those treated by opiates are apt to terminate by convulsions and coma (MOREHEAD, PEDDIE, LAW, CAHILL, LAYCOCK). If it be true, also, that opium and alcoholic stimulants singly are to be deprecated in the treatment of *delirium tremens, à fortiori* in their combination there is a twofold danger; and alike in tropical as in temperate regions it is a course of treatment attended with much hazard, and which, when systematically followed, is certain of leading to unfortunate results (See Morehead's *Researches on Diseases in India;* also, "Notes on Treatment of Delirium Tremens," by W. Hanbury, 33d Regiment, in *Madras Quarterly Journal,* July, 1863).

The strength must be supported by diet of the most nutritious kind, in a fluid and mild form, such as yoke of eggs, soups, and the like; food should be given in small quantities and often. Beef-tea, spiced soup, and egg-flip, are each to be commended at different periods of the day.

Arrangements should be made so that the patient does not catch cold; and if he continues to digest food, the danger is much diminished. The danger in the first instance is from exhaustion; and careful nursing is above all things necessary, so that protection may be adequate and the food adapted to the state of the digestion, which is always feeble. The disease must be treated as one spontaneously curable; not by withholding remedies, but by using them in strict subordination to good nursing and carefully adjusted diet and regimen (WARE, HOOD, PEDDIE, LAYCOCK, W. T. GAIRDNER). Active specifics for *delirium tremens* appear to be founded on the idea that the disease is one nominally of high mortality. Hence the enormous doses of *digitalis* (JONES) and of *Cayenne pepper* (KINNEAR and LYONS of Dublin); but before resorting to the use of such remedies as *digitalis,* it will at least be judicious practice to adopt such means as are calculated to restore the powers of nature—namely, nutrients and rest; while the stimulus of such a spice as *Cayenne pepper,* given in the soup, on the atonic stomach, will have a favourable influence on absorption. Under the care of my friend, Dr. Lyons, of Dublin, numerous cases of *delirium tremens* have rapidly yielded to *capsicum,* in doses of xx. to xxx. grains in the form of a bolus.

I have tried it in one case only, and its influence in soothing the patient, ending in securing sleep, was certainly remarkable.

Chloral is now a most important and valuable addition to our remedial agents in this disease. Sir Thomas Watson recommends it; and cases of its successful employment are recorded by Messrs. Chapman and Barnes, in the *Med. Times and Gazette* for October 2, 1869, and *Lancet* for November 27, 1869. It produces sound and refreshing sleep, followed by relief to all the symptoms and a complete cure.

The late Dr. Jones, of Jersey, gave as much as half ounce doses of the *tincture of digitalis* till three doses had been taken; and then if excitement were not subdued, nor sleep induced, two fluid drachms were repeated every three or four hours (*Med. Times and Gazette*, Sept. 29, 1860). But Dr. Ringer justly cautions against such remedies; and records two instances in which the patient suddenly fell back dead. The disease no doubt proves suddenly fatal, sometimes independently of any remedy; but the powers of *digitalis* in a tincture are much too uncertain to be relied upon as safe in such enormous doses. *Bromide of potassium* has been found of great use in calming the excitement of delirium and procuring sleep, especially in the earlier stages of the disease, before the delirium has become furious. The dose may be xx. or xxx. grains every two hours. Its good effects are, however, very uncertain (Ringer).

In some cases purgative remedies are indicated from the first. "These cases are known by the flushed, bloated appearance, the very foul tongue, the *mawkish*, peculiar odour of the breath, the fetid discharges from the bowels, and the history of a recent surfeit of eating as well as drinking" (W. T. Gairdner, *Clinical Medicine*, p. 271).

Opium may be administered with safety and advantage only in protracted cases, provided the quantity given in twenty-four hours is never allowed to exceed the full dose which would be considered safe for a healthy person of the age and sex of the patient. Where it appears to be indicated in protracted cases, it ought to be pushed as rapidly as possible for two or three doses, while its effects are carefully watched. Its use must be discontinued for at least a good many hours, as soon as a full maximum amount of ʒiss. to ʒii. of the tincture, in all, has been reached, or even sooner if the pupils have *become at all considerably contracted during its use.* This remedy should always be given in the fluid form, otherwise it is apt to accumulate in the bowels, owing to the weakened state of the digestion; and a laxative, or even a purgative, should be alternated with opium, followed by a bitter tonic, which always operates favourably in lingering cases of nervous and dyspeptic exhaustion. Narcotics are thus only safe in *delirium tremens* when they are given with the object of aiding and seconding the natural cure of the disease, employed in moderate doses, and given only at the later stages. The *heroic* use of them, as heretofore too often advocated even by the most eminent physicians, is now recognized as a treatment which merely substituted *narcotic* poisoning for *alcoholism* or *delirium tremens.*

Chloral will now serve all the purposes intended by opium.

Envelopment in a wet sheet, and then a blanket round the wet sheet, is recommended by Dr. Wilks and Niemeyer as a valuable sedative appliance. As soon as hot vapour, so generated, surrounds the patient, he falls into a quiet sleep (*Med. Times*, Sept. 19, 1868).

PARALYSIS OF THE LOWER LIMBS PRODUCED BY THE USE OF *LATHYRUS SATIVUS.*

LATIN EQ., *Paralysis ex Lathyro;* FRENCH EQ., *Paralysie causée par Lathyrus;* GERMAN EQ., *Lähmung durch Lathyrus;* ITALIAN EQ., *Paralisi da Lathyruz.*

Definition.—*A specific form of paralysis, commencing more or less suddenly by stiffness in the legs about the knees, weakness of the loins, unsteadiness of gait, till at last paralysis becomes confirmed, and the feet are so dragged upon the ground that, with the feet tending to turn inwards, and the knees bent, the great toe scrapes the ground. The disease occurs from the use of the flour of the beans of the* Lathyrus sativus; *and ill-health is apt to occur when the flour of this vetch exceeds one-twelfth part; and if the proportion used as food amounts to one-third, the consequences may be serious.*

Pathology.—Attention has recently been called to this form of paralysis by Dr. Irving, civil surgeon of Allahabad, as extensively prevalent in part of that district. Village after village in Pergunnah Barra, on the right bank of the Jumna, contain many cripples and lame persons, whose paralysis is well known by the natives themselves to be due to their having lived too much upon bread made from the flour of the *Lathyrus sativus;* and they are well aware that it has a peculiar effect on the lower part of the spine (IRVING). From statistics that have been collected on the subject, it is found that a proportion of 3·19 *per cent.* of the population are rendered useless by this disease (Court and Irving in *Indian Annals* for 1857). Different villages are affected in different degrees and proportions. The country where it prevails has the appearance of a vast swamp, and it appears that the *L. sativus* is the vetch which is most extensively cultivated as an article of food. It is common enough in most parts of India, and is frequently sown with wheat or barley, and cut down green as fodder for cattle. The ripe bean is used as food when made into flour, but is generally used with wheat or barley flour; and it is only when it exceeds *one-twelfth* part that it is injurious; and when it exceeds *one-third*, then the specific paralysis sets in. Wheat flour will not grow in the district, therefore the natives are in a great measure left to feed upon this deleterious bean, and suffer in consequence. This form of paralysis is also known in Thibet; and even in Europe it has been known to follow the use of the *L. sativus* as an article of food; and other species of the same genus are occasionally known to render bread poisonous (DON, TAYLOR, LOUDON). Cattle, horses, and birds, when fed on the beans, are said to become paralyzed (SLEEMAN, IRVING). The use of bread made from the flour of the *L. cicera* has been known to establish complete paralysis of the lower extremities in a young and healthy man in a few weeks. Six or seven individuals of the same family, who had been in the habit of eating such bread, suffered more or less from similar symptoms, and one died (Vilmorin, *Ann d'Hyg.*, Avril, 1847, p. 469; Taylor *On Poisons*, p. 536). Further accounts of this vetch as a cause of paralysis may be found in *Indian Annals of Medical Science*, vol. vii., 1861, by the late Dr. Kinloch Kirk, p. 144; also by Dr. Irving, pp. 127 and 501. An incidental reference is also made in Thomson's *Travels in the Western Himalaya* and *Tibet*, p. 391, footnote.

Symptoms and Phenomena.—The paralysis is observed most frequently during the rainy season in India—cold and wet being perhaps an exciting cause, so that the first lameness may be a mixture of palsy and rheumatism. Men who had gone to bed quite well awoke in the morning feeling their legs stiff, especially at the knees, their loins weak, and their gait unsteady. Fever does not seem to attend the accession of the more obvious phenomena; but pain gets worse, and eventually the lower limbs become quite paralyzed. The patient walks with difficulty, the toes turn inwards, the legs waste, and the great toe nail scrapes the ground, till, in persons who go barefooted, the nail has been known to get rubbed down to the quick. Males are said to be more often affected than females; and the *Ryots* are more liable to the disease than the *Zemindars.*

Treatment.—Some cases seem to have been benefited by generous diet, tonics, the use of strychnine, and of blisters to the loins; but nothing is known definitely on the subject, nor have we any records of the morbid state of the spinal marrow in such cases. Of course, the food must be obtained free from all poisonous elements.

THE

SCIENCE AND PRACTICE OF MEDICINE.

PART IV.

MEDICAL GEOGRAPHY; OR, THE GEOGRAPHICAL DISTRIBUTION OF HEALTH AND DISEASE OVER THE GLOBE.*

CHAPTER I.

SCOPE AND AIM OF THIS BRANCH OF SCIENCE.

THIS department of the Science of Medicine treats of the manner, and endeavours to investigate the conditions under which diseases are distributed over the world, or are confined to certain districts. It embraces a consideration of topics which constitute the basis of *Hygiology*, and which are of the greatest importance to Practical Medicine, of the utmost interest to Science, and of inestimable value in Political Economy. It embraces the medical application of the facts of *physical geography*, combined with those of *vital statistics;* and it has been variously named MEDICAL GEOGRAPHY, or NOSO-GEOGRAPHY.

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* Our knowledge on this subject is as yet only beginning to assume a shape; and the limits of this text-book merely permit the most faint outline to be given. To Alexander Keith Johnston, F.R.S.E., the medical profession in this country is indebted for bringing the subject prominently forward, in a communication to the Epidemiological Society of London, published in their *Transactions* for 1856, p. 25, and also in his *Physical Atlas of Natural Phenomena*, where his observations at p. 117 are illustrated by a map. "No scholar out of the domain of medicine has contributed documents more valuable than these to medical literature." That map Mr. Johnston has reduced to a scale suited to this handbook, and thus liberally permits me to use it in illustration. The most important works or monographs which have been published on this subject are Mühry's *Outlines of Noso-Geography*, in two volumes; and Boudin's *Traité de Géographie et de Statistique Médicales, et des Maladies Endémiques*, Paris, 1857, 2 vols. 8vo. A paper on Acclimation by Dr. J. C. Nott, in a work entitled *The Indigenous Races of the Earth;* Sir Alexander Tulloch's *Army Statistics;* Marshall's "Sketch of the Geographical Distribution of Diseases," in the *Edinburgh Medical and Surgical Journal*, vol. xxxviii., p. 330, and vol. xliv., p. 28; Dr. A. S. Thomson's *Thesis on the Influence of Climate on the Health and Mortality of the Inhabitants of the different Regions of the Globe*, Edinburgh, 1837; and Sir Ranald Martin's work on the *Influence of Tropical Climate*, are the sources from which the outline here given has been compiled.

Geographical Distribution of Disease-Realms.—As the physiological conditions of plants and animals vary according to different degrees of latitude, or rather with the different lines of equal temperature and moisture north and south of the equator, so do the pathological characters of diseases differ; and races of men are influenced as to health in proportion as they migrate from the land of their birth. It is the ascertained facts in meteorology and climatology of our globe which will help to explain the geographical limits of particular diseases, and their regulated distribution according to atmospheric temperature and moisture, the density and electricity of the air, and the vegetation with which they are surrounded. Such causes determine some of the laws by which diseases may be geographically distributed; but other concurrent causes must also be taken into account in considering the special diseases of countries. For example, topographical situation, geological nature and elevation of the soil, and state of the vegetation; in short, *physical climate*, generally and properly so called, combined with the habits of the people, their attention to personal hygiene and general sanitary arrangements, all concur to stamp the diseases of certain countries with a special character, and facilitate or retard their propagation.

There are facts which show that certain diseases are so completely under the influence of temperature that they are susceptible of being arranged systematically in zones of geographical distribution. The diseases susceptible of being thus classified are those of a communicable type, which require a certain range of temperature and concurrence of physical conditions for their prevalence and propagation. They are chiefly *yellow fever*, *plague*, *typhus fever*, *typhoid fever*, and *cholera*. The geographical distribution of these diseases into zones, north and south of the equator, appears to be regulated in a great measure by relative degrees of temperature and humidity in the several places where they prevail, in America, Asia, Africa, and Europe. Such diseases as have been mentioned, and whose realms are bounded in a great measure by isothermal lines, are not only associated with locality, with characteristic vegetation, with heat and humidity, but they also follow the physiological habits of the several animal inhabitants of the different latitudes. *Malarious fevers*, *yellow fever*, *plague*, *typhus and typhoid fevers*, have particular climates or zones where each predominates, and beyond the limits of which the disease is rarely if ever perpetuated when imported, unless change of seasons gives rise to a state of climate analogous to that in which the specific disease is known to flourish.

Isothermic Zones bear a most prominent part in relation to the geographical distribution of diseases. They connect the different places on the earth which have the same mean temperature, and which Humboldt was the first to indicate. *Insect-realms* have been similarly indicated by Latreille. The late Professor Edward Forbes described *homozoic belts* of marine life. Cuvier, Blumenbach, Morton, Latham, Prichard, and others, have indicated *homoicephalic zones* or *realms of men;* and, following out these ideas, we have realms of disease defined by Dr. Mühry and Mr. Keith Johnston.

Realms of Disease and Description of the Map.—These zones, belts, or realms of particular types of disease, thus marked out on the globe by these observers, are intimately associated with temperature, and may be generally indicated by the regions of the *tropical*, *temperate*, and *polar* zones, distinguished on the map by the respective colours of *brown*, *green*, and *blue*.

I. The northern limit of the tropical zone unites with the southern limits of the temperate zones, and the lines of union of the two colours on the map (green and brown) indicate Humboldt's mean annual isothermal line of 77° Fahr. or 19° Reaumur. It passes through Cuba and Florida in America; skirts the Cape de Verd Islands to Africa, where, extending beyond the usual limits of the tropics, it passes the northern part of the great desert (Sahara) below Algiers, runs through Egypt, Northern Arabia, and Persia, into China, where it is lost in the Pacific Ocean, below the limits of the northern tropic. The limiting line of this zone ascends somewhat in summer, when the sun is north of the equator; and descends again in winter, when the sun is to the south of it. To the south of the equator the same *isothermal line* (77°) marks the southern limits of the tropical *disease-realm*, where it joins with the northern limits of the *south temperate* zone. It crosses South America near the Amazon district, and approaching southwards in Africa towards the Cape, crosses over and embraces the northern half of Australia. This is the *realm* of tropical diseases, and is coloured *brown* on the map. The class of diseases which characterize this realm are the worst forms of *malarious* (*intermittent* and *remittent*) *fevers*, associated more especially with *dysentery, diarrhœa, malignant cholera, specific yellow fever, hepatic affections, and their results.* Our summer and autumnal affections, characterized by *biliousness, diarrhœa, and bilious, gastric, or typhoid fevers*, approach, by the phenomena they express, the type of the *tropical* diseases. The paludal fevers of this tropical *disease-realm* prevail in their greatest intensity in flat, low-lying countries in the vicinity of marshes, the borders of lakes, shores of rivers and of the sea, and especially where the soil is damp underneath, and of certain geological formation. Sir Ranald Martin has clearly shown how the various soils affect powerfully the temperature and humidity of a place. Argillaceous and ferruginous soils appear in this realm to be especially insalubrious. The malarious fevers of this region make their appearance soon after the setting in of the rainy season, or when overflowed grounds, such as rice-fields, the partially dried-up beds and mouths of rivers, or irrigated plains, begin to dry up and leave portions of the surface of the land, whose subsoil is constantly wet, exposed to the rays of a tropical sun. From such a surface the belief is now universal that a miasm (of the specific nature of which we know nothing) emanates, and acts as a poison upon the blood; and is apt to be developed under similarity of climate, season, and soil, and to produce diseases whose symptoms and course express a constancy and similarity of type. The great centres of these malarious diseases in the different continents are,—(1.) In America—the shores of the Gulf of Mexico, the West India Islands, and the northern portion of South America; (2.) In Asia—India, China, Borneo, Ceylon; (3.) In Africa—the countries round the Gulf of Guinea on the west, Madagascar and Mozambique on the east, Algeria and the shores and islands of the Mediterranean on the north.

The whole mortality within this realm of disease amongst native troops (as estimated by Mr. Johnston) is about 18 per 1,000 for all India, and about 75 per 1,000 for the more unhealthy districts. An average mortality may therefore be stated of about 46 per 1,000 for the realm of disease now under consideration, or 4·6 *per cent.* annually. Active disease and premature death have been such common occurrences in India, that people have been led to believe these results to be the price which we must pay for the tenure of that country. Because disease and premature death were so common, they ceased to arrest public attention, and the causes of them, so frequently pointed out by medical authorities in India, were generally disbelieved by the home authorities when reported to them. At last the *Report of the Royal Commission on the Sanitary State of the Army in India* has disclosed *to the public* the real state of the case, so long known to the intelligent but helpless medical officer. The state of affairs had been unceasingly represented by such men as Jackson, Sir Ranald Martin, Norman Cheevers, Morehead, W. C. Maclean, Macpherson, and many others. It has been promised by the Home Government that the sanitary reforms and improvements indicated by the Commission in their published Report shall be carried out. Several great measures advocated in the Report, and most ably urged by Miss Nightingale in her excellent pamphlet, *How to Live and not Die in India*, have already been carried out. A Commission of Health has been appointed for each Presidency, and that for Bengal has already given public evidence of its zeal. These Commissions have been put into communication with the Barrack and Hospital Improvement Commission at the War Office, which now contains members representing the Indian Government; and by this time the Indian Commissions at the several Presidencies are in possession of all the more recent results of sanitary works and measures which have been of use at home. The condition of the soldier in India is being improved. The worst personal causes of ill-health to which he was in former times exposed have been, or are being, removed. The soldiers are allowed and encouraged to cultivate gardens and to work at trades. The regulation *two drams* of alcoholic spirits have been reduced to one, and that *one dram* is to be diluted with water. "But the main causes of disease in India—want of drainage, want of water-supply for stations and towns, want of proper barracks and hospitals—remain as before in all their primitive perfection."

The maximum intensity of *dysentery*, *yellow fever*, *diarrhœa*, *malarial fevers*, and *affections of the liver*, is observed in those countries which are situated under the line of greatest annual mean temperature—namely, 82° Fahr., which is the assumed equator of heat of the globe. This line of greatest heat also intersects the region of the earth where the greatest amount of water is deposited. Northward to 23° of north latitude, 53 *per cent.* of all the deaths are attributable to these diseases. In latitude 35° north, where the greatest annual temperature is only 77° Fahr., and just where the zone of the tropical disease-realm merges into that of the temperate realm of disease, these diseases cause about 14 *per cent.* of the whole mortality. To the southward, again, in the same latitude—namely, 35°, at the Cape of Good Hope—these diseases yield a loss of only 3 *per cent.* of the whole deaths.

II. The next well-marked *realm* is that in which varied forms of *continued febrile* disease take the place of the *malarious* or *paludal* fever of the torrid zone. The region where diseases of this type prevail embraces *realms* to the north and south of the equator which may be generally described as in the north and south temperate zones. The southern boundary of the northern realm corresponds to the northern boundary of the tropical *disease-realm*, as already indicated by the isothermal line of 77°. In a northern direction it extends to latitude 60° north, and includes the British Isles, Norway, and Sweden; while in America its northern limit includes part of Nova Scotia and Newfoundland. Its boundary line to the north passes from 60° north latitude in a south-eastern direction on both continents, till it gradually declines towards the borders of Asiatic Russia in the Old World, and to the district between Boston and Philadelphia on the eastern shores of the New, and corresponds nearly with the annual isotherm of 41°. In the southern hemisphere it embraces the southern two-thirds of South America, the district of the Cape of Good Hope, and the southern half of Australia. It embraces the most healthy regions of the world, in which the prevailing causes of ill-health are mostly due to the *condensation of people in towns*, and the insalubrious and depressing conditions which necessarily arise from that cause. Emanations from nuisances tend to accumulate, where mechanical and chemical arrangements of a sanitary kind do not remove the concentrated impurities. But where proper sanitary measures exist for the supply of pure water and free air, the artificial disadvantages inseparable from town life may be greatly modified in their influences, so much so, that in our country (the least unhealthy of all) the mortality may range from 1·7 to 3·6 *per cent.* annually (Reg.-Gen. *Rep.* for 1853, p. 15). Nearly every type of disease has a representative in this realm; but, generally speaking, the general and mixed diseases are the classes which furnish the greatest mortality. *Typhus* and *typhoid fever* prevail between the parallels of 44° and 60° in Western Europe; *yellow fever* has prevailed on the southern shores of Spain, the north-west coast of France, and Northern Italy; *intermittent fever* in the Netherlands, Sweden, and Central Italy, and generally where marshy undrained lands exist. *Small-pox* especially prevails where vaccination has not checked its ravages; *leprosy* and *elephantiasis* prevail in Scandinavia; *pellagra* in Italy, France, and Spain; *plica polonica* in Poland and Tartary; and *consumption* and *rheumatism* everywhere. While, therefore, this temperate zone embraces, on the one hand, the extremes of temperature of the torrid and the frigid zones in the seasons of summer and winter, it is also observed to have representatives of the types of disease which prevail in both these realms; and according as we approach its northern or its southern limits we find that the characters of disease become so modified and their types are found so to mingle together on the confines of these disease-realms that the *continued* type of febrile diseases peculiar to the temperate zones tend, as we approach the tropics, to merge into and to participate in those irregular intermittent characters which are peculiar to the type of the tropical malarious districts. It is only to a limited extent, however, that such modifications are found to occur; and there are some diseases which rarely tend to pass their geographical boundaries unless especially favoured by *tropical* identity of climate on the one hand, or *temperate* identity on the other. Under the former circumstance yellow fever has been met with at Gibraltar, Cadiz, Lisbon, St. Nazaire, and even so far north as Plymouth Sound and Southampton Water; and it has also extended in America southwards beyond its usual limits; but so soon as the temperature falls below 55° Fahr., the importation of yellow fever into *this zone*, and its propagation or persistent existence in it, becomes impossible. Diarrhœa, also, and dysentery are apt to prevail during the summer and autumn months; while under bad diet, defective ventilation, and generally imperfect sanitary measures, *contagious typhus fever* is apt to be propagated in crowded localities of towns, and in huts, hospitals, and barracks. Typhus and typhoid fevers seem to have their special habitation in this zone, especially between 30° and 40° of north latitude, and likewise the true glandular plague; and which may be occasionally propagated beyond these limits by a secondary and specific poison, generated from human bodies and propagated by human intercourse. The zone, therefore, which we inhabit, while it may be said to exhibit the greatest variety of disease-processes and types of disease, may, nevertheless, be considered a highly favoured district of the globe, inasmuch as the development and progress of disease and of epidemics are much more manageable, less intense, and on the whole less fatal than similar types of disease in the tropical realm, or even than they were centuries ago. Under proper sanitary regulations *typhus fever* and many other diseases in this country are greatly under control. *Cholera* may be said to be the only scourge which occasionally, as an epidemic, becomes *rapidly* destructive to life. But, by scientifically directed sanitary measures, and the increasing experience of an enlightened age, the spread of the *plague* in this country has ceased. In this realm "history records the existence of diseases of former days now happily unknown; while, on the other hand, scourges unknown to ancient times devastate modern populations" (PLINY, SYDENHAM, BOUDIN). In the sixteenth century the Oriental plague, "like the destroying angel, spread its wings on the blast," and added to the miseries of that stormy and bloody time. Our physician-poet tells us that when the fate of England was about to be decided on "Bosworth's purple field," that plague, of most gigantic arm,

> "Rushed as a storm o'er half the astonished isle,
> And strewed with sudden carcases the land."

Then, as now, our standing armies and bodies of men congregated together especially suffered from pestilential disease. It so thinned the ranks of Henry's victorious army, that few were left to see the conqueror crowned with Richard's diadem. Asiatic cholera may now be said to have taken the place of the Oriental plague, and it is often no less destructive and important in its results—sternly demonstrating "how one dread year performs the work of ages, when the pestilence mocks in his fury the slow hand of time."

III. To the northward of this temperate zone, in the northern hemisphere, there is another disease-realm, where *catarrhal affections*, *influenza, scurvy, erysipelas, diseases of the skin* and *digestive organs*, and *various* CONSTITUTIONAL *affections* more especially prevail, to the exclusion of *malarious febrile diseases*, except on very rare occasions in summer, and when *cholera* and *dysentery* may prevail. This *disease-realm*, in the polar isothermal zone, rejoices in a climate directly opposite to that of the tropical zone. Its southern limits are the northern boundaries of the previously defined region—namely, the isothermal line of 41° Fahr., or 2° or 3° of Reaumur. Commencing on the western coasts of North America above Sitka, it extends southward across the district of the Canadian lakes, sinking south and east into Canada and Newfoundland to Boston and New York or Philadelphia. Thence it continues north-west nearly on 41° Fahr. annual temperature, when it crosses to Europe, and ascends till near the borders of Iceland, whence it sinks towards Norway and Sweden, and, running above St. Petersburg and Moscow, crosses to Siberia. Iceland being the best-known locality of this district, Mr. Johnston takes its peculiar diseases as the representatives of this realm. Every year, in spring or in early summer, it is visited by *catarrh;* and at short intervals it is visited by *catarrhal fever* —a *true influenza*, which has usually a great effect on the mortality. The majority of Icelanders are said to die before the age of fifty, from *asthmatic* or *catarrhal affections*, which are also prevalent in Greenland and Labrador.

Dr. Lawson has attempted to establish (see vol. i., "*Epidemic, endemic*, and *pandemic* influences") the occurrence between 1817 and 1836 of a series of *oscillations of febrile diseases*, following each other over the world with amazing regularity. These he attributes to a cause or influence which, from its extent and progressive character, he names a "pandemic wave," to distinguish the influence from that usually understood as "epidemic," referring to a single form of disease, affecting a limited space. Under the influence of this pandemic wave he believes that there is a constant progressive tendency to the development of various endemic febrile diseases in the Atlantic and western parts of the Indian Ocean, from south or south-east to north or north-west.

Many of the facts and data on which he founds are, however, not sufficiently trustworthy to rest a judgment upon, and in some cases are susceptible of a totally different interpretation from that which Dr. Lawson has assigned to them. Although, therefore, his theory is "not proven," yet the expression of it is calculated to do good, by drawing attention to this view of the subject.

CHAPTER II.

ON MALARIA AND PLACES KNOWN AS MALARIOUS.

GEOGRAPHICAL facts, collected by medical writers from Hippocrates downwards, show that every country is unhealthy in proportion to the quantity of marsh, or of undrained alluvial soil it contains, the inhabitants of such districts dying often in the ratio of 1 in 20, instead of 1 in 38—the average mortality in healthy countries. The connection of a given class of disease—represented by remittent and intermittent fever—with marshy districts is now distinctly established and generally recognized.

Ancient Rome was once the seat of so many fatal epidemics that the Romans erected a temple to the goddess Febris. These epidemics were known to arise from the great masses of water poured down from the Palatine, Aventine, and Tarpeian hills becoming stagnant in the plains below, and converting them into swamps and marshes. The elder Tarquin ordered them to be drained, and led their waters by means of sewers to the Tiber. These subterraneous conduits ramified in every direction under the city, and were of such considerable height and breadth that Pliny terms them "*operum omnium dictu maximum suffossis montibus atque urbe pensili subterque navigata.*" This system of drainage, which was continued as late as the Cæsars, rendered Rome proportionably healthy, and the seat of a larger population than has since perhaps been collected within the walls of any city. On the invasion of the Goths, however, the public buildings were destroyed, the embankments of the Tiber broken down, the aqueducts laid in ruins, the sewers obstructed and filled up, and the whole country being now again overflowed, Rome has once more become the seat of an almost annual paludal fever, as in the times of her earliest foundation. The insalubrity of the Pontine Marshes, past or present, is notorious. Three hundred years, however, before the Christian era, Appius Cladius drained them, by making canals, building bridges, and by constructing that magnificent road, portions of which still remain, and still bear his name. On the invasion of Italy by Theodoric, Cæcilius Decius gave a free course to the waters in the neighbourhood of Rome, and the re-establishment of these immense marshes was one of the many disasters which resulted from the attacks of the Goths on Italy. Their present state is such that the Tuscan portion of Maremme, and indeed the whole of that district, may be said in summer to be absolutely depopulated, not a single house retaining an inhabitant, except the guard-houses, with a few soldiers and custom-house officers; and these are relieved twice or thrice during the summer, with the Maremme fever almost invariably upon them.

Many districts in the East and West Indies, in Pennsylvania, and Continental Europe, are known to be active in the evolution of *malarious influences.* Such places are generally the deltas, marshy banks, and embouchures of rivers, in the plains extending from the bases of mountain ranges; partially inundated and irrigated lands, or such as are traversed by percolating streams or canals in wooded districts, termed *jungles;* the seaboard, especially where there is jungle or salt marsh; and in the Bengal district the stations of Calcutta, Chinsurah, and Berhampore are highly malarious (MARTIN). The woods and marshes of the Sunderbunds, covering a superficies of more than 20,000 miles, and extending 180 miles south and east of Calcutta, composed of marshy land, covered with forest and underwood, together with the numerous embouchures of the Ganges, are well-known unhealthy districts. The partially dried-up marshes and beds of rivers have too often been fatal to our armies when imprudently and ignorantly encamped in their vicinity. In 1810 the plains of Spain, along the course of the Guadiano, with its "lines of detached pools" and its ravines always "half-dried," could tell of a fever-striken army. The pages of history also remind us how our British soldiers perished on the low, dry-looking, sandy plains of Walcheren and of Rosendaal in 1794 and 1809-10. Our last war with Russia, during the campaign in Bulgaria, and especially at Varna in 1854, furnishes a no less melancholy record of the sufferings of British troops, and the persistent pernicious influence of a residence in malarious districts.

In China we know of the miasmatic nature of the deltas of the Blue and the Yellow Rivers. In Africa we know of the Zais, the Orange, and the Zambesa as unhealthy rivers. In America the Amazon, the Orinoco, and the Rio-del-Norte are similarly deleterious. In England we have the fens of Norfolk and Lincolnshire still a source of disease; in short, there is hardly a country which has not its marshy lands, so that abundance of work exists for chief commissioners of sewers to direct for good, where such "heads" of "*bodies*" exist; and the extent of disease proceeding from marshes has been shown in many places in Italy, Sicily, and Greece to be so great as to occasion more than two-thirds of the average mortality.

Removal and Neutralization of Malaria.—Of towns that have been drained and remain healthy there are many examples in ancient and modern history. Hippocrates tells us that the city of Abydos had been several times depopulated by fever; but the adjoining marshes having been drained, it became healthy. London, in the time of Sydenham, was infested with epidemic intermittent fever and dysentery, the mortality from the former alone averaging, in a comparatively small population, from one to two thousand persons annually. In the present day, owing to the formation of sewers and a general system of drainage, a case of ague contracted in London is hardly known. Many other towns, both of this country and of France, as Portsmouth, Rochefort, and Bordeaux, from being the constant seat of paludal fevers, have been, from the same causes, rendered in like manner comparatively healthy. Dr. Wood, of Pennsylvania, relates an interesting fact regarding the neutralization of miasmatic effluvia. He tells us they are in some way rendered innocuous by the air of large cities. This fact is notorious in relation to the city of Rome; and it is abundantly confirmed in the larger towns of the United States, in the neighbourhood of which these diseases have prevailed.

Nature of the Noxious Agent.—Thus the intimate connection between marshy districts and certain forms of disease is established by a great amount of direct and indirect testimony; but two questions still remain—namely, What is the nature of the noxious agent of malarious districts? and what circumstances are necessary to its formation or extrication? It seems certain that the deleterious agent is neither heat alone nor moisture alone, nor any known gas extricated from the marsh. It cannot be *heat* alone, for many of the hottest parts of the West Indies are free from fever. It cannot be *moisture* alone, for no persons enjoy better health than the crews of clean ships at sea, even when cruising in tropical climates, as long as they have no communication with the land. While *carbonic acid, azote, oxygen,* or *carbureted hydrogen,* the gases collected by stirring the bottom of marshes, have all been inspired without producing any disease similar to paludal fever, it seems to follow almost as a necessary consequence that the remote cause must be a *miasm, poison,* or *malaria,* whose presence is solely detected by its action on the human body; and two hypotheses have been imagined to account for its origin:—the one, that it is a product of vegetable decomposition—the other, that it is an exhalation from the earth, favoured by the conditions of marshiness. A theory, often hinted at, is beginning to find expression—namely, that very minute fungi of rapid growth, like *smut, rust, mildew,* and the like, are active agents in the propagation of malaria. Deputy Inspector-General Reid has shown this in regard to the recent epidemic in the Mauritius, and Staff-Assistant-Surgeon Massey in regard to Ceylon (see *Army Med. Report* for 1867).

The general evidence in favour of vegetable decomposition (and therefore growth) being the remote cause is, that all countries are for the most part free from paludal diseases while the crops are growing, and only become unhealthy after the harvest, when large quantities of vegetable matters are left on the ground at the time the rain begins to fall. Marshes are in general healthy till the summer's sun, or other cause, has diminished their waters, and bared a greater or less portion of their bed. The part thus exposed almost always contains a large portion of vegetable matters, which, running into rapid decomposition, generates other vegetable growths of a fungoid nature especially, which may be or may convey the poison which gives origin to this class of disease. It is during the periods of the year when the drying process is in greatest activity that unhealthiness prevails with greatest severity in the East Indies—namely, before the commencement and after the termination of the rainy season. The particular evidence of vegetable decomposition being the source of the poison is of the following nature: —Lancisi gives the history of an epidemic which for several summers infested, and almost depopulated, the ancient town of Urbs Vetus, situated on an elevated and salubrious part of Etruria, and which was traced to the circumstance of the peasants steeping their flax in some stagnant water in the neighbourhood of the town. This practice was therefore prohibited in 1705, and the epidemic ceased to appear. The steeping of flax being productive of paludal fever is a fact the knowledge of which is not limited to Italy; for the ancient as well as the new "*coutumes*" of almost all the provinces of France have proscribed the steeping of flax, "la rouissage," even in running waters, from the fear of infection. In the Netherlands the same belief has prevailed. In July, 1627, the King of Spain passed an ordinance prohibiting the steeping of flax in the streams and canals of Flanders. The prohibition may be explained on another ground—namely, because the flax poisons the water and kills the fish. In Ireland, and perhaps also elsewhere, steeping flax in a running stream is therefore forbidden by law.

The experience of the indigo-planter is to the same effect. In India, after the colouring matter has been extracted from the indigo plant, it was formerly the custom to throw the detritus into large heaps or masses in the immediate neighbourhood of the works, and which, at the end of three or four years, becomes manure of an excellent quality. It was found, however, that these heaps, wetted from time to time by the heavy rains, and afterwards heated by the rays of a burning sun, rapidly decomposed, and at length emitted *miasmata*, which produced all the effects of those extricated from the marsh. The workmen who lived near, and more especially those to leeward of these masses, were found to be very commonly attacked by fever, chiefly of the remittent type, and similar to those which prevail in the paludal districts of that country. This consequence is now so well established that the most intelligent indigo-planters no longer allow these heaps to be formed either near the works or in the immediate neighbourhood of the cottages of their workmen.

Ships also afford additional evidence of the truth of the hypothesis of vegetable decomposition and growth being the remote cause of malaria.

All intertropical regions, where the nature of the locality admits only of a rice cultivation, are well known to be unhealthy.

These facts render it highly probable that the noxious agent must be a product of vegetable development, growth, and propagation, evolved on the soil, and moving in the lower regions of the atmosphere. No analysis of the air has yet disclosed any immediate chemical principle to which the unhealthy influence of miasms may be ascribed. The atmospheric air collected at the embouchure of the Valtelline—a country where it is impossible to sleep without being attacked with fever—gives, on analysis, the same chemical constituent parts and proportions of gases as that collected at the summit of the Alps, or in the narrowest streets in London. But such places, on the other hand, give evidence of abundant minute fungoid vegetation, which may be active poisons, or active agents in propagation of malaria.

If we consider the paludal poison to be a product of vegetable decomposition, or of vegetable growth, it follows that heat and moisture, quantity of vegetable matter, and nature of the soil, though not the essential agents, must have a sensible influence on its formation, must vary its intensity or quantity, and also must limit paludal diseases to particular localities, seasons, and latitudes. A certain temperature, for example, under certain conditions as to moisture, is evidently necessary to its extrication and development.

It is certain also that a given quantity of moisture is as necessary to vegetable decomposition or growth as a given temperature, and that the extrication of the paludal poison will be most abundant from that soil which contains no more moisture than is necessary for that process; for an excess in quantity, by dividing and separating the particles, and by preventing the access of atmospheric air, will either retard or altogether put a stop to putrescency. Hence in some countries frequent and heavy rains will render marsh fevers prevalent, by saturating the whole of the open country; while privation of rain will in others produce exactly the same effect in other instances, merely by diminishing the superfluous quantity of water. Thus, in the West Indies, an uncommonly rainy season seldom fails, in the perfectly dry and well-cleared Island of Barbadoes, to induce for a time general sickness; while at Trinidad, whose central portions are described as a sea of swamp, and where it rains nine months in the year, an excess of moisture is a preservative from sickness; for should at any time rains fall only eight months in the year instead of nine, the swamps become dry and bared to the sun, and remittent fevers of the worst kind are sure to make their appearance. The same result follows on the subsiding of the waters of rivers that have overflowed their banks, as those of the Nile, the Rhone, the Danube, the Tigris, the Ganges, and many others.

It is evident from these data that the swamp, on its approach to dryness, is the source of disease and death; while an excess of rain has a preservative power so long as moisture is in excess. On the contrary, on the rich and dry plains, and even on the hills of tropical countries, rain is the cause not only of vegetable decomposition, but also of disease; while absence of rain tends to preserve health.

In estimating, however, the dryness of a country, its superficial appearance is often deceitful. In the years 1748 and 1794 the summers were dry, and our troops took up the encampments of Rosendaal and Ousterhout in South Holland. The soil in both places is a level plain of sand, with a perfectly dry surface; and where no other vegetation existed, or could exist, but a few stunted heath plants; yet in both years fever became epidemic among the troops in each place. On digging for water the cause was discovered, for the soil was found to be saturated with water to within a few inches of the surface. It is probable, therefore, that this country was originally formed of vegetable and other detritus, brought down by the Rhine and the Waall, and afterwards covered with sand thrown up by the sea, and which, heated by the summer's sun, became the powerful cause of the extrication of marsh miasmata. From the exceeding malignity of the salt marshes, it has been supposed that a mixture of salt and fresh water renders a marsh more pernicious than either of them alone. It has been found, however, that on coasts where these marshes have been kept up to one uniform level by means of flood-gates, the surrounding country is healthy; and it has therefore been inferred that the sickness produced is a consequence of the perpetual alteration of the level of the waters of the marsh, and not owing to the admixture of sea and spring water.

It is probably owing to a great excess of temperature that rocky countries, as Gibraltar and the Ionian Islands, are so often and so severely attacked with malarious fever. It is on the summits of these rocks that springs arise. The slightest frost produces fissures, into which fungi, as "*mould*," and other vegetable matters insinuate themselves, while the bare rock becomes heated to an intense degree. Humboldt, on ascending the Orinoco, found the station at the great fall depopulated by fever, which the natives attributed to the bare rocks of the rapids. He determined the heat of these rocks to be 118·4° Fahr., while the temperature of the air immediately around was only 78·8° Fahr. Again, the rock of Gibraltar is known to be percolated with water, so that we can hardly conceive a more pestilential focus of disease when the causes necessary to the formation of miasm or fungi are combined. The existence of paludal fever in dry and rocky districts, therefore, although it may appear extraordinary and unexpected, is not necessarily an exception to the general law of paludal diseases being generated by miasmata, the result of vegetable decomposition or of vegetable development. In many hot climates the most deadly sites for encampments have been the dried-up beds of rivers, or their immediate vicinities (MARTIN).

These facts seem, therefore, unquestionably to prove that heat and moisture, though not the primary cause of paludal disease, are conditions essentially connected with the development of vegetable growths, like fungi, of the noxious miasmata, and consequently furnish a strong additional argument in favour of the hypothesis of vegetable decomposition generating the remote cause which produces or propagates some miasmatic diseases. It is certain, however, even when the conditions of heat, moisture, and vegetable matter most abound, that paludal diseases do not always assume their severest forms; and there seems reason to believe that differences of geological formation, by favouring or otherwise influencing vegetable putrefaction and growth, may variously affect the health of countries similarly situated in other respects.

It is perfectly well known that different soils radiate heat with different degrees of intensity, and consequently are, under the same circumstances, of different temperatures, having very different powers of attracting moisture; and possibly, also, they may have other and more direct properties favourable to the generation of the paludal miasm. Nothing, for instance, is better determined in husbandry than that the *carbonate of lime,* mixed with the ordinary matters of a compost, greatly forwards the processes of putrefaction, so that the mass thus prepared is fit in a much shorter time for the purposes of manure.

There are some soils peculiarly favourable to the decomposition and growth of vegetable matters, and consequently to the more abundant extrication of marsh miasmata; and it is remarkable that those countries most celebrated for paludal fevers have been found similar in their geological formation to each other, and to those artificial conditions which most favour rapid vegetable decomposition and growth.

To predicate all the facts connected with paludal diseases is not yet possible; for the variations of atmospheric temperature, the changes in the quantity and nature of the electric fluid, the quantity of water, the nature of the soil, the amount and character of the vegetable matters and their growth, form a problem extremely complicated, and one whose smallest variation as to quantity or time may occasion marked differences in the result. As a general rule, however, it may be stated, that in no climate do paludal fevers prevail to an equal degree all the year round. In the winter, much of the vegetable matter has already undergone decomposition and further growth, while the dryness of the season and the diminished temperature are little favourable to its further development. When the spring, however, arrives, and the rain falls, and the heat of the sun increases, the earth again evolves a miasm of mitigated intensity. In summer the products of vegetable decomposition are used up in affording nourishment to the growth of many vegetable forms; and this season, like the winter, is in general healthy. But in the autumn, and after the harvest has been gathered, when the ground is covered with vegetable debris, when the rain falls in torrents now and again, and when the solar heat has acquired its greatest intensity, all the conditions for the greatest amount of growth of vegetable matter, of moderate moisture, and of highest temperature, change and are united; so that the season which realizes the hopes of the husbandman is also the period of pestilence and of his greatest danger. There are two other facts, also, which are too prominent to be mistaken. The one is, that the miasmata vary greatly in intensity in different countries, and also in different parts of the same country: again, the diseases they produce, though annually *endemic* in given districts, yet become, in certain years, and from the action of causes not yet determined, *epidemic.*

In the same countries, also, it is determined that difference of altitude is equivalent to difference of latitude; and, as a general law, it may be stated that in the Antilles, on the continent of America, from Boston to Rio de Janeiro, and also on the continents of Asia and Africa, while in the low country severe remittent or yellow fever prevails, still in the higher country, though immediately contiguous, the type is changed to intermittent and mild remittent. The interesting fact stated by Humboldt, that the *vomito prieto* never appears on the table-lands of Mexico, is strictly in accordance with the observations made in every other equatorial part of the world at a similar elevation above the level of the sea. The circumstance of intermittents passing into remittents, and remittents into malarious yellow fever, and, conversely, of remitting and malarious yellow fever often terminating in intermittent—facts observed not only in the East and West Indies, but on the continents of America and of Africa—demonstrates a unity of cause as firmly as the best established facts in medicine.

That paludal diseases, like many diseases produced by morbid poisons, are annually endemic, and only occasionally epidemic, is unquestionable. A few years ago intermittent fever was epidemic in particular districts in this country; but of late years the cases of ague have been comparatively rare. In Demerara it has been observed that malarious yellow fever is epidemic about every seventh year. At Gibraltar, although sporadic cases of paludal fever occur annually, still malarious yellow fever is only occasionally epidemic, but so irregularly that it assumed that character in 1804, then in 1810, again in 1813, and in 1814; and from that period the garrison suffered no similar visitation till 1828. The physical causes on which this greater virulence and greater spread of the disease depend are not determined. In temperate climates it has been observed that paludal fevers have been most prevalent when a hot summer has succeeded a wet spring.

Infecting Distance of Miasmata.—As a general law, the danger of infection is in proportion to the proximity to a marsh. But there are many disturbing causes which produce remarkable exceptions to this law. These disturbing causes are, the extent of surface which generates the miasmata, their intensity, the direction of the wind, its force, the season of the year, the time of the day, and the attracting influence of the surface over which the miasmata pass.

The Altitudinal Range.—The Monte Mario, which adjoins Rome, is, according to Breslack, about 165 yards perpendicular height above the Pontine Marshes, and is extremely unhealthy. Trivoli, which is about 230 yards above the level of the same marshes, is infinitely more salubrious; while at Serre, 340 yards perpendicular height, the inhabitants enjoy an entire exemption from the paludal diseases which prevail below. In Italy it is estimated that an altitude of 1,400 to 1,600 feet is necessary to assure an exemption from paludal disease; but in the West Indies, where the poison is of so much greater intensity than in Italy, it is estimated that an elevation of 2,000 to 2,500 feet is necessary to give a similar immunity. The different latitudes may account for this.

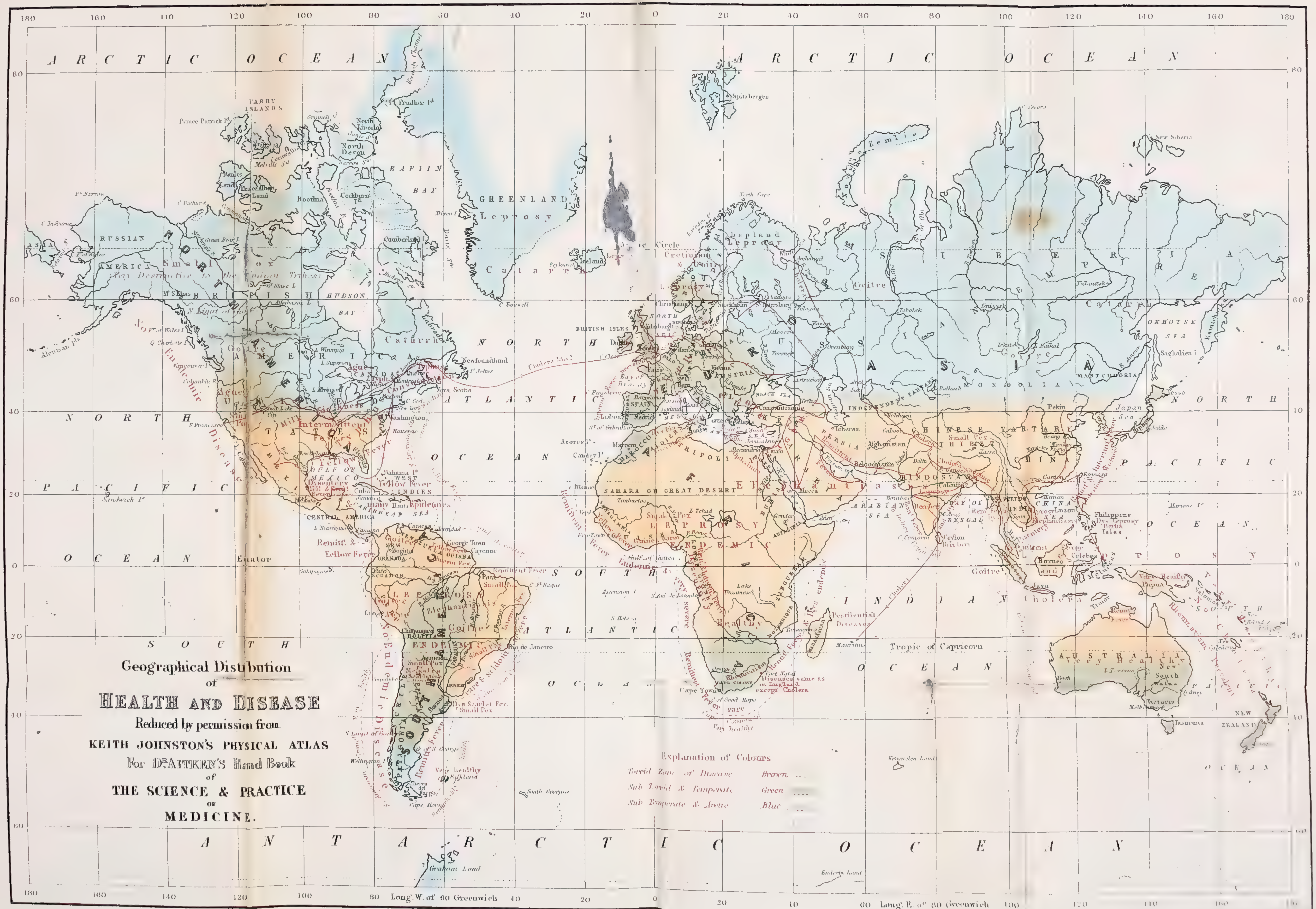
In towns partially freed from marsh miasmata by extensive drainage, the difference of a few feet perpendicular height makes an almost inconceivable difference in the liability of persons to paludal disease. The barracks of Spanish Town, the capital of Jamaica, for instance, consist of two storeys, or of a ground floor and of a first floor; but it being found that two men were taken ill on the ground floor for one on the first floor, it was at length ordered that the ground floor should be no longer occupied. Dr. Cullen remarked a similar result at Portobello; Dr. Fergusson in St. Domingo; and Sir Gilbert Blane in the expedition to Walcheren. This law is so well understood in the West Indies that in Demerara, and in many other parts, the houses are built on dwarf columns, after the manner of corn stacks, in order that a stratum of air may be interposed between the house and the ground. In Rome, and in other towns of Italy, it is also so well known that the lower rooms of the houses are abandoned, the family occupying the upper rooms, as affording a greater protection from the paludal poison.

The Lateral or Horizontal Spread of marsh miasmata is a problem still more difficult than that of the altitudinal range. The least complicated cases are those when water alone intervenes between the marsh and the recipient. In the year 1746-47, while our troops lay in Zealand, the sickness was so great among four battalions quartered there that some of those corps had hardly 100 men fit for duty, or less than a seventh part of a battalion. In one corps, the Royals, only four men escaped. At the time, however, of this remarkable prevalence of fever on shore, Commodore Mitchell's squadron lay at anchor between South Beveland and the Island of Walcheren, and the fever raged at both places; but, nevertheless, in the midst of all the sickness that reigned around, the seamen were neither affected with fever nor flux, but continued to enjoy perfect health. These observations of Sir John Pringle were fully confirmed by those of Sir Gilbert Blane during the last disastrous expedition to Walcheren. "I had," says this physician, "the opportunity of observing the extent to which this noxious exhalation extended, which was found to be less than was generally known. Not only the crews of the ships in the Road of Flushing were entirely free from this epidemic, but also the crew of the guard-ship, which was stationed in the narrow channel between this island and Beveland.

The width of this channel is about 6,000 feet; yet, though some of the ships lay nearer to one shore than the other, there was no instance of any of the men or officers being taken ill with the same disorder as that with which the troops on shore were affected." It appears, therefore, that in Europe the horizontal spread of marsh miasmata over fresh water is less than 3,000 feet. With respect to the spread of the miasmata over salt water, Sir Gilbert Blane wrote that in tropical climates ships at a distance of 3,000 feet from a swampy shore—a distance to which the miasmata did not extend in Zealand—and even farther, were affected with the noxious exhalations. Dr. John Hunter considered a few miles to be a necessary interval for a ship lying to leeward of a swamp, in order to insure a complete exemption from the disease. When, however, the swamp or other source of the poison is of small extent, a much less space is sufficient to assure an exemption. In the epidemic on the coast of Spain, the fisherman living with his family on board his boat has been rarely attacked, though lying at anchor close in shore. Also, during the late epidemics at Gibraltar, it was not unusual for the richer inhabitants to hire a Moorish vessel and to live on board in the bay; and there was scarcely an instance of those persons having been affected, though keeping up a free communication during the day, either directly or indirectly, with the town.

The extent to which the marsh miasmata may spread from its source over land in a horizontal direction is a much more complicated question. The effect of trees in intercepting the paludal poison is remarkable, and appears to have been known to the ancients, who are supposed to have surrounded their temples with groves, on account of their protecting influence. Pope Benedict XIV. ordered a wood to be cut down which separated Villatri from the Pontine Marshes, and for many following years there raged throughout the whole country, and in places never before attacked, a most severe and fatal fever. The same effects were produced from a similar circumstance in the environs of Campo Santo. In the West Indies it is quite wonderful how near the marsh the planter, provided he is protected by trees, will venture to place his habitation.

Different soils also affect the transmission of the paludal poison. The spot, for instance, on which the new National Dock and Arsenal are built was a marsh of about 700 acres, and on either side of it are the villages of Greenhithe and of Northfleet. The peculiarity here is, that the inhabitants of these villages rarely suffer from intermittent fever, whilst those on the hills beyond are greatly afflicted with that disease. Dr. Maton mentions a similar fact in the neighbourhood of Weymouth, and the same circumstance is observed in the neighbourhood of Little Hampton and the marshy districts in Sussex.



Geographical Distribution
of
HEALTH AND DISEASE
Reduced by permission from
KEITH JOHNSTON'S PHYSICAL ATLAS
For Dr. AITKEN'S Hand Book
of
THE SCIENCE & PRACTICE
of
MEDICINE.

Explanation of Colours

Torrid Zone of Disease	Brown
Sub Torrid & Temperate	Green
Sub Temperate & Arctic	Blue

CHAPTER III.

ACCLIMATION, OR THE INFLUENCE OF CLIMATE ON MAN.

Definition.—*Acclimation consists in "a profound change in the organism, produced by a prolonged sojourn in a place whose climate is widely different from that to which one is accustomed, and which has the effect of rendering the individual who has been subjected to it similar in many respects to the natives of the country* (indigènes) *which he has adopted"* (ROCHOUX). *The process takes place to a certain extent so far as some individuals are concerned; but the ability to become acclimated is not possessed to the same extent by all nations.*

It is found that the *white races* reach their highest physical and intellectual development, as well as most perfect health and greatest average duration of life, above 40° of latitude in the western, and 45° in the eastern hemispheres; and whenever they emigrate many degrees below these lines they begin to deteriorate, from increased temperature, either alone or combined with other morbific influences, incident not less to change of climate than to habits of life—to evils social and civil, but more or less remediable by attending to the common principles of sanitary science.

The laws of climate show that each race of mankind has its prescribed salubrious limits. All of them seem to possess a certain degree of constitutional pliability, by which they are able to bear to a certain extent great changes of temperature and latitude; and those races that are indigenous to temperate climates support best the extremes of other latitudes. The inhabitants of the arctic regions, and of the tropics, have a certain pliancy of constitution; but while the inhabitants of the middle latitudes may emigrate 30° south or 30° north with comparative impunity, the Esquimaux in the one extreme, or the Negro, Hindoo, or Malay in the other, have no power to withstand the vicissitudes of climate encountered in traversing the 70° of latitude between Greenland and the equator. The fair races of Northern Europe below the arctic zone find Jamaica, Louisiana, and India, to be extreme climates: and they and their descendants are no longer to be recognized after a prolonged residence there. When an Englishman is placed in the most beautiful part of Bengal or Jamaica, where malaria does not exist, although he may be subjected to no attack of acute disease, but may live with a tolerable degree of health his threescore years and ten, he nevertheless ceases to be the same healthy individual he once was; and, moreover, his descendants degenerate. He complains bitterly of the heat, and becomes tanned; his plump, plethoric frame becomes attenuated; his blood loses fibrine and red globules; both mind and body become sluggish; grey hairs and other marks show that age has come on prematurely—the man of forty looks fifty years old; the average duration of life is shortened (as shown in life insurance tables); and the race in time would be exterminated if cut off from fresh supplies of emigrants from the home country. The European in the Antilles *struggles with existence*—a prey to fever and dysentery. He is unequal to all labour, becomes wasted and wan, and finally perishes. His decay is premature; and, but for the constant influx of fresh European blood, he becomes rapidly extinct as a race. The European inhabitants of Jamaica, of Cuba, of Hispaniola, of the Windward and Leeward Islands, have made no progress since their first establishment there. They cannot execute labour—hence the necessity for preserving and maintaining the black population. Their offspring are pale, wan, and sickly, and in half a century cease to be productive (MORELL, p. 107). Our army medical historians tell that our troops *do not become* acclimated in India. Length of residence in a distant land affords no immunity from the diseases of its climate, which act with redoubled energy on the stranger from the temperate zones. On the contrary, the mortality among officers and troops is greatest among those who remain longest in those climates (JOHNSON, MARTIN, TULLOCH, MACPHERSON, BOUDIN). Dr. Macpherson also makes the significant remark, that the small mortality among officers, compared with soldiers, in India is due to the greater facilities they enjoy of obtaining change of climate when they fall sick. Although the constitution of the man may be so modified that comparative health may be retained, yet there is a *morbid degradation* of the physical and intellectual constitution. If, however, he or his descendants are taken back to their native climate, they may yet revert to the healthful standard of their original types. The good effects of limiting the period of service of our troops in the West Indies to three years has shown this, in sustaining for a greater period the strength of the regiments, a protracted residence of the European regiments in India having been followed by the most disastrous results. "European regiments in India have melted away like the spectres of a dream. A thousand strong men form this year a regiment: a year passes, and *one hundred and twenty-five* new recruits are required to fill up the broken column; and, eight years having come and gone, not a man of the original thousand remains in the dissolving corps." With regard to the Bombay fusilier European regiment, for instance, Dr. Arnot has shown that its losses averaged 104 per 1,000 per annum—a loss equivalent to the entire absorption of the regiment in nine years and seven months. In Bengal, also, it is an ascertained fact that a British regiment of 1,000 men dissolved entirely away in eleven years, even in favourable times, and with all the improved conditions of the service. Dr. Arnot's statistics show that the Bengal army lost annually 9 *per cent.* of its numbers, giving a total loss in eight years of upwards of 14,000 men out of an army of 156,130 men. (See *Trans. Med. Ph. Society of Bombay* for 1855; *Indian Annals of Medical Science;* and *Sanitary Review* for October, 1857; whence these statements are quoted.)

In the *Statistical Reports of the Army Medical Department* (of which ten annual volumes have now been published, for the years 1859 to 1868 inclusive), full information respecting the health of the army all over the world has been given by Dr. T. Graham Balfour, F.R.S., Deputy Inspector-General of Hospitals, to whom the science of medicine is so largely indebted in the direction of "Medical Geography." The leading facts are embraced in the following account of—

The Sickness and Mortality of British Troops at Different Places over the Globe, from 1859 *to* 1868 *inclusive.*

I. **In the Stations of the United Kingdom** the sickness is represented by 956 admissions, and the mortality by 9·57 deaths *per* 1,000 men. The former have ranged between 1,053 admissions in 1860, and 853 in 1866, and the latter between 10·90 deaths in 1868, and 8·72 *per* 1,000 in 1862.

The class of diseases which gives rise to the largest proportion of cases is that of *Venereal,* by which nearly one-third of all the admissions into hospital is caused. Next to it rank *miasmatic diseases, diseases of the respiratory system,* and *diseases of the integumentary system. Tubercular diseases,* chiefly *phthisis,* occasion exactly one-third of the whole mortality, and diseases of the *respiratory system* stand next to them. The admissions by *tubercular diseases* average 17 *per* 1,000, and the deaths 3·17; the admissions by diseases of the *respiratory system* amount to 86 *per* 1,000, and the deaths to 1·31 *per* 1,000.

II. **The Mediterranean Stations.**—1. *Gibraltar.* The sickness here is represented by 789 admissions, and the mortality by 8·98 deaths *per* 1,000 men. The admissions have varied from 949 *per* 1,000 in 1859, to 587 in 1866, and the deaths from 23·74 in 1865, to 4·36 in 1866. The high ratio of mortality in 1865 was caused by a visitation of epidemic cholera, which cut off 15·83 *per* 1,000 of the strength. Omitting that year, the mortality was highest in 1860, when it amounted to 11·06 *per* 1,000.

Of *miasmatic diseases* the *continued fevers* are the most prevalent, and at the same time the most fatal. The sickness from them is represented by 77 admissions, and the mortality by 1·80 deaths *per* 1,000 men. The causes of such prevalence are represented to be—(1.) Overcrowding—the space in one of the barracks having been, during part of the time included in the Reports, only 322 cubic feet per man! (2.) Exposure to sun-heat; (3.) Imperfect drainage and sewerage. *Diarrhœa* and *dysentery* are FOUR TIMES as prevalent as among troops at home, and have occasioned 49 admissions, *rheumatism* 36 admissions, and *ophthalmia* 39 admissions *per* 1,000 men.

Venereal diseases give rise to nearly one-fourth of the admissions into hospital.

2. *Malta.* The sickness at this station is represented by 882 admissions, and the mortality by 14·34 deaths *per* 1,000 of mean strength. The admissions have ranged from 1,214 in 1859 to 666 in 1863, and the deaths between 26·44 in 1865 and 6·53 in 1864. The high rate of mortality in 1865 was caused by epidemic cholera, which occasioned 86 deaths, or 15·57 *per* 1,000 of the force. This disease again broke out in 1867, when the deaths by it were 22, or 4·47 *per* 1,000; but in the same year fever was very prevalent and fatal, the deaths by it being 7·93 *per* 1,000 of force.

The diseases of the *miasmatic class,* in the order of their greatest prevalence, are as follow:—

Continued fever, represented by 188, *dysentery* and *diarrhœa,* by 72, *ophthalmia*, by 67, admissions *per* 1,000 men. Of these diseases *continued fever* has been the most fatal, particularly in the first and last two years of the disease, the deaths having amounted in 1859 to 8·85, in 1867 to 7·93, and in 1868 to 6·32 per 1,000 of the force.

3. The *Ionian Islands* were garrisoned by British troops till May, 1864, when, on their being ceded to Greece, the troops were withdrawn. During the preceding five years the admissions into hospital amounted to 782, and the deaths to 8·88 *per* 1,000 of mean strength.

The most prevalent diseases were *continued fevers, dysentery* and *diarrhœa*, and *ophthalmia.* The *contiued fevers* were the cause of two-fifths of the mortality. Their prevalence and fatal character in Corfu were attributed, and apparently with justice, to the unhealthy situation of the Fort-Neuf Barracks, their overcrowded state and defective ventilation, and to the generally insanitary condition of the town.

III. **The Stations in British America.**—1. In *Canada* the sickness is represented by 686 admissions *per* 1,000 of mean strength, and the mortality by 9·87 deaths *per* 1,000. The admissions have varied from 539 *per* 1,000 in 1860 to 730 in 1868, and the deaths from 11·62 in 1864 to 8·36 in 1862. The miasmatic diseases, in the order of their greatest prevalence, are as follow:—

Rheumatism, represented by 30 admissions *per* 1,000 of mean strength; *sore throat* and *influenza*, by 30; *dysentery* and *diarrhœa*, by 23; *continued fevers*, by 17; and *ophthalmia*, by 15 *per* 1,000. Tubercular diseases, accidents, and diseases of the respiratory and circulatory systems are the chief causes of mortality. The deaths by *continued fevers* amounted only to ·65 *per* 1,000 of the strength on the average of ten years.

2. In *Nova Scotia* and *New Brunswick* the sickness is represented by 541 admissions, and the mortality by 7·76 deaths *per* 1,000 of mean strength. The admissions have fluctuated between 604 *per* 1,000 in 1864, and 473 in 1868, and the deaths between 5·17 in 1860 and 9·65 *per* 1,000 in 1868. The most prevalent diseases of the miasmatic class are *sore throat* and *influenza, rheumatism* and *ophthalmia.* The tubercular diseases are the most fatal, causing one-fifth of the whole mortality. Next to them stand accidents, diseases of the respiratory organs—chiefly *pneumonia* and *bronchitis*—and of the circulatory system.

3. In *Newfoundland* the sickness is represented by 582 admissions, and has ranged between 1,409 in 1860 and 324 in 1864, and the mortality by 10·80 deaths *per* 1,000 of mean strength, the highest ratio having been 19·80 *per* 1,000 in 1865, while in 1860 no deaths occurred. The high ratio of admissions in 1860 and also in 1859 was owing to the force being composed of old soldiers enrolled in a veteran company, and the great range in the proportion of deaths in different years is chiefly a result of the small numbers under observation. The most prevalent diseases are *sore throat, influenza, bronchitis,* and *rheumatism.*

4. *British Columbia* was occupied by a detachment of Royal Engineers during the four years 1859-62. For that period the sickness is represented by 702 admissions *per* 1,000 mean strength, and the mortality by 14·52 deaths *per* 1,000; but of the eight deaths which occurred, five were by drowning, and one that of a man frozen to death; so that

two only, or in the ratio of 0·36 *per* 1,000 of mean strength, were the result of disease. *Influenza* and *venereal* were the prevailing diseases.

5. In *Bermuda* the sickness is represented by 724 admissions, and the mortality by 30·15 deaths *per* 1,000 of mean strength. The admissions have varied from 976 in 1864 to 537 in 1859, and the deaths from 169·54 *per* 1,000 of strength in 1864 to 8·55 in 1860. The enormously high ratio in 1864 was caused by an outbreak of yellow fever, which in twenty weeks cut off 173 men from a strength of 1,135 at the commencement of the epidemic. Bermuda has been subject to visitations of yellow fever in an epidemic form at irregular intervals; but during the ten years from 1859 inclusive, 1864 was the only year in which it so appeared. In 1866 and 1868 the mortality by *continued fevers* was high, but there were no cases of yellow fever. *Continued fevers, dysentery* and *diarrhœa,* and *diseases of the integumentary system* are the most prevalent diseases, and after yellow fever those of the nervous system the most fatal, owing principally to the number of deaths from *delirium tremens.* The mortality by tubercular diseases and by accidents is also high.

IV. **West Indian Stations.**—1. Among **European Troops** of the *Windward* and *Leeward Command* the sickness is represented by 1,189 admissions *per* 1,000 of mean strength, and the mortality by 13·35 deaths *per* 1,000. The admissions have ranged between 1,619 *per* 1,000 in 1866, and 1,001 in 1861, and the deaths between 29·29 in 1866, and 5·55 in 1861. The high rate of admissions in 1866 was caused by the prevalence of *paroxysmal fevers* in Demerara; the ratio was also unusually high in 1864 from an epidemic of *remittent fever* at Barbadoes, fortunately not of a fatal character. The great mortality in 1866 was from an epidemic of *yellow fever* in Demerara, when the deaths amounted to one-fourth of the force. The white troops have since been withdrawn from the station. The rate of mortality was much above the average in 1865 from *paroxysmal fevers* in Demerara, and in 1859 from an outbreak of *yellow fever* at Trinidad. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow:—*Paroxysmal fevers,* represented by 262 admissions; *ophthalmia,* by 75; *dysentery* and *diarrhœa,* by 61; *continued fevers,* by 51; and *rheumatism,* by 24 admissions *per* 1,000 of mean strength.

Among **African and Coloured Creole Troops** the sickness is represented by 919 admissions, and the mortality by 21·14 deaths *per* 1,000 of mean strength, the former ranging between 1,043 in 1867 and 805 in 1862, and the latter between 16·48 in 1863 and 38·14 *per* 1,000 in 1867. *Enthetic* and *miasmatic* diseases are the most prevalent, and *tubercular* and *miasmatic* the most fatal. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow:—*Rheumatism,* represented by 78 admissions *per* 1,000 of mean strength; *paroxysmal fevers,* by 71; *ophthalmia,* by 21; *dysentery* and *diarrhœa,* by 20; *sore throat* and *influenza,* by 19; *eruptive fevers,* by 16; and *continued fevers,* by 16. *Tubercular diseases* are most fatal to this class of troops, causing one-third of all the deaths; next to these rank *diseases of the respiratory and nervous systems, paroxysmal fevers,* and *dysentery and diarrhœa.*

2. The sickness in *Jamaica* among **European Troops** is represented by 1,023 admissions *per* 1,000 mean strength, ranging between 13·97 in

1865 and 5·80 in 1868, and the mortality by 21·01 deaths, ranging from 71·07 in 1867 to 7·35 in 1864. The very high ratio of deaths in 1867 was caused by an epidemic of yellow fever. Except in that year, the highest proportion of deaths was in 1865, when it amounted to 27·87 *per* 1,000 of the strength, chiefly from fever. The most prevalent of the *miasmatic* diseases are as follow:—*Paroxysmal fevers,* represented by 125 *per* 1,000 of mean strength; *continued fevers,* by 92; *ophthalmia,* by 50; *dysentery and diarrhœa,* by 45; *rheumatism,* by 29; *sore throat* and *influenza,* by 17; and *yellow fever,* by 13 admissions *per* 1,000.

The most fatal diseases are *yellow, paroxysmal,* and *continued fevers.*

Among the **African and Creole Troops** in *Jamaica* the sickness is represented by 1,264 admissions, and the mortality by 26·01 deaths *per* 1,000 of mean strength. The admissions ranged between 15·31 in 1866 and 8·18 in 1860, and the deaths between 34·05 in 1864 and 10·97 in 1868 *per* 1,000 of mean strength.

The most prevalent *miasmatic* diseases are *paroxysmal fevers,* represented by 219 admissions *per* 1,000 of mean strength; *rheumatism,* by 83; *continued fevers,* by 53; *dysentery* and *diarrhœa,* by 30; *sore throat* and *influenza,* by 27; *ophthalmia,* by 24; and *eruptive fevers,* by 7 admissions *per* 1,000 of mean strength.

Miasmatic diseases have been the most fatal, one-half of the deaths by them being by paroxysmal fevers; tubercular diseases and those of the respiratory system rank next, and have been the cause of two-fifths of all the deaths.

3. In the *Bahamas,* among **African and Creole Troops,** the sickness is represented by 923 admissions, and has ranged between 580 in 1863 and 1,303 in 1868; and the mortality is represented by 26·17 deaths *per* 1,000 of mean strength, ranging from 14·48 in 1863 to 40·37 in 1859.

Miasmatic diseases are the most prevalent, and tubercular the most fatal. The mortality is also very high from those of the respiratory system.

4. In *Honduras,* among the **African and Creole Troops,** the sickness is represented by 912 admissions, and the mortality by 20·60 deaths *per* 1,000 of mean strength. The highest ratio of admissions was 1,523 in 1861, and the lowest 607 in 1868; the highest ratio of deaths was 52·00 *per* 1,000 of the strength in 1866, and the lowest 6·21 *per* 1,000 in 1859.

The prevalent diseases are *miasmatic.* Skin diseases, especially *eczema, ulcers,* and *boils,* and venereal diseases also abound. Tubercular diseases have been the most fatal; but miasmatic diseases and those of the nervous, circulatory, and respiratory system have also furnished a high ratio of deaths.

V. **West African Stations.**—1. In *Sierra Leone* the sickness is represented by 998 admissions, and the mortality by 29·43 deaths *per* 1,000 of mean strength. The admissions ranged from 542 in 1859 to 1,383 in 1866, and the deaths from 14·02 in 1859 to 41·33 in 1867.

2. At the *Gambia* the sickness is represented by 1,148 admissions, and the mortality by 32·99 deaths *per* 1,000 of mean strength; the former ranging between 653 in 1859 and 1,667 in 1863, and the latter between 17·74 in 1865 and 52·38 in 1866.

3. For the *Gold Coast* and *Lagos* the sickness is represented by 1,495

admissions, and the mortality by 45·87 deaths *per* 1,000 of mean strength. The lowest ratio of admissions was 581 *per* 1,000 in 1859, and the highest 23·67 in 1864; the lowest ratio of mortality was 9·57 in 1860, and the highest 83·06 in 1864.

The most prevalent miasmatic diseases in *Sierra Leone* are as follow:—*Paroxysmal fevers,* represented by 146 admissions *per* 1,000 mean strength; *rheumatism,* by 66 *per* 1,000; *dysentery* and *diarrhœa,* by 32; *ophthalmia,* by 15; *eruptive fevers,* by 11 *per* 1,000.

The most prevalent on the *Gambia* are,—

Paroxysmal fevers, represented by 173 admissions *per* 1,000 of mean strength; *dysentery, diarrhœa,* and *cholera,* by 84 *per* 1,000; *rheumatism,* by 61 *per* 1,000; *eruptive fevers,* by 26 *per* 1,000; *ophthalmia,* by 18 admissions *per* 1,000 men.

The most prevalent on the *Gold Coast* and at *Lagos* are,—

Paroxysmal fevers, represented by 367 admissions *per* 1,000 mean strength; *dysentery* and *diarrhœa,* by 220; *rheumatism,* by 59; and *ophthalmia,* by 20 *per* 1,000. *Small-pox* and *measles* are sometimes epidemic; and *Guinea worm,* among parasitic diseases, holds a prominent place; indeed, in 1861, this affection was the cause of one-third of the admissions into hospital. During the ten years 1859–68 there were 412 admissions into hospital by this disease, being in the ratio of 90 *per* 1,000 of the strength. The admissions by this disease at Sierra Leone during the same period was 59, and at the Gambia only 3. It is stated that almost all these cases were men who had contracted the disease while serving on the Gold Coast.

VI. **St. Helena.**—The sickness is represented by 830 admissions *per* 1,000 mean strength; the mortality, by 9·00 deaths *per* 1,000. The admissions have ranged from 1,037 *per* 1,000 of mean strength in 1861 to 612 in 1867; and the deaths from 12·90 in 1859 to 4·75 *per* 1,000 in 1868.

The prevalent miasmatic diseases are *continued fevers,* represented by 84; *dysentery* and *diarrhœa,* by 77; *rheumatism,* by 32; *ophthalmia,* by 26; *sore throat* and *influenza,* by 20; and *paroxysmal fevers,* by 17 admissions *per* 1,000 of mean strength.

The most fatal diseases are the tubercular and those of the nervous and circulatory systems. *Intemperance* prevails to a great extent.

VII. **Cape of Good Hope.**—The sickness is represented by 975 admissions *per* 1,000 mean strength, ranging between 841 in 1863 and 1,241 in 1867; the mortality by 11·02 deaths *per* 1,000; the lowest ratio having been 9·73 in 1862, and the highest 13·16 *per* 1,000 in 1860.

The most prevalent miasmatic diseases are *ophthalmia,* represented by 81 admissions *per* 1,000 mean strength; *rheumatism,* by 47; *continued fever,* by 46; *dysentery* and *diarrhœa,* by 40; *sore throat* and *influenza,* by 24; *paroxysmal fevers,* by 17 admissions *per* 1,000. The proportion of paroxysmal fevers, however, has been unduly increased by the arrival in the command of several regiments from China, where the men had contracted a liability to ague, which continued to affect them for a considerable time after their arrival at the Cape, but was in nowise attributable to its climate.

The admissions into hospital at the Cape have been greatly increased during the last five years of the decade by the prevalence of venereal diseases; during that period they have been the cause of one-third of the total admissions.

The diseases which have caused the greatest mortality have been those of the circulatory and nervous systems, continued fevers, and accidents. Intemperance prevails to a large extent, and is among the chief causes of the prevalence of diseases of the heart in this station (NICHOLSON, TAYLOR). *Cardiac disease* is the cause of 1·73 deaths *per* 1,000 of the mean strength, and also of a considerable amount of invaliding from the Cape.

VIII. **Island of Mauritius.**—The sickness is represented by 1,039 admissions, ranging between 608 in 1860 and 2,336 in 1868. The very high ratio in the latter year, and also in 1867, was due to the prevalence of a wide-spread epidemic of malarious fever, affecting both military and civil population. The mortality is represented by 20·63 deaths *per* 1,000 of mean strength, and has ranged between 7·97 in 1865 and 43·92 in 1862, the latter chiefly from epidemic cholera. The epidemic fever in 1867 and 1868 caused a very high ratio of deaths (40·95 and 27·31), and in 1860 the ratio was also very high (23·86 *per* 1,000), from an outbreak of malignant cholera.

The prevalent miasmatic diseases have been *paroxysmal fevers,* represented by 261 admissions *per* 1,000 of mean strength; *dysentery* and *diarrhœa,* by 128; *continued fevers,* by 66; *ophthalmia,* by 29; *spasmodic cholera,* by 10; *rheumatism,* by 26; *sore throat* and *influenza* by 8 admissions *per* 1,000 men. The high ratio of cases of paroxysmal fevers has been entirely due to the epidemic of the last two years of the decade, the average of the preceding eight years having been only 14 *per* 1,000.

The most fatal diseases have been *spasmodic cholera, dysentery* and *diarrhœa, paroxysmal* and *continued fevers.*

IX. **Ceylon.**—The sickness among **European Troops** is represented by 1,445 admissions, and the mortality by 24·29 deaths *per* 1,000 of mean strength. The admissions have ranged between 1,706 *per* 1,000 in 1864 and 1,088 in 1867; and the deaths between 35·06 in 1859 and 14·15 *per* 1,000 in 1867.

The most prevalent diseases of the miasmatic class are *dysentery* and *diarrhœa,* represented by 208 admissions *per* 1,000 men; *paroxysmal fevers,* by 117; *ophthalmia,* by 80; *continued fevers,* by 76; *rheumatism,* by 53; *sore throat* and *influenza,* by 15; and *malignant cholera,* by 4 *per* 1,000.

The most fatal diseases are *dysentery, diarrhœa,* and *cholera;* diseases of the digestive system, particularly *hepatitis, tubercular diseases,* and those of the *nervous system.*

Among the **Asiatic and Cingalese Troops** the sickness is represented by 1,013 admissions, and the mortality by 14·81 deaths *per* 1,000 of mean strength; the admissions have ranged from 1,339 in 1862 to 477 in 1868, and the deaths 32·47 in 1864 to 9·18 in 1865; the high ratio in the former year was chiefly due to deaths of men who had been wounded, from a wing of the regiment serving at Hong Kong.

The prevalent diseases are *paroxysmal fever,* represented by 376 admissions; *dysentery* and *diarrhœa*, by 61; *ophthalmia*, by 64; *rheumatism*, by 50; *continued fevers*, by 28; *eruptive fevers*, by 9; and *cholera*, by 7 admissions *per* 1,000 men.

The fatal diseases are *spasmodic cholera*, *dysentery* and *diarrhœa*, *paroxysmal fevers*, and *tubercular diseases*.

Trincomalee is the most sickly station. Ague prevails there from the marshy soil of *paddy* fields and cocoa-nut gardens.

X. **Australasia.**—1. *Australia and Tasmania.* The sickness is represented by 708 admissions *per* 1,000 of mean strength, with a mortality of 13·41 deaths *per* 1,000. The admissions ranged between 587 in 1865 to 888 in 1860, and the deaths between 7·51 in 1867 and 22·00 in 1862. The average ratio of deaths, however, have been unduly increased by the withdrawal of a portion of the troops for field service in New Zealand, the weakly and delicate men being left with the wings of the regiments which remained in Australia.

The miasmatic diseases, in the order of their greatest prevalence, are *ophthalmia*, represented by 39 admissions *per* 1,000; *rheumatism*, by 36; *sore throat* and *influenza*, by 33; *dysentery* and *diarrhœa*, by 32; *continued fevers*, by 18; and *paroxysmal fevers*, by 5 admissions *per* 1,000 men.

The most fatal diseases in this command are the *tubercular*, which caused 4·81 per thousand of strength. Diseases of the *circulatory system* gave rise to 2·16; and those of the *nervous system* to 1·57. Miasmatic diseases gave rise to 1·47 deaths per 1,000 of the strength, one-half of these being caused by *continued fevers*.

2. *New Zealand.* The sickness is represented by 588 admissions *per* 1,000, and the mortality by 16·08 deaths. But of these 4·96 were the result of wounds in action, leaving a proportion of 11·12 *per* 1,000 from the ordinary contingencies of colonial service. The admissions varied from 749 in 1868 to 448 in 1866, and the deaths from 30·43 in 1860 to 4·44 in 1859. If the casualties of war be omitted, the highest ratio of deaths was 16·58 in 1864.

The miasmatic diseases, in the order of greatest prevalence, are *dysentery* and *diarrhœa*, represented by 54 admissions *per* 1,000 men; *ophthalmia*, by 47; *continued fevers*, by 32; *rheumatism*, by 31; *sore throat* and *influenza*, by 25; and *paroxysmal fevers*, by 4 admissions *per* 1,000 men.

The highest ratio in deaths, next to casualties in action, has been caused by accidents, the deaths by them having amounted to 2·34 *per* 1,000. The most fatal diseases have been miasmatic, tubercular, and those of the circulatory system. *Continued fever* was the most fatal of the miasmatic diseases, the ratio of deaths by them having been 1·21 per 1,000 of strength.

XI. **China.**—The sickness among **European Troops** is represented by 2,054 admissions, and the mortality by 54·85 deaths *per* 1,000. The sickness among **Native Troops** is represented by 1,259 admissions, and the mortality by 26·93 *per* 1,000.

1. *In Southern China* the sickness among **European Troops** is represented by 2,224 admissions *per* 1,000, with a mortality represented by 45·78 *per* 1,000. The admissions have ranged between 2,783 in 1859 and 1,088 in 1868, and the deaths between 114·10 in 1865 and 14·97

in 1868. The excessive mortality in 1865 was due to an epidemic of *fever*, chiefly among the troops at Kowloon.

The miasmatic diseases, in the order of greatest prevalence, are as follow:—*Paroxysmal fevers*, represented by 609 admissions *per* 1,000; *continued fever*, by 237; *dysentery*, and *diarrhœa*, by 206; *ophthalmia*, by 74; *rheumatism*, by 64; *sore throat* and *influenza*, by 16; *malignant cholera*, by 5·6; and *eruptive fevers*, by 3·1 *per* 1,000 men.

The fatal diseases were *dysentery* and *diarrhœa*, *paroxysmal* and *continued fevers*, and *spasmodic cholera*.

The sickness amongst **Native Troops** in *China* is represented by 1,576 *per* 1,000, and the mortality is represented by 31·67 *per* 1,000. The admissions ranged between 2,637 in 1860 and 981 in 1868, and the deaths between 53·75 in 1859 and 17·06 in 1865 *per* 1,000 of mean strength. The diseases, arranged in the order of greatest prevalence, are as follow:—*Paroxysmal fevers*, represented by 676 admissions *per* 1,000 men; *dysentery*, *diarrhœa*, and *cholera*, by 200; *rheumatism*, by 103; *continued fever*, by 42; *ophthalmia*, by 14; *eruptive fevers*, by 8; *cholera*, by 8; and *sore throat* and *influenza*, by 5 *per* 1,000 men.

The most fatal diseases were *dysentery* and *diarrhœa*, *paroxysmal fevers*, *cholera*, and *continued fever*.

In *North China* the results are so much affected by the peculiar circumstances of field service in the campaign of 1860 and the subsequent military occupation of Shanghai, Tein-tsin, and the Taku Forts, that they would only mislead if stated as the effect of climate. It may, however, be interesting to state the amount of sickness and mortality among the **European Troops** during the three years of such special service. The admissions into hospital in 1861, 1862, and 1863 were 1,415, 1,880, and 1,849 *per* 1,000 of mean strength, and the deaths were 51·08, 124·46, and 67·73 *per* 1,000 in these years respectively.

The prevalence of miasmatic diseases on the average of the three years was as follows:—*Dysentery* and *diarrhœa*, 334 admissions *per* 1,000 men; *paroxysmal fevers*, 270; *continued fevers*, 169; *ophthalmia*, 67; *rheumatism*, 46; *malignant cholera*, 37; *eruptive fevers*, 13; *sore throat* and *influenza*, 10 *per* 1,000. *Dysentery* and *diarrhœa* and *malignant cholera* were the most fatal diseases, the former having cut off 24·60 and the latter 23·56 *per* 1,000 of mean strength.

In 1861, at Tein-tsin, 19 deaths were recorded from *sunstroke* between the 17th and 23rd of July, the *maximum* temperature during that period ranging from 95° to 108° Fahr., in the shade—giving a maximum average of 104° 6′; and the minimum temperature ranged from 73° to 83° Fahr., in the shade; the average being 79° 2′ (Dr. Lamprey).

In 1862 cholera broke out in the field force in the neighbourhood of Shanghai, and cut off 58 *per* 1,000 of the strength. It also attacked the small garrison left at Taku, and the mortality in it amounted to 40 *per* 1,000. It was at this time very fatal among the civil population: "it was intimated on good authority, that between Shanghai and Seon-Kiang (distant about 40 miles) about one-eighth of the population died from cholera alone."

XII. **Japan.**—In 1864, Yokohama became, for the first time, a station for British troops. During the five years, 1864-1868, the admissions into hospital were, on the average, 1,502 per 1,000 of the force annually, ranging from 1,706 in 1864 to 1,265 per 1,000 in 1868. The average ratio of mortality was 20·76 *per* 1,000, ranging between 49·24 *per* 1,000 in 1864, and 11·63 in 1867. These ratios, however, do not fairly represent the influence of the climate of Japan, as prior to 1868 the garrison was composed of troops sent from Hong Kong, in some instances for change of climate, on account of their extremely unhealthy condition at that station. *Paroxysmal fevers* were the most frequent cause of admission into hospital, and were clearly the result of previous service in China—as in 1868, when the troops had come from the Cape of Good Hope, the admissions by paroxysmal fevers amounted to only 3 in 785 men. *Cholera, dysentery,* and *diarrhœa* were the most fatal diseases, but they also in most instances occurred in men broken down by previous service in China. In two years, 1868, 1869, when the health of the troops had not been deteriorated in this manner, the mortality amounted only to 11·7 *per* 1,000 of the force.

XIII. **Stations in India.**—The prevalence of sickness, on the average of the nine years, 1860-1868, as shown in the *Army Medical Department Reports,* is represented by 1,592 admissions *per* 1,000 mean strength, and the mortality is represented by 27·06 deaths. The relative sickness and mortality are distributed over the three Presidencies, as follow:—

Bengal.—The sickness in Bengal is represented by 1,672 admissions *per* 1,000 of the strength, ranging between 2,023 in 1860 and 1,098 in 1868; and the mortality by 29·89 *per* 1,000—the highest ratio being 39·37 in 1860, and the lowest 22·56 in 1864.

The prevalence of the various groups of miasmatic diseases *per* 1,000 of mean strength has been as follow:—*Paroxysmal fevers,* 461; *dysentery* and *diarrhœa,* 136; *continued fevers,* 131; *rheumatism,* 68; *ophthalmia,* 47; *sore throat and influenza,* 21; *malignant cholera,* 12; and *eruptive fevers,* 2. The deaths by *malignant cholera* have averaged 7·82 *per* 1,000 of strength; by *dysentery* and *diarrhœa,* 3·52; *paroxysmal fevers,* 2·05; *continued fevers,* 1·47; and *eruptive fevers,* ·29 *per* 1,000. Of the other classes of diseases, those of the *digestive* system have been much the most fatal, the ratio of deaths having been 3·75 *per* 1,000, chiefly from *hepatitis.*

Madras.—In the Madras command, the admissions were, on the average of nine years, in the ratio of 1,395 *per* 1,000 of mean strength, ranging between 1,538 in 1864 and 1,254 in 1861; the deaths were in the ratio of 22·17 *per* 1,000, and ranged from 24·96 in 1865 to 15·83 in 1861.

The principal miasmatic diseases furnished on the average the following proportion of admissions *per* 1,000 of strength:—*Dysentery* and *diarrhœa,* 173; *paroxysmal fevers,* 164; *continued fevers,* 75; *rheumatism,* 60; *ophthalmia,* 33; *sore throat* and *influenza,* 17; *malignant cholera,* 5; and *eruptive fevers,* 1 *per* 1,000. The mortality by them was—*dysentery* and *diarrhœa,* 2·99; *malignant cholera,* 2·56; *continued fevers,* 1·19; *paroxysmal fevers,* ·68; and *eruptive fevers,* ·08 *per* 1,000. As in Bengal, the highest ratio of deaths in the other classes was by diseases of the *digestive* system, which amounted to 4·27 *per* 1,000, the most fatal disease being *hepatitis.*

Bombay.—On the average of the nine years, 1860-1868, the admissions into hospital amounted to 1,522 *per* 1,000 of mean strength annually; and ranged between 1,933 in 1860 and 1,098 in 1868. The mortality during the same period was 22·60 *per* 1,000, highest ratio having been 31·70 in 1860, and the lowest, 15·06 in 1866. The admissions by the different groups of miasmatic diseases during the period were as follow:—*Paroxysmal fevers,* 439 *per* 1,000 of strength; *dysentery* and *diarrhœa,* 123; *continued fevers,* 84; *rheumatism,* 57; *ophthalmia,* 48; *sore throat* and *influenza,* 20; *malignant cholera,* 8; and *eruptive fevers,* 2 *per* 1,000. The mortality by these groups of diseases has been—by *malignant cholera,* 4·81 *per* 1,000 of strength; *dysentery* and *diarrhœa,* 2·76; *paroxysmal fevers,* 1·58; *continued fevers,* 1·15; *eruptive fevers,* ·17 *per* 1,000.

As in the other two Presidencies, diseases of the *digestive* system have been the most fatal of the local classes, the deaths being almost entirely by *hepatic* disease. Tubercular diseases have been less fatal in Bombay than in either of the other Presidencies, the ratio of deaths by them having been,—in Bengal, 2·49; in Madras, 2·17; and in Bombay, 1·97 *per* 1,000 of strength—the deaths by these diseases among the invalids on passage to England and at Netley being included.

The lives wasted in India in each year of service (according to the "Report of the Commissioners appointed to inquire into the Sanitary State of the Army in India in 1859," and published in 1863), appear to have been greater in the Queen's regiments than in the Company's regiments. Half of the deaths take place during the first five years; and the numbers invalided increase slightly in each quinquennial period. The decrement year by year of the force in India has been such that 1,000 effectives are reduced to 96 men in twenty years (that is, by death alone, to 344—by death and invaliding, to 216—by death, invaliding, and other causes, to 96). The mean term of service in India has been 8·6 years, and 11 recruits are required annually to every 100 men; so that to maintain 85,856 men 10,000 annual recruits are required; and these are reduced to less than half their original numbers in *eight* years. The half of a regular army so constituted consists of men who have served less than six years; and not more than a fourth of the men are veterans of ten years' standing, on whom the discipline and solidity of an army greatly depend.

The Endemic Diseases of India in the order of greatest prevalence are *paroxysmal fevers, continued fevers, dysentery, diarrhœa, rheumatism, ophthalmia, spasmodic cholera, sore throat* and *influenza.* Arranged in the order of comparative mortality, the diseases are as follow:—*Spasmodic cholera, dysentery, diarrhœa,* and *continued* and *paroxysmal fevers.* These are especially the diseases of the sultry plains of India.

The Fevers are especially the paludal fevers; but both typhus and typhoid fevers are now known to occur in India.

Scarlet Fever also has undoubtedly found its way to India, as shown by a case recorded by Assistant-Surgeon Thomas Maunsell, R.H.A., in the *Indian Medical Gazette* for December 1, 1870. He is the first person who publicly reported the occurrence of scarlet fever in India. It occurred in the hills, and was well marked by sore throat and eruption, followed by a fine mealy scurf, and subsequent desquamation. At the time it occurred there were very many exactly similar cases in the station. No one in the same house took the disease, but many of the servants in the adjoining compound had "fever and sore throat," and other cases occurred at the same station in children, which were well marked, but did not prove contagious.

Staff-Surgeon A. F. Bradshaw, surgeon to his Excellency the Commander-in-Chief in India, has also since shown that *scarlet fever* is known in India; and besides having a family of seven—a mother and six children—under his care, at Simla, suffering from this disease, he gives good grounds for the inference that scarlet fever was not unknown to Jackson, Murchison, and Morehead, or a disease very like, if not identical with it.

The occurrences of twelve cases of scarlet fever are also recorded in the statistical reports of the Army Medical Department between 1860 and 1868 inclusive, in the Bengal Presidency, of which three died; one also occurred in Madras and two in Bombay, one of which died (*Indian Med. Gazette*, August 1, 1871).

The histories of Maunsell's and Bradshaw's cases are clearly histories of scarlet fever; and the latter observer gives a well-marked example of the importation of the disease into India during the first quarter of this year (1871), when the fever appeared on board a troop ship bringing to India a number of detachments. Of these, one proceeded in March to join the 92nd regiment at Jullundur, and another to reinforce the 58th at Sealkote. In both regiments scarlet fever broke out in about a month afterwards among the children, and some of them died; and in the 58th numerous cases of peculiar sore throat were observed at the same time among the men. Dr. Lundie, of the 58th regiment, has since also recorded his evidence of its occurrence in India, and so has Surgeon Chapple of the Royal Artillery.

When the free intercourse between this country and India, and the rapid passages now made to India from this country are taken into account, the importation of contagious diseases between the two countries is obviously facilitated.

The Dysentery of India is most prevalent in the plains during the hot and rainy seasons; and amongst British soldiers it is computed that eleven cases of dysentery occur to one amongst the native soldiery. The cases of dysentery present "a spectacle of distress of as pitiable a kind as can be found in the history of human suffering."

As to Diseases of the Liver no statistics can give any idea of the extent of these diseases; and in acute inflammation, so prevalent in the plains, the danger to life is imminent from the first, while in the event of recovery, impairment of health, more or less permanent, is certain.

Cholera is the most acute of acute diseases. Troops, both European and Native, are most liable while on the march to seizure by this disease; and when the attacks have occurred in cantonments after the march, the prevalence of the epidemic is generally in proportion to the length of the march. (See under "Cholera," vol. i., for its endemic area.)

The annual rate of mortality among soldiers is now less than 10 *per* 1,000 in England; in India, according to the *Commissioners' Report*, it was 67 *per* 1,000 (Bengal), of which 58 *per* 1,000 were due to zymotic diseases, the fevers killing 17 men in 1,000; dysentery and liver disease, 20; cholera and diarrhœa, 18 men in 1,000. *Delirium tremens, catarrh, syphilis, rheumatism*, and *scurvy* are much more fatal in India than in England.

Nearly all the diseases fatal in India are accompanied by profuse discharges, with which the air, water, linen, bedding, closets, walls of hospitals, and barracks become more or less infected; so that the *materis morbi* come into contact with all the inmates of buildings where the disease prevails. In India the soldier's sickness is doubled. As to how far this sickness and mortality are aggravated by the unsanitary state of the towns and the mode of life in India, the reader is referred to the *Report of the Royal Commission on the Sanitary State of the Army in India*, and to the second edition of Miss Nightingale's pamphlet, entitled *How to Live and not Die in India.*

As legitimate deductions from the preceding statements, the following summaries of results are here given for the years named below:—

(A.)

Stations of the British Army, arranged in the Order of the GREATEST NUMBER OF ANNUAL ADMISSIONS per 1,000 of Mean Strength.		Annual Admissions per 1,000 Mean Strength.	FLUCTUATIONS.	
			Highest Ratio.	Lowest Ratio.
WHITE TROOPS.				
1. South China, . .	1859-68	2,224	2,783	1,088
2. Bengal, . . .	1860-68	1,672	2,023	1,374
3. Bombay, . . .	1860-68	1,522	1,933	1,098
4. Japan, . . .	1864-68	1,502	1,706	1,265
5. Ceylon, . . .	1859-68	1,445	1,706	1,088
6. Madras, . . .	1860-68	1,395	1,490	1,255
7. Windward and Leeward Command,	1859-68	1,189	1,619	1,001
8. Mauritius, . .	1859-68	1,039	2,336	608
9. Jamaica, . . .	1859-68	1,023	1,397	580
10. Cape of Good Hope, .	1859-68	975	1,241	841
11. United Kingdom, .	1860-68	956	1,053	853
12. Malta, . . .	1859-68	882	1,214	666
13. St. Helena, . .	1859-68	830	1,037	612
14. Gibraltar, . .	1859-68	789	949	587
15. Ionian Islands, .	1859-64	782	881	559
16. Bermuda, . . .	1859-68	724	976	537
17. Australia, . .	,,	708	888	587
18. Canada, . . .	,,	686	730	539
19. New Zealand, . .	,,	588	749	448
20. Newfoundland, .	,,	582	1,409	324
21. Nova Scotia, &c., .	,,	541	604	473
COLONIAL CORPS.				
1. Gold Coast and Lagos,*	1859-68	1,495	2,367	581
2. Jamaica,† . . .	,,	1,264	1,531	818
3. South China,‡ . .	,,	1,259	2,637	987
4. Gambia,† . . .	,,	1,148	1,667	653
5. Ceylon,§ . . .	,,	1,013	1,339	477
6. Sierra Leone,† . .	,,	998	1,383	542
7. Bahamas,† . . .	,,	923	1,303	580
8. Windward and Leeward Command,†	,,	919	1,043	805
9. Honduras,† . .	,,	912	1,523	607

* Africans.
† Africans and Coloured Creoles.
‡ Asiatics.
§ Cingalese and Natives of India.

(B.)

Stations of the British Army, arranged in the Order of the Greatest Annual Mortality per 1,000 of Mean Strength.		Annual Mortality per 1,000 Mean Strength.	Fluctuations.	
			Highest Ratio.	Lowest Ratio.
European Troops.				
1. South China,	1859-68	45·78	114·10	14·97
2. Bermuda,	1859-68	30·15	169·54	8·55
3. Bengal,	1860-68	29·89	45·57	22·56
4. Ceylon,	1859-68	24·29	35·06	14·15
5. Bombay,	1860-68	23·58	36·64	15·06
6. Madras,	1860-68	22·17	24·96	15·83
7. Japan,	1864-68	20·76	49·24	11·63
8. Jamaica,	1859-68	21·01	71·07	7·35
9. Mauritius,	,,	20·63	43·92	7·97
10. New Zealand,*	,,	16·08	16·58	4·44
11. Malta,	,,	14·34	26·44	6·53
12. Australia,	,,	13·41	22·00	7·51
13. Windward and Leeward Command,	,,	13·35	29·29	5·55
14. Cape of Good Hope,	,,	11·02	13·16	9·73
15. Newfoundland,	,,	10·80	19·80	...
16. Canada,	,,	9·87	11·62	8·36
17. United Kingdom,	1860-68	9·52	10·90	8·72
18. St. Helena,	1859-68	9·00	12·90	4·75
19. Gibraltar,	1859-68	8·98	23·74	4·36
20. Ionian Islands,	1859-64	8·88	12·72	5·77
21. Nova Scotia, &c.,	1859-68	7·76	9·65	5·17
Coloured Corps.				
1. Gold Coast,†	1859-68	45·87	83·06	9·57
2. Gambia,‡	,,	32·59	52·58	17·74
3. Sierra Leone,‡	,,	29·43	41·33	14·02
4. South China,§	,,	26·93	61·17	17·06
5. Bahamas,‡	,,	26·17	40·37	14·48
6. Jamaica,‡	,,	26·01	34·05	10·97
7. Windward and Leeward Command,‡	,,	21·14	38·14	16·48
8. Honduras,‡	,,	20·60	52·00	6·21
9. Ceylon,‖	,,	14·81	32·47	9·18

* Exclusive of men killed in action or who died of their wounds.

† Africans.

‡ Africans and Coloured Creoles.

§ Asiatics.

‖ Cingalese and Natives of India.

Dr. Balfour's valuable series of reports show at a glance the great diversity in the amount of sickness and mortality to which British troops were liable (in former times) at these stations, while serving in our widely extended colonial possessions; and it will now appear how great has been the reduction, especially *in the mortality* of the troops, during the ten years comprised in the preceding tables, as compared with the rates when Dr. Balfour and those with whom he laboured first brought the sickness and mortality of the army to the notice of the Secretary of State for War.

Several features are especially characteristic, namely, the great fluctuations of the rates in tropical regions compared with slight fluctuations in temperate regions; also the great mortality in some years marked by epidemics; and a high rate of mortality amongst coloured troops—suggestive of inquiry as to whether their sanitary condition is sufficiently looked after

Leaving out of consideration Sierra Leone, as an extreme instance of unhealthiness, and from which it has been deemed expedient altogether to withdraw European troops, the admissions into hospital ranged, during the twenty years subsequent to 1836, *i. e.*, up to 1856, between 529 *per* 1,000 of the strength in New Zealand and 2,117 *per* 1,000 in Bombay; while the mortality ranged between an average of 11 *per* 1,000 in Newfoundland and 76·2 *per* 1,000 in Bengal.

Not only, however, did these great differences exist in the amount of sickness and mortality at different stations on the average of that series of years, but they occurred to as great an extent in different years upon the same station. Thus, in the period 1817 to 1836, the sickness in the Windward and Leeward Command ranged between 1,512 and 2,365 *per* 1,000—during the period from 1859 to 1868, it has ranged between 1,001 and 1,619; in Jamaica it ranged between 1,389 and 2,423—during the period now under review it has ranged from 580 to 1,397; in Ceylon it ranged between 1,216 and 2,895—since reduced to a range of between 1,028 and 1,706. At Gibraltar it ranged between 631 and 1,498—now the range is between 587 and 949; and even in Canada it varied from 847 to 1,409—which has been reduced to a range between 539 and 730 *per* 1,000.

But the difference in the mortality in different years is even more striking: thus, in the Windward and Leeward Command it varied between 43 and 162 *per* 1,000—while the summary now given shows a range from 5·55 to 29·29; in Jamaica, the range, formerly, between 61 and 307, has been reduced to between 7·35 and 71·07; in Ceylon, the range was between 34 and 218—the ratio is now from 14·15 to 35·06; in Gibraltar, a range between 8 and 128 has been reduced to between 4 and 24; in Canada, a range between 9 and 48 now appears between 8 and 11; and in Nova Scotia, a range between 7 and 40 has since been reduced to between 5 and 9 *per* 1,000.

These facts strikingly illustrate the necessity for a prolonged period of observation to obtain a fair average, and for great caution in making deductions from observations extending over limited periods and insufficient numbers.

Another striking fact shown by Dr. Balfour's tables is the great reduction which has taken place during the last thirty years in the mortality at some of the stations—a result fairly attributable, in a considerable degree at least, to the removal of those causes of disease which were brought to light by the Statistical Reports, and to the adoption of the measures recommended from time to time for improving the health of the troops. The most striking examples are to be found in Jamaica, where the deaths have fallen from an average of 128 to 60·8, and now to 26·01 *per* 1,000; in Newfoundland, from 37·7 to 11, and now 10; in St. Helena, from 25·4 to 12·3, and now 9; and in Ceylon, from 74·9 to 38·6, and now 24·29 *per* 1,000—"*now*" meaning the ten years 1859 to 1868 inclusive.

Although military statistics may not be proper *criteria* of the healthiness of a place, nor of the diseases affecting the civil community, yet the information is of great value, and the following tables, kindly prepared for this edition by Dr. T. G. Balfour, F.R.S., show at a glance the *sickness* and *mortality* of the British army at all the stations occupied by it, and the classes of diseases by which *sickness* and *mortality* are caused; and, if compared with previous statements, will show the great reduction which has taken place in the amount of sickness and mortality compared with former times:—

TABLE SHOWING THE PROPORTION OF ADMISSIONS INTO HOSPITAL AND DEATHS PER 1,000 OF MEAN STRENGTH, AT EACH OF THE FOLLOWING STATIONS, ON THE AVERAGE OF THE PERIODS NOTED.

I.—EUROPEAN TROOPS.

Stations and Period of Observation.	United Kingdom, 1860–68.		Gibraltar, 1859–68.		Malta, 1859–68.		Ionian Islands, 1859–63.		Nova Scotia, &c., 1859–68.		Canada, 1859–68.		Newfound-land, 1859–68.		Bermuda, 1859–68.		Windward and Leeward Command. 1859–68.		Jamaica, 1859–68.	
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.
I. 1. Miasmatic Diseases,	194·0	1·07	241·2	4·36	409·3	7·00	308·4	4·11	121·0	·69	136·5	1·18	173·0	..	254·4	18·70	503·2	6·03	395·0	11·90
2. Enthetic ,,	310·6	·11	188·1	·04	78·0	..	118.8	·05	119·4	..	168·0	·03	41·0	..	64·5	..	186·3	..	119·7	..
3. Dietic ,,	8·5	..	13·5	·10	14·9	·19	7·6	·10	14·5	·36	19·2	·15	38·0	·75	34·0	..	34·4	·46	32·6	·43
4. Parasitic ,,	37·9	·07	6·9	..	10·9	..	4·2	..	9·3	..	7·9	..	4·1	..	4·3	·25	5·7	..	10·3	..
II. 1. Diathetic Diseases,	3·9	·13	3·9	·10	4·6	·16	1·5	·25	1·6	·07	3·1	·03	2·6	..	4·5	·08	8·3	..	3·2	·14
2. Tubercular,	16·8	3·17	8·8	1·26	10·5	1·88	9·4	1·32	10·0	1·44	8·8	1·69	10·8	2·61	12·2	2·28	11·0	1·67	9·9	1·74
III. 1. Diseases of the Nervous System,	19·2	·73	17·4	·43	17·7	·57	16·7	·66	13·6	·94	16·4	1·05	32·1	1·86	30·6	3·04	35·6	1·76	22·8	1·45
2. ,, ,, Circulatory ,,	8·9	1·09	6·5	·80	8·7	·90	4·9	·61	3·4	·94	4·6	1·27	9·3	1·49	6·2	1·18	7·9	·93	6·7	1·90
3. ,, ,, Respiratory ,,	86·2	1·31	47·5	·45	44·4	·81	41·8	·61	50·9	1·30	70·4	1·47	77·5	1·86	40·7	·59	33·1	·28	42·3	·58
4. ,, ,, Digestive ,,	37·2	·58	37·0	·31	46·9	·79	41·0	·46	25·7	·44	34·7	·42	32·4	..	50·9	·92	67·3	1·02	65·4	1·16
5. ,, ,, Urinary ,,	2·9	·17	2·3	·18	2·2	·19	2·3	..	1·8	·03	2·0	·15	2·2	·37	1·5	·08	1·9	..	2·3	·14
6. ,, ,, Reproductive ,,	10·3	..	16·4	..	12·6	..	3·6	..	4·9	..	7·0	..	3·3	..	9·7	..	17·2	..	6·2	..
7. ,, ,, Locomotive ,,	4·2	·02	4·0	·06	4·7	..	2·8	·05	2·3	·03	3·1	·02	4·8	..	3·4	..	3·6	..	5·4	·14
8. ,, ,, Integumentary ,,	124·8	·04	113·3	..	126·3	·03	132·4	..	75·6	..	99·5	·05	51·4	..	103·7	..	163·2	..	140·7	..
IV. Diseases of Nutrition,	2·4	·03	2·6	·01	5·0	·05	4·3	..	1·0	..	1·9	..	1·5	..	4·8	..	3·4	..	24·5	·14
V. 1. Accidents,	85·1	·65	75·0	·43	81·6	1·38	78·0	·46	83·3	1·16	100·8	1·90	94·7	1·49	95·6	2·19	102·0	·74	132·3	·43
3. Homicide,	..	·02	..	·06	..	·01	·1	·05	..	·14	..	·10	..	..	..	·84	..	..	..	·14
4. Suicide,	·2	·31	·2	·33	·1	·35	·1	·10	..	·22	·1	·34	..	·37	..	..	·4	·46	·1	·72
5. Execution,	..	·01	..	·06	..	·03	..	·05	..	..	..	·02	..	..	..	..	..	..	..	..
6. Corporal Punishment,	1·6	..	2·5	..	2·2	..	1·9	..	2·1	..	1·5	..	1·9	..	2·3	..	2·0	..	3·3	..
Diseases not Specified,	..	..	1·4	..	1·2	..	1·4	..	·8	..	·3	..	..	1·9	·9	..	2·0	..	·5	..
No Appreciable Disease,	1·1	..	1·6	..	·3	..	..	..	..	..	·7	..	..	..	·2	..	·1	..	..	..
Total,	955·8	9·51	789·1	8·98	882·1	14·34	782·2	8·88	541·2	7·76	686·5	9·87	582·5	10·80	724·4	30·15	1188·6	13·35	1023·2	21·01

I.—EUROPEAN TROOPS—*Continued.*

Stations and Period of Observation.	St. Helena, 1859-68.		Cape of Good Hope, 1859-68.		Mauritius, 1859-68.		Ceylon, 1859-68.		Australia and Tasmania, 1859-68.		New Zealand, 1859-68.		South China, 1859-68.		Japan, 1864-68.		Bengal, 1860-68.		Madras, 1860-68.		Bombay, 1860-68.	
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.
I. 1. Miasmatic Diseases,	280·5	2 00	276·1	2·41	544·5	12·42	564·1	10·70	176·2	1·47	204·0	2·02	1280·9	30·14	518·2	8·83	906·3	15·92	545·3	8·52	791·2	11·14
2. Enthetic ,,	152·1	..	278·8	·09	118·6	..	198·7	·11	159 6	..	27·1	·04	354·3	·06	532·0	..	261·0	·32	253·0	·35	250·3	·27
3. Dietic ,,	37·8	·20	17·1	·44	22·2	·17	38·0	·22	30·9	·09	12·5	·32	40 3	·38	28·0	·25	13·5	·18	25·0	·20	20·5	·23
4. Parasitic ,,	5·4	..	10·2	..	6·1	..	6·3	..	3·8	..	15·2	..	6·6	..	11·7	..	8·3	..	12·1	..	10·4	..
II. 1. Diathetic Diseases,	4·2	·20	3·9	·04	3·2	..	7·3	·11	6·1	·19	5·4	·10	6·0	·32	4·9	..	8·1	·24	6·7	·23	9·8	·20
2. Tubercular ,,	7·2	1·40	9·7	1·61	9·8	2·16	17·5	2·93	15·2	4·81	8·6	1·76	14·3	2·94	8·6	1·04	11·0	2·49	16·5	2·17	10·1	1·97
Diseases of the																						
III. 1. Nervous System,	26·9	1·60	24·8	1·27	19·2	1·05	45·2	2·36	24·6	1·57	20·1	·80	34·1	2·62	15·6	1·04	35·6	1·84	37·6	1·64	33·7	1·31
2. Circulatory ,,	11·8	·60	16·7	1·73	9·0	·70	19·7	1·01	10·0	2·16	8·1	1·70	11·0	1·02	3·4	1·56	10·5	1·06	16·8	1·30	12·2	·96
3. Respiratory ,,	38·5	1·00	47·0	·95	44·2	·70	81·0	1·01	52·3	·59	51·5	·80	78·6	1·40	46·7	3·12	54·0	1·13	52·1	·65	47·1	·80
4. Digestive ,,	53·4	·40	48·2	·76	67·8	1·52	131·7	3·04	43·9	·78	33·0	·74	101·5	2·36	33·5	1·04	132·5	3·75	150·0	4·27	114·3	3·24
5. Urinary ,,	4·0	..	2·2	·12	2·4	·05	5·6	·22	3·1	·39	2·4	·24	2·4	·13	3·6	·78	2·7	·18	3·1	·17	2·6	·07
6. Reproductive System,	6·2	..	12·0	..	11·4	..	16·2	..	4·0	..	5·5	..	10·9		14·0	..	15·2	..	17·1	..	12·2	..
7. Locomotive ,,	2·0	..	4·8	..	5·4	..	3·7	·22	2·2	..	3·7	..	5·0	·06	4·9	..	5·8	·02	5·0	·02	4·7	·01
8. Integumentary ,,	65·0	..	116·9	..	95·2	·05	165·2	..	66·6	..	70·9	·10	130·8	·13	128·0	..	103·1	·06	125·3	·07	97·5	·07
IV. Diseases of Nutrition,	·7	..	1·7	..	7·3	·12	8·1	·22	..	..	·7	..	26·9	·70	10·4	..	9·9	·18	11·7	·15	6·9	·13
V. 1. Accidents,	130·4	1·40	98·0	1·30	65·6	·93	131·0	1·80	100·0	·98	89·5	2·34	100·1	2·62	134·2	2·34	90·0	1·87	112·5	1·95	96·1	1·62
2. Battle,	..	..	..	..	..	..	..	..	..	..	12·5	4·96	..	..	..	..	·5	·15	..	..	..	..
3. Homicide,	..	..	..	·09	..	..	..	..	..	..	·1	·66	..	·32	..	·25	..	·02	..	..	..	·05
4. Suicide,	·3	·20	·1	·19	·4	·76	..	·34	..	·29	·1	·04	..	·45	..	·51	·1	·32	..	·35	·1	·44
5. Execution,	..	..	..	·02	..	..	..	..	..	..	..	·02	..	..	..	..	..	·01	..	..	..	·02
6. Corporal Punishment,	1·3	..	3·0	..	2·9	..	2.9	..	2·2	..	15·5	..	2·8	..	3·1	..	·9	..	2·1	..	1·4	..
Diseases not Specified,	2·3	..	4·1	..	3·3	..	3·0	..	6·5	·09	1·6	·04	17·2	·13	..	..	2·9	·15	3·1	·13	·6	·07
No Appreciable Disease,	·3	..	..	..	·2	..	·3	..	·5	..	..	..	..	..	1·5	..	..	..	5	..	..	..
Total,	830·3	9·00	975·3	11·02	1038·7	20·63	1445·5	24·29	707·7	13·41	588·0	16·08	2223·7	45·78	1502·3	20·76	1671·9	29·89	1395·5	22·17	1521·7	22·60

TABLE II.—COLONIAL TROOPS.

Stations and Period of Observ..tion.	Malta, 1859-68.		Windward and Leeward Command, 1859-68.		Jamaica, 1859-68.		Bahamas, 1859-68.		Honduras, 1859-68.		Sierra Leone, 1859-68.		Gambia, 1859-68.		Gold Coast and Lagos, 1859-68.		Ceylon, 1859-68.		China, 1859-68.	
	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.	Admitted.	Died.
I. 1. Miasmatic Diseases,	286·0	2·20	248·5	3·93	448·0	6·95	310·6	3·58	403·1	2·90	286·5	5·61	485·0	4·13	691·0	23·30	615·3	8·53	1070·4	19·69
2. Enthetic "	64·3	..	280·3	..	396·0	·55	236·2	·55	126·0	..	272·4	·23	229·0	1·50	232·5	..	25·3	..	97·2	·48
3. Dietic "	·6	..	1·3	..	2·5	..	2·5	..	2·2	·97	1·2	·46	2·6	..	3·9	·21	1·1	·07	2·5	..
4. Parasitic "	2·7	..	6·7	..	8·8	..	4·7	..	11·6	..	38·3	..	146·	..	100·9	..	51·6	..	32 9	..
II. 1. Diathetic Diseases,	2·2	·16	5·1	·47	5·8	·55	12·4	·55	4·5	..	5·1	..	9·4	1·50	10·9	·65	5·9	·52	14·1	1·24
2. Tubercular "	4·5	1·51	20·3	7·01	21·8	6·81	27·3	11·03	7·0	3·54	19·6	8·88	11·6	7·13	14·6	6·31	2·9	1·34	3·8	1·10
III. 1. Diseases of the Nervous System,	16·4	1·00	19·2	2·02	24·2	1·77	16·8	1·93	15·8	2·58	12·1	1·87	13·5	1·88	21·0	2·83	11·1	·89	18·3	·34
2. " " Circulatory "	2·3	·84	2·8	1·19	2·3	·68	2·2	·55	2·9	1·93	2·6		2·6	·75	1·5	·86	1·3	·74	1·6	·62
3. " " Respiratory "	73·6	·33	72·1	2·50	78·2	4·09	83·8	4·90	53·1	1·93	67·0	4·68	106·1	8·63	75·9	4·79	52·1	·96	87·8	2·75
4. " " Digestive "	148·3	1·51	32·4	1·31	37·4	2·04	21·8	1·38	33·5	·64	29·4	4·68	34·1	2·62	42·6	1·52	38·6	·89	55·4	·83
5. " " Urinary "	2·0	..	3 0	·59	4·7	·13	2·7	·55	1·9	..	3·3	·46	7·1	·75	5·8	·43	·4	..	2·5	..
6. " " Reproductive System,	2·8	..	13·2	..	14·4	..	15·7	..	14·2	..	15·0	..	14·6	..	14·8	..	8·0	..	3·5	.07
7. " " Locomotive "	..	..	2·6	..	6·0	..	5·2	..	3·2	..	12·6	·23	8·2	·37	8·6	·43	1·0	..	1·8	·07
8. " " Integumentary "	136·4	..	125·5	·11	133·2	·27	100·0	·55	139·4	·32	161·5	..	129·4	..	174·6	..	123·0	·07	89·1	·21
IV. Diseases of Nutrition	..	..	1·2	·23	3·2	·13	·8	..	2·9	..	1·2	·23	1·5	..	1·5	·21	1·3	·22	28·8	2·89
V. 1. Accidents,	78·7	·84	72·3	·59	73·5	·68	75·0	·27	80·8	1·93	56·8	·70	64·1	..	83·9	2·18	73·0	·29	62·9	·83
2. Battle,	..	..	..	..	..	..	..	..	4·2	1·93	8·6	·70	9·0	1·50	3·6	·21	..	..	·9	·07
3. Homicide,	·1	·33	·2	·36	·2	·27	..	..	..	·32	..	..	·3	·37	..	·43	..	..	..	·07
4. Suicide,	·3	..	·1	·72	..	·82	·2	·27	·6	·97	·2	·70	·3	1·12	·2	·65	..	·22	·1	·21
5. Execution	..	..	..	·11	..	·27	..	..	..	·32	..	..	..	·37	..	·65	..	..	..	·07
6. Corporal Punishment,	·6	..	4·4	..	2·8	..	3·3	..	3·9	..	3·7	..	2·2	..	6·3	..	·7	..	·8	..
Diseases not Specified,	..	..	8·0	..	1·4	..	2·2	..	·9	·32	·9	..	2·2	·37	·6	·21	·6	·07	1·3	·13
No Appreciable Disease,	..	..	·1	..	..	..	..	..	..	..	..	..	·3	..	..	..	..	..	..	..
Total,	821·8	8·72	919·3	21·14	1264·4	26·01	923·4	26·17	911·7	20·60	998·1	29·43	1147·7	32·99	1494·7	45·87	1013·2	14·81	1575·7	31·67

CHAPTER IV.

PERSISTENT PERNICIOUS INFLUENCE OF MALARIOUS CLIMATES.

SUFFICIENTLY authenticated examples are now on record, which prove the persistent pernicious influences of malaria both on races of mankind and on bodies of men subjected to their influences for periods of time beyond six or eight weeks, and especially during a temperature above 60°. It is known that after the Walcheren expedition our troops continued to suffer from paludal fevers for five, six, eight, and eleven months after their return to this country, although located in as salubrious quarters as could be procured for them. M. Boudin, also, in his *Lettres sur l'Algérie*, fully shows the persistent pernicious influence of paludal poison on the French and English colonists there. During the recent war against Russia, the persistent pernicious influence of the residence of the troops in Bulgaria, during a period of three of the hottest months of the year, continued to make itself more or less manifest throughout the whole of the campaign in the Crimea.

The more chronic and enduring influence of marsh malaria on a race finds abundant illustration amongst the people who inhabit Campagna, Maremma, Pontines, and other insalubrious localities in classic Italy; and in France, in Forez, La Brenne, Sologne, Berry, Dombée, and La Bresse.

Every page of Sir Ranald Martin's classic work *On Climate* goes to establish the belief in the *degrading* influence of the Indian climate, *where malaria prevails*, on European constitutions. The slow, increasing influence of a fever which no acclimation overcomes maintains the high death-rate; and Twining also long since noticed that, "*after careful inquiry, he was unable anywhere to find a sample of the third generation from unmixed European stock.*" Thus upon European colonization, where malaria is permitted to exist, in India, "Nature has set her ban—a blighting interdiction"—till the progress of sanitary measures shall make this fine country the great garden of the world.

The characteristics of degeneracy, as shown by paludal races and inhabitants of such regions, may be shortly summed up as follow:—A more or less intense state of cachexia, stunted growth, engorgement of the chief viscera, especially of the spleen, languor, and inertia of all the functions—aggravation of ordinary diseases, the superaddition to them of lesions only explicable by the atony and diminished power of reaction of the nervous centres, and finally, a diminished longevity. The influence of the degenerating principle in the sphere of the intellectual and emotional faculties is no less remarkably manifested by the torpor of intelligence, the apathy, a kind of habitude, passing on under some circumstances to a state of idiocy, and under all to the most extreme indifference (MORELL, and Reviewer in *Med.-Chir. Review*, January, 1858, p. 82).

The length to which this volume has extended will not permit of further remarks on the Geography of Disease. Enough, perhaps, has been written in illustration of some of the more important directions which the study may take, and to show that the subject presents a vast field for investigation, and claims the united exertions of every one in its exploration, who is interested in the progress of the Science of Medicine, of Politics, and of Social Health. The immediate object of the study is to ascertain the laws by which disease is distributed or propagated, or the manner in which certain conditions inimical to health are found to prevail in certain localities or regions, with a view to the prevention of disease by sanitary measures; and how many of the phenomena relative to the regional distribution of disease are elucidated by the facts of *physical geography.*

The sources of information on the subject exist in reliable tables of sickness and mortality, a knowledge of the physical conformation of the earth's surface, and the meteorological agencies to which it is exposed. A *statistical* branch has now been created in the Office of the Army Medical Department of this country, " thus at last affording an official recognition of the value of statistics in the investigation of those questions on which the efficiency of our army so much depends, and by the satisfactory solution of which so much sickness and mortality among soldiers may be prevented, and economy both in life and in money attained."

As the sun proceeds northward in the ecliptic, so the sickly season advances from the southern to the northern islands. In the Mediterranean the mortality is doubled in the hot season between July and October; and in the Northern States of North America the posts of the army are regularly abandoned as the hot or sickly season approaches. In temperate regions this order is reversed. Throughout Europe generally, the maximum mortality occurs at the end of winter, and the minimum in the middle of summer. The Registrar-General of England calculates that a fall of the mean temperature of the air from 40° to 4° or 5° below the freezing-point destroys from 300 to 500 of the population of London. The agency of the wind is manifested in the distribution of heat and moisture, and in the comparative density of the air, as well as by its direct influence as a distributor of malarial poison. The absence of wind was uniformly noted as a concomitant of cholera, which, in Britain and elsewhere, has been observed to be developed, and to be most virulent, when the calm was the greatest, and often began to abate when the wind rose.

It may not be out of place merely to indicate some definite and practically important subjects of study relative to this interesting department of medical science.

1. A study of the climatology and the diseases of the different quarters of the globe illustrate most clearly that the morbid conditions produced by certain pathological states, while they are of a fixed character, are more intense in their severity, more continuous in their development, and more prolonged in their existence, in some places than in others.

2. A study of the climatology of towns, and such circumscribed

districts, is of the greatest practical importance to the physician in all questions relative to change of air for the invalid. On this subject the work of Sir James Clark *On Climate*, with its inestimable series of meteorological tables, is still a classic source of reference; while many monographs have also been written of late on particular localities.* The reader will also consult with advantage an admirable account of the effects of change of climate on diseases of the lungs, by Dr. Walshe, in the appendix to his work *On Diseases of the Lungs.*

3. Our medical officers of health are industriously mapping out the realms of disease, which often too definitely manifest themselves amongst the vilest purlieus of our cities. Systematically and energetically, many of the causes of diseases are thus more effectually exterminated. Those who have to care for the sanitary state of our armies now believe that similar means of preventing disease may be successfully applied abroad. Sanitary officers are now appointed to great military expeditions, and a board of sanitary officers is recommended, and has been established, in all the Presidencies of India. For this purpose a study of the geographical distribution of diseases, and of the causes which lead to their special distribution, obviously becomes of the greatest practical use. By it we learn that certain classes of diseases, rather than others, are mainly under the influence of terrestrial and meteorological causes—namely, those of the ZYMOTIC class, and which are stamped with special *miasmatic* characters. In proportion, therefore, as we become capable of knowing that particular diseases of this class are limited to certain portions of the earth, and can trace the meteorological laws of their geographical distribution and diffusion, we necessarily obtain clearer conceptions of their causes and modes of propagation, as well as more practical knowledge of the means of their prevention and of cure.

By such knowledge large masses of men may be more successfully cared for in foreign countries; and the topographical position and construction of habitations may be determined upon with a certain definite knowledge to guide the chooser. Military barracks and hospitals may be judiciously provided for abroad, and with all the aids of scientific knowledge, the diet, the clothing, and the military exercising of the troops may be arranged so as to suit the physical climate of the place in which armies are to campaign or garrisons to be located.

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* Of these the following may be referred to:—

Diseases of the New Zealanders, by Dr. Arthur Thomson. *Medico-Chirurgical Review*, commencing April, 1854.

On the Climate of Algiers, by Dr. Arthur Mitchell. *Medico-Chirurgical Review*, commencing January, 1856.

A Comparative Inquiry as to the Preventive and Curative Influence of the Climate of Pau and other Places, by Dr. Alexander Taylor.

Change of Climate, and an Account of the Climates of Spanish Towns, by Dr. Francis.

On the Climate of Spain and Australia, by Dr. Burgess. *Medico-Chirurgical Review*, October, 1854.

Climatology of the United States, by Blodget.

CHAPTER V.

CONCLUSION.

In bringing these volumes to a close (for the *sixth* time), the Author is painfully conscious of many sins of omission and of commission in the execution of his task. Much has still been left unwritten "of the thousand ills that flesh is heir to," for which it might have been desirable to find a place in these pages; but if the work shall serve merely as a guide to the acquisition of knowledge, and the acquirement of practical skill in the Art of Medicine, the Author will have accomplished his design.

After the student has read and carefully studied all that is contained in books of this kind, he will still find that he has only made a commencement of the great study and real labour of his professional life. To extend his knowledge, and to acquire practical skill, he must possess, cultivate, and foster the faculty of observation above all things, combined with the exercise of a sound judgment. From such a work as this (which simply transmits to the future a sketch or record of our present knowledge of the natural history of well-defined diseases and their remedies), he may acquire a *book-knowledge* of the Science of Medicine; and, by clinical instruction, with such a book as his guide, and experienced clinical teachers to point out to him the best methods of investigation, he may also acquire some *practical experience.* It is such a method of study which will give the young medical man an extensive view of the Science of Medicine; and, at the same time with lessons in exactness of knowledge, some experience in the art of investigation, as well as in the direct and intelligent management of disease. It is exactness of knowledge which alone can give directness and intelligence of purpose in the treatment of diseases; and such knowledge will eventually place him in the best position to begin the practice of his profession.

It has been well observed by the late Dr. Joseph Bullar, of Southamptom—that "the treatment of disease is an ART requiring daily and hourly practice; and nothing but actual practice, with the responsibility on a man's own shoulders, can make a practical man." As this practice requires close attention, reflection, and judgment, as well as prompt action, the more a man's mental powers are educated by his college training the better a practitioner he must in time become, if he devotes himself to his duties. Those who succeed in a marked way in acquiring a knowledge of the more theoretical parts of medicine, and who show their power strikingly in obtaining a full and exact knowledge of the natural history of diseases, and of the literature of the Science of Medicine, have sometimes been set down as not likely to be practical men. This is an unjust conclusion—a popular fallacy. It requires only time and the proof of success to show that, when such men apply their powers to practice, they become the most successful of physicians. They bring to the bedside of the sick, minds well stored with professional knowledge—vigorous, quick, and well-cultivated mental powers, which cannot fail to place them amongst the most useful and trustworthy of practical men, in the application of remedies for the prevention, cure, or relief of diseases—the chief end and object of all our study.

APPENDIX TO VOL. II., SIXTH EDITION.

Referring to the subjects of Embolism and Metastatic Abscesses, vol. i., *page* 782; *and vol.* ii., *page* 804.

In a paper read before the Medico-Physical Society of Wurzburg, June, 1871, "On Metastatic Foci" ("Abscesses"), by Dr. von Recklinghausen, he mentions that, "With regard to the nature and the origin of the manifold Metastatic Abscesses which come under our observation, after various infectious diseases in the organs belonging to the great and to the minor circulation, many doubts have been entertained whenever Endocarditis happened to be absent at the same time. It appeared most probable, according to Virchow's view, that their origin had to be searched for in capillary emboli."

Dr. v. Recklinghausen has succeeded in discovering in a long series of infectious diseases—chiefly Pyæmia, Puerperal Fever, Typhus, Acute Rheumatism of the Joints, also in Urinary Infiltration and Gangrene of the Lungs—the cause of those small abscesses, to be *miliary accumulations* of minute organisms, which come under the heading of Micrococcus. They are identical with those forms observed by Drs. Buhl, Oertel, and Nasiloff, in Diphtheritis (see *Centralblatt*, 1870, p. 634, and 1871, p. 134), and those described by Klebs in "Cystitis and Pyelo-nephritis" (inflammation of the pelvis of the kidney), and they may be safely distinguished from all substances of an animal nature, or such as are derived from them by way of decomposition, both by their appearance and by the great resistance they show when treated with chemical agents, even with caustic alkalies.

The author noticed this fungoid deposition in its greatest abundance in the kidneys, sometimes in a quite recent state, without any re-active appearance, but more frequently surrounded by an area of hæmorrhagic or purulently infiltrated tissue. This accumulation is not confined to the blood-vessels, among which the smallest veins contain the largest quantities, but the author met with them also inside Bowman's capsules and the urinary tubuli: from the latter, if carried down by the urine, they could be detected either during lifetime or, at *post-mortem* examinations, in the contents of the bladder. Often those various channels are filled with such bulky masses of them that they cause a nodular swelling, and, at a later stage, perforation and dropping out of fungoid masses.

The author discovered another form of fungus, different in every respect from Micrococcus, consisting of nodular beam-like bodies of a green colour, in a case of scarlatina, forming the constituent parts of similar small foci (abscesses) in the renal pyramids. The urine of the patient who died the sixth day contained cylinders (tubuli), which were covered with the same elementary bodies.

The frequency of the occurrence of those various fungoid formations, especially in the kidneys and the urine, as well as their distribution over a large field, and their large number are, in the opinion of the author, significant indications of the localization of these problematical germs in infectious diseases generally.

These fungoid metastatic productions can not be considered as emboli, such as defined by Virchow. Not only the extra-vascular situation of the material (contents) carried off is opposed to this view—(for the author could determine the presence of Micrococci in the alveoli of the lungs)—but, especially, the circumstance that in a large number of carefully observed cases Endocarditis never occurred simultaneously. The thin covering layers which, in one case only, were observed near the mitral valve, are most probably to be considered as metastatic in the same sense as the foci in the other organs. (From *Centralblatt fur Medicinischen Wissenschaften,* von Dr. Rosenthal. November, 1871.)

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